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REVIEW AND SYNTHESIS

Fishing out marine parasites? Impacts of fishing on rates of parasitism in the ocean

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Abstract

Among anthropogenic effects on the ocean, fishing is one of the most pervasive and extends deepest into the past. Because fishing reduces the density of fish (reducing transmission efficiency of directly transmitted parasites), selectively removes large fish (which tend to carry more parasites than small fish), and reduces food web complexity (reducing transmission efficiency of trophically transmitted parasites), the removal of fish from the world's oceans over the course of hundreds of years may be driving a long-term, global decline in fish parasites. There has been growing recognition in recent years that parasites are a critical part of biodiversity and that their loss could substantially alter ecosystem function. Such a loss may be among the last major ecological effects of industrial fishing to be recognized by scientists.

Keywords

Community ecology, community-level impacts of fishing, fisheries, host–parasite interactions, indirect effects of fishing, parasitology, species interactions.

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INTRODUCTION

Two decades ago, Dobson & May (1987) suggested that incorporating a consideration of parasites into fisheries management might shift the optimal choice among management plans. They argued that fishing could drive host populations below the threshold density for parasite transmission, leading to extirpation or 'fishing out' of the parasite, a desirable outcome for fisheries affected by parasites that increase mortality or decrease marketability of their hosts. Dobson and May's clever idea was never formally incorporated into the fisheries management tool kit and its adoption seems unlikely (Jackson *et al.* 2001), but in recent years, as fisheries managers and conservationists have increasingly embraced the value of marine protected areas (MPAs; Lubchenco *et al.* 2003), others have retooled Dobson and May's model to suggest that an unexpected consequence of protecting fished species could be a rise in infectious disease towards historically higher levels (McCallum *et al.* 2005). Thus, theoretical work has been pushing towards the conclusion that large-scale fishing can 'fish out' marine parasites.

It would be difficult, however, to assess the hypothesis that a temporal decline in parasites is due to fishing, given that many factors other than fishing could affect parasites (Lafferty & Kuris 1999, 2005; Marcogliese 2001; Mouritsen & Poulin 2002a; Lafferty & Holt 2003; Lafferty *et al.* 2004). For instance, habitat destruction, pollution, and climate change could drive a loss of parasite abundance, diversity, or both, by reducing the abundance of hosts (Hudson *et al.* 2006; Dobson *et al.* 2008). Brooks & Hoberg (2007) argue that parasites are unlikely to become extinct, given their ability to switch hosts, and several authors have suggested that marine parasitism could be on the rise because anthropogenic stressors can increase the susceptibility of marine life to parasitic infection (Epstein 1998; Harvell *et al.* 1999, 2004; Bustnes *et al.* 2000). In short, arguments can be made that parasites should decrease, increase, or remain unaffected by anthropogenic pressures on the oceans (Lafferty 2003).

Here, we consider the evidence for fishing's impacts on parasites. Given recent emphasis on ecosystem-based management of fisheries, we also consider the potential for indirect effects (i.e., conditions under which fishing could change rates of parasitism in non-target species). We present several mechanisms by which fishing affects parasite communities, using a conceptual framework that distinguishes among mechanisms by the scale of their effects on

ecosystems (i.e., on population vs. community processes) and whether they increase or decrease rates of parasitism (Fig. 1). We do not consider the reciprocal impacts of parasites on fisheries, a topic that has received some attention in the fisheries literature and is beyond the scope of this work.

We use the term 'parasite' to refer all natural enemies that exploit only one victim in a single life stage (i.e., to distinguish from predators, which exploit many victims) and that rely upon infectious processes to find new hosts. Our definition therefore includes 'typical parasites' (sometimes called 'macroparasites'), as well as organisms that are commonly considered 'pathogens' or 'microparasites' (Lafferty & Kuris 2002). The dividing line between the two groups concerns the intensity-dependence of host pathology: for 'typical parasites', pathology increases in severity with the number of independent infection events, but no such relationship exists for 'pathogens', because they multiply within their host.

We define the impacts of 'fishing' narrowly, as *the removal of wild fish and invertebrates from the ocean*. We therefore exclude from consideration several additional ways in which the fishing industry can affect the abundance and diversity of marine parasites, including habitat destruction, introduction of exotic parasites, relocation of native parasites from high-infection to low-infection areas, and concentration of parasites by aquaculture operations. Although these other impacts may be important, fish removal itself is perhaps the most pervasive human influence on the oceans, being globally distributed and having affected marine ecosystems for hundreds of years (Jackson *et al.* 2001).

This review suggests that, although its effects can be divergent and complex, fishing will generally tend to reduce the abundance and diversity of parasites as predicted by Dobson & May (1987). Given the ubiquity of overfishing, it seems likely that humans have rearranged the distribution patterns of marine parasites, with important consequences for ecosystems.

HOST POPULATION PROCESSES

The impacts of fishing, as defined here, occur largely at the population level. However, population-level impacts of fishing can either increase or decrease the abundance of parasites. We begin by reviewing theoretical predictions and empirical studies addressing the question of whether fishing a single host population can change the abundance of that host population's parasites. A key driver of this relationship is the extent to which the individuals removed by a fishery depart from a random sample of the population.

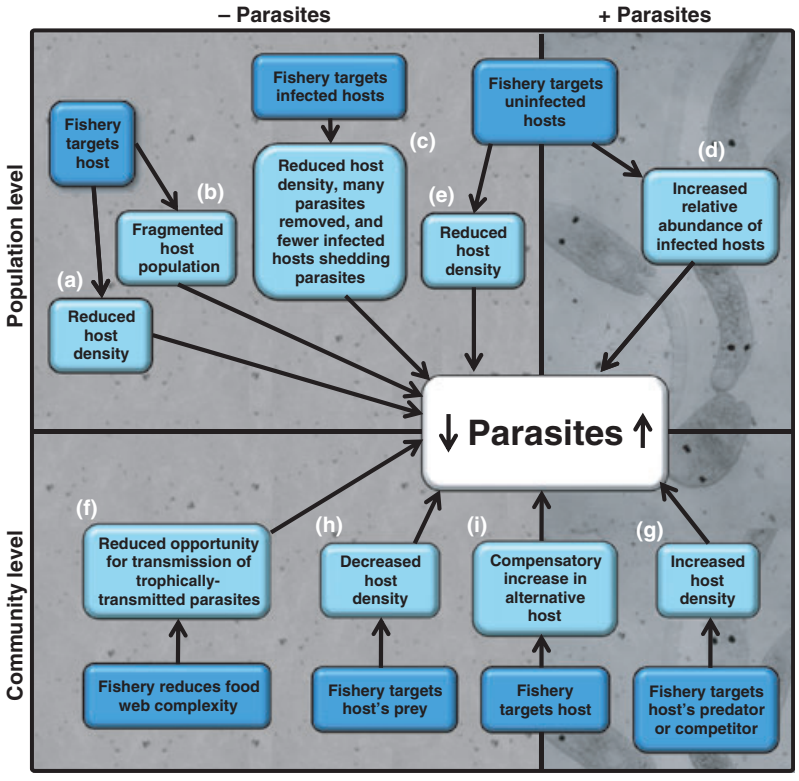

Figure 1 Impacts (a–h) of fishing on rates of marine parasitism. Impacts described in the left region of the diagram tend to decrease rates of parasitism (− parasites), while impacts described in right region tend to increase rates of parasitism (+ parasites). Impacts described in the top region of the diagram tend to act on host populations, while impacts described in the bottom region tend to act on communities in which hosts are embedded. Actions taken by fisheries are denoted in dark blue boxes and epidemiologically relevant impacts of each action are denoted in light blue boxes. See in-text citations for further explanation of each impact (a–h).

Fishery targets host, reducing host density

Theory

As fishing reduces the density of fish hosts, transmission of parasites among those hosts should decline (Fig. 1a). This linear density-dependent transmission is a key assumption of mass-action consumer–resource models, which have facilitated a great deal of progress in our understanding of the population dynamics of infectious diseases (McCallum *et al.* 2001). These results are illustrated by McCallum *et al.* (2005), who demonstrate that infection prevalence declines substantially with increasing fishing mortality (due to reductions in transmission) in a mathematical model of abalone and the directly transmitted *Rickettsia*-like prokaryote pathogen that causes abalone withering syndrome. Such patterns can be reinforced by mitigation of crowding-related 'stress' or competition for resources, which would increase immunocompetence and decrease susceptibility to infection at low host densities (Lafferty 1997; Lafferty & Kuris 1999).

Whereas linear density-dependent transmission is the classic assumption for epidemiology, other forms of transmission are possible and nonlinear relationships between transmission and density may be relatively common for a variety of reasons (McCallum *et al.* 2001). For instance, social interactions or other modes of aggregation can maintain high contact rates among hosts – and, hence, high transmission rates – at low host densities (Lafferty & Gerber 2002). Even without the occurrence of host aggregations, the contact rate among individuals probably saturates (i.e., asymptotes) at high host densities, capping transmission rates (which is potentially destabilizing for parasites, in that it makes invasion of the host population more difficult). In contrast with the patterns expected under conditions of host sociality or aggregation, low mixing rates of individuals (e.g., in territorial hosts) can result in spatial clustering of infection, but slow spread among host populations. Finally, density-dependent transmission does not hold for vector-transmitted diseases (e.g., viruses, bacteria and trypanosomes transmitted to fish hosts via leech vectors), which depend on vector biting rates and cannot, therefore, be directly tied to host density. Though these caveats suggest that the assumption of linear density dependence does not always hold in fished populations, they do not preclude a negative effect of fishing on parasites; they simply shift the degree of fishing required to observe an effect on parasites via host density-mediated mechanisms (e.g., transmission; McCallum *et al.* 2001). In other words, where there is nonlinear density dependence of transmission or vector-transmitted disease, fishing might need to be more intense (i.e., reducing host density to very low levels) for a negative effect on parasites to be observable. Furthermore, although factors like social aggregation can decouple transmission from host density at local scales, fishing should still reduce the abundance of parasites at the metapopulation or species scale, by isolating individual host populations and thereby increasing the likelihood of parasite loss from those populations and reducing the likelihood of parasite dispersal among populations (see Fishery targets host, fragmenting host population).

Sometimes, the density of multiple species will influence transmission. The combined density of various hosts can drive the dynamics of parasites with low host specificity, such that non-fished 'reservoir' hosts can govern transmission to a fished species (see Variability in the response of parasites to fishing). Trophic cascades add further complexity if they release the prey of fished species from predation pressure, leading, indirectly, to increases in transmission at lower trophic levels (see Fishery targets host's predator, competitor, or prey).

Outcomes of the interaction between fishing-mediated host density and parasite abundance will also be affected by spatial scale. Potentially influential attributes include the recruitment patterns of the host and parasite, which can be either relatively 'open' or relatively 'closed' at the scale of a fishery. Recruitment is closed at a given spatial scale if local recruitment is dependent upon local production of larvae. For hosts, local recruitment is usually the result of limited adult movement, short pelagic larval duration, larval behaviour that tends to increase retention of larvae, and low rates of flushing of the water mass. Parasite populations tend to have low dispersal when the free-living infectious stage endures for only a short period of time or has limited mobility, the mobility of the host is limited, transmission is direct, or the water mass experiences low rates of flushing (Kuris & Lafferty 1992). The recruitment pattern of both the parasite and the host influence the likelihood of a fishery to shift the abundance of fish parasites; for example, parasites with closed recruitment are more likely to be 'fished out' because they cannot be sustained by larvae from outside populations, especially if the host has open recruitment, which allows it to persist even at high fishing pressures. In contrast, because their abundance is not dependent on local host density, open-recruitment parasites would be difficult to 'fish out' (Kuris & Lafferty 1992). Relationships between fishing and parasites would be difficult to detect at local spatial scales if hosts move broadly, and comparisons of parasites inside and outside of protected areas can only reveal effects of fishing if there is relatively little movement of hosts between protected and fished areas. For instance, in a small fishery closure, Loot *et al.* (2005) found the effects of fishing to be more evident for the parasites of sedentary mussels than for parasites of mobile fish.

Counterintuitive hump-shaped relationships between host removal and prevalence – and even between host

removal and the absolute number of infected hosts – can arise when hosts develop long-term immunity to disease. First, host removal can increase recruitment into the susceptible class, increasing transmission. For instance, if immune individuals have low reproductive rates, the population birth rate is density-dependent, and a random subset of the population is removed by fishing (i.e., the fishery does not target fishes based on infection status), low to moderate host removal might increase the prevalence of infected hosts because the fishery acts to stimulate the production of new, susceptible individuals (Choisy & Rohani 2006; Holt & Roy 2007). However, immune individuals need not be demographically compromised and birth rate need not be density-dependent for this effect to occur; if fishing primarily removes immune individuals (e.g., in a fishery in which older individuals are both more likely to possess immunity and are larger), the relative abundance of susceptible individuals will increase, increasing transmission (Bolzoni *et al.* 2007). Whether this effect occurs in a natural population of fish will depend heavily on the exploitation rate; where rates of host removal exceed the low to moderate values at which prevalence and parasite abundance peak in these models, the counterintuitive increase in parasites with increasing fishing effort will not be observed (i.e., the system will be on the 'downhill side' of the hump-shaped curve). Such models also require the restrictive assumption that the fished species develops immunity to disease, which is most likely to happen in fisheries for mammals rather than fish (Bernstein *et al.* 1997; Rice & Arkoosh 2002). Finally, these models are unlikely to apply to most macroparasites, which tend not to induce permanent immunity to new infections (Dobson & May 1987).

Therefore, theory indicates that reduced host population densities might directly decrease or indirectly increase parasite abundance, depending on density-dependent processes in the host population, community-level interactions, immunity, degree of fishing pressure, and the parasite's transmission mode and efficiency. But despite these caveats, the dramatic reductions in host density associated with fishing should impair the transmission of host-specific parasites. In other words, because fisheries are extraordinarily effective at reducing the density of fish, they are generally expected to 'fish out' parasites from exploited stocks (Dobson & May 1987).

Empirical evidence

Several empirical studies conclusively demonstrate the relationship between host density and parasite abundance (Arneberg *et al.* 1998; Morand & Poulin 1998; Hochachka & Dhondt 2000; Arneberg 2002), but the lack of monitoring efforts for infectious diseases of marine organisms makes it difficult to assess temporal trends for ocean parasites. Ward & Lafferty (2004) tracked changes in the proportion of disease-related literature on various marine taxa published between 1970 and 2001. Their data reveal that reports of disease increased for turtles, corals, marine mammals, urchins, and molluscs, and did not change significantly over time for seagrasses, fishes, decapods, or sharks (see Fig. 2 and Appendix S1 in Supporting information); therefore, all taxa that have experienced substantial fishing-induced declines in abundance (i.e., fish, decapods, sharks) displayed non-significant, negative correlations of normalized disease reports with time, while taxa that have not experienced fishing-induced declines in abundance (i.e., turtles, corals, mammals, urchins, molluscs) displayed significant, positive correlations. This suggests that fishing may have a very general influence on marine disease dynamics.

A direct test of the relationship between fishing and parasite abundance would require comparing rates of parasitism before and after fishing relative to a control. This experiment has not yet been conducted, but many studies are suggestive. In one, museum specimens of lake trout (*Salvelinus namaycush*) from all five Great Lakes were examined for the presence of *Cystidicola stigmatura*, a swim bladder nematode parasite of salmonids. Whereas *C. stigmatura* was found to be prevalent in museum specimens collected before 1925, no parasites were found in specimens collected after 1925. This coincides with a

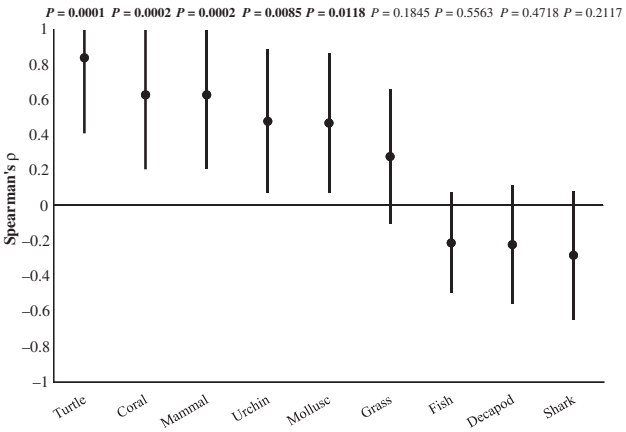

Figure 2 Change in incidence of disease among nine marine taxa between 1970 and 2000. Spearman's ρ was calculated by Ward & Lafferty (2004) for the relationship between time and per cent of literature reporting disease (where per cent of literature reporting disease is equal to number of publications reporting disease in a taxon divided by the total number of publications on that taxon times 100), except for Spearman's ρ of fish, which was calculated by the present authors (see Appendix S1 in Supporting information for methods and interpretation). Error bars represent 95% confidence intervals calculated using the methods of Hotelling (1953).

precipitous fishing-driven decline of lake trout populations, providing a likely example of a parasite that has been entirely 'fished out' of its host population (Black 1983, 1985). Similarly, a 2004–2005 mortality event of sea scallops (*Placopecten magellanicus*), caused by sponge, polychaete, and prokaryote infection, affected an MPA, the Nantucket Lightship Closed Area, but was not observed in nearby fished areas of the Gulf of Maine. This MPA had among the highest densities of scallops in the entire Gulf and the die-off was most severe in the northeast corner of the MPA, where the density of scallops was greatest. This strongly suggests that the parasites were able to take hold only where fishing restrictions had produced a relatively dense host population (Stokesbury *et al.* 2007). Finally, in a six-year experiment carried out in a Norwegian lake, experimental reduction of the density of whitefish (*Coregonus* spp.) dramatically reduced the prevalence of the cestode parasite *Diphyllobothrium ditremum* in the remaining whitefish population. *Diphyllobothrium ditremum* is indirectly transmitted, and the abundance of the other two hosts in its life cycle (copepods and birds) did not change through the course of the experiment; therefore, in this example, the effect of host density echoed through the entire parasite life cycle to negatively affect parasite abundance in the original, limiting host (Amundsen & Kristoffersen 1990). This final example is remarkable in that indirectly transmitted parasites typically have much lower host density thresholds than do directly transmitted parasites (Dobson & May 1987).

Fishery targets host, fragmenting host population

Although fishing does appear to increase fragmentation of fish populations sometimes, particularly when fish are strongly associated with benthic habitat (e.g., seagrass, mangrove, intertidal; Musick *et al.* 2001; Marteinsdottir & Pardoe 2008), fragmentation is probably less common in marine than terrestrial or freshwater systems due to the relatively high mobility and strong dispersal capabilities of many marine organisms (Fig. 1b; Hockey & Branch 1994). The effect of fragmentation on parasites will increase with host specificity and decrease with the persistence of free-living infective stages (Hoberg & Brooks 2008). If habitat fragmentation temporarily concentrates individuals into smaller habitat refuges, transmission could increase (see Fishery targets host, reducing host density; Holmes 1996); for example, the establishment of MPAs in an otherwise heavily fished area might cause a temporary aggregation of individuals within reserve boundaries, and a corresponding increase in parasites. Although this effect of fragmentation has proven to be a legitimate concern in terrestrial systems, it is unclear whether this effect would result from fishing.

Fishery targets host based on infection status

The previous sections addressed impacts on parasites when fisheries alter the abundance and spatial distribution of fish hosts; here, we review cases in which fisheries remove hosts selectively.

Fishery targets infected hosts

Selectively removing infected hosts reduces R_0 by making parasite mortality a function of fishing mortality (in addition to reducing overall host density), and in endemic populations, such selective removal can reduce parasite prevalence (Fig. 1c; Packer *et al.* 2003). Most fisheries target individuals possessing characteristics correlated with likelihood of infection. Large body size is perhaps the most-sought attribute of exploited fish, and this preference has driven dramatic shifts towards smaller mean body size not only across exploited species (Pauly & Watson 2005; Pauly *et al.* 2005), but also within exploited species (Bianchi *et al.* 2000; Friedlander & DeMartini 2002b; Jennings & Blanchard 2004; Pauly *et al.* 2005; Sala & Knowlton 2006; McClenachan 2008). In fish, body size increases with age, so the relatively higher parasite burden of large fish reflects the longer period of time over which those individuals have been accumulating parasites (though this will generally hold true only for macroparasites; Pacala & Dobson 1988; Hudson & Dobson 1995; Lo *et al.* 1998; Zelmer & Arai 1998; Poulin 2000). There is also evidence to suggest that large body size *per se* (i.e., independent of age) can be correlated with heightened parasite load, because large-bodied animals have relatively higher rates of movement and consumption and present a larger surface area for parasite contact (Guegan & Hugueny 1994; Arneberg 2002). Finally, another correlate of body size, trophic level, is also likely to influence the number of parasite species and individuals infecting a given fish, because high trophic-level predators are likely to host a greater number of trophically transmitted parasites.

We have only limited empirical data to demonstrate that abundance and diversity of trophically transmitted parasites increase with trophic level of the host (Lafferty *et al.* 2006), but this relationship is strongly suggested from first principles. First, because diversity of prey items consumed (i.e., intermediate hosts eaten) increases with increasing trophic level, high trophic-level hosts are exposed to the larval stages of a greater number of parasite species than are low trophic-level hosts. Because body size tends to increase with trophic level and large individuals consume a larger absolute amount of food, high trophic-level species will also tend to be exposed to a greater number of individual parasite larval stages than low trophic-level species. Finally, high trophic-level species integrate trophically transmitted parasites across a longer food chain length than do low

trophic-level species. In other words, high trophic-level species should 'bioaccumulate' parasite larval stages of their prey's prey. Though the relationship between trophic level and parasite richness and intensity has received only limited empirical attention (Lafferty *et al.* 2006), the logic outlined here suggests that there is reason to believe that rates of parasitism should increase with trophic level.

It is therefore likely that, as industrial fishing expanded, parasites were removed from fish populations at a rate disproportionately greater than the rate of removal of fish, due to the parasites' association with the large-bodied hosts preferred by fishers – that is, as we 'fished down marine food webs' (Pauly & Watson 2005; Pauly *et al.* 2005), we also 'fished out' marine parasites. For example, in the scallop (*Pecten alba*) fishery of Port Phillip Bay, Victoria, Australia, fishers target larger scallops, which tend to be more heavily infected with a bucephalid trematode parasite than are smaller individuals. Over time, the fishery has removed a substantial proportion of the larger scallops, causing the prevalence of the bucephalid parasite to decline precipitously (Sanders & Lester 1981). The tendency of parasites to be overdispersed in host populations (i.e., relatively few hosts possess most of the parasites within a host population; Shaw *et al.* 1998) should magnify the effects of such selective fishing.

Fishery targets uninfected hosts

In industrial fisheries, selective targeting of uninfected hosts or discarding of infected hosts (i.e., sorting infected hosts from the catch and releasing them) can be a common practice when parasitic infection renders fish less marketable. For example, in exploited crab populations that host nemertean egg predators (which affect only female crabs), the common policy of releasing trapped females to encourage population growth essentially removes only uninfected and unsusceptible crabs and returns all infected and susceptible crabs to the population. This can increase the prevalence of parasites in that population (Kuris *et al.* 1991; Kuris & Lafferty 1992), and the tendency of parasites to be overdispersed in host populations (Shaw *et al.* 1998) would magnify this effect. If transmission is frequency-dependent, low mortality of infected individuals due to fishing could increase transmission rates by increasing prevalence (Fig. 1d). However, if transmission is density-dependent, such fishing would reduce transmission opportunities by reducing host density (Fig. 1e; Packer *et al.* 2003). In either transmission scenario, fishing reduces the overall availability of habitat for parasites. Furthermore, given emerging legal restrictions on discards, the difficulty of diagnosing some parasitic infections in the field, and other considerations, it is probably impossible for most fisheries to target uninfected hosts, even if doing so would increase marketability of the catch.

COMMUNITY PROCESSES

Impacts of fishing can also arise at the community level, either when fisheries directly target multiple taxa or when the effects of fishing one taxon ripple through the community via species interactions. Fishing can either increase or decrease the local abundance and diversity of parasites through its effects on marine communities.

Fishery reduces food web complexity

Theory

When fishing simplifies communities by reducing the abundance, mean size, or mean age of multiple host taxa, it reduces the availability of opportunities for parasite transmission (Fig. 1f). A simplified food web affects directly and indirectly transmitted parasites in different ways. For directly transmitted parasites, food web simplification can reduce host density (see Fishery targets host, reducing host density) or decrease diversity of available alternative hosts (relevant for non-host-specific parasites), reducing transmission efficiency and therefore reducing parasite abundance. Fishing can also increase host density, if the fishery targets the predator or competitor of the focal host (see Fishery targets host's predator or competitor), increasing transmission and parasite abundance. For indirectly transmitted parasites, which require multiple host taxa to complete their life cycles, fishing can create life-cycle bottlenecks by reducing the density of one of the required hosts, thereby reducing transmission efficiency among host species.

Empirical evidence

A few studies have demonstrated a relationship between fishing-driven food web simplification and loss of parasites. In a study of the impacts of fishing on parasites of central Pacific coral reef fishes, Lafferty *et al.* (2008) found that the number of parasite species per host, parasite prevalence, parasite species richness, and overall parasite abundance were higher among fishes at Palmyra Atoll, which has experienced little fishing pressure and retains a relatively intact coral reef food web, compared to heavily fished Christmas Island (Stevenson *et al.* 2007; Sandin *et al.* 2008). Cestodes utilizing sharks as definitive hosts were uncommon at Christmas Island, suggesting that the loss of large, predatory fishes (Myers & Worm 2003; Pauly & Watson 2005; Pauly *et al.* 2005) can be a dimension of food web simplification that is particularly influential for parasites in this system (Lafferty *et al.* 2008).

Marine protected areas paired with fished areas provide excellent 'natural experiments' to test the impacts of fishing, as MPAs tend to relieve only fishing pressures, leaving other anthropogenic pressures, like pollution and climate change,

constant. Along the coast of central Chile, a parasitic trematode (*Proctoeces lintoni*) with an indirect life cycle involving two intermediate hosts (the intertidal mussel *Perumytilus purpuratus* and the keyhole limpet *Fissurella crassa*) and one definitive host (the clingfish *Sicyases sanguineus*) was found to be more abundant in intermediate hosts collected in MPAs relative to hosts collected in areas open to fishing of all three hosts (Loot *et al.* 2005). The difference between protected and unprotected areas was probably due to the relatively greater density of definitive clingfish hosts in protected areas (Hechinger *et al.* 2008). In the French MPA Cerbère-Banyuls, parasite species richness in *Gobius bucchichii* was higher within the protected area than at adjacent, unprotected sites (Sasal *et al.* 1996). The authors suggest that the greater overall species richness of *G. bucchichii* parasites within the MPA was driven in part by the greater size and older age of *G. bucchichii* within the reserve. One indirectly transmitted acanthocephalan parasite of *G. bucchichii*, *Acanthocephaloides propinquus*, was not present outside of the reserve, and a suite of digenetic trematodes (*Helicometra* sp.) were over four times more abundant in *G. bucchichii* within than outside the reserve, suggesting that the greater food web complexity of the MPA has particularly benefitted trophically transmitted parasites. Similarly, Bartoli *et al.* (2005) characterized the distribution of digenean trematode parasites in teleost fish within the no-take Scandola Nature Reserve, off the coast of northern Corsica, France, and compared these results to those from studies of unprotected areas in the Mediterranean and Black Seas, including nearby sites with comparable characteristics along the Italian coast. The authors show that the mean number of digenean species per host species is greater in Scandola, and attribute this difference to the reserve's long history of protection and high diversity of free-living hosts (Bartoli *et al.* 2005). In contrast, Ternengo *et al.* (2009) found no evidence for a relationship between protection status and parasite abundance and diversity among sites with varying protection levels within another Corsican MPA, the Bonifacio Strait Marine Reserve, despite the existence of a more diverse and abundant fish community at the highly protected site. Their results suggest that other factors, such as the identity of the hosts favoured by protection and movement of hosts in and out of reserves, can be more important than food web complexity in driving parasite abundance and distribution (Ternengo *et al.* 2009).

Fishery targets host's predator, competitor, or prey

Theory

Fishing top predators can cause trophic cascades that allow prey populations to expand (Sala 1998; Pinnegar *et al.* 2000; Dulvy *et al.* 2004; Baskett *et al.* 2007). Similarly, competitors of a fished species may respond to the increase in resource availability attending their competitor's removal with an increase in abundance (McClanahan 1992; McClanahan *et al.* 1994; Baskett *et al.* 2007). Jackson *et al.* (2001) suggest that fishing predators and competitors may be the primary cause of increasing infectious disease for low trophic-level species (Fig. 1g). Mathematical models of parasite exchange in a predator–prey framework also suggest that the removal of predators will increase the incidence of parasites in prey. Packer *et al.* (2003) demonstrate that predator removal increases the prevalence of microparasites and macroparasites in prey populations by increasing the number of infected prey individuals and decreasing the number of uninfected prey individuals; this holds true both for microparasites that induce immunity in their hosts (i.e., in SIR models) and for those that can reinfect previously infected hosts (i.e., in SI models). It may also be possible for a fishery to target the prey of a focal host [e.g., harvest of Atlantic menhaden (*Brevoortia tyrannus*), an important prey item of many fishes, including striped bass (*Morone saxatilis*), bluefish (*Pomatomus saltatrix*), tunas, and sharks], causing declines in the predator and, therefore, of its parasites (Fig. 1h). However, given the tendency to 'fish down marine food webs', it is probably uncommon for a prey species to be exploited before its predator.

The loss of one host species through fishing may drive increases in the abundance of its competitors or prey, and this could favour the parasites of these compensating species. If this is the case, fishing might not have a net impact on parasites. Whether compensation offsets declines in the abundance of directly transmitted parasites of the fished species would depend on host specificity, the relationship between parasite richness and trophic level, and the strength of compensation. If the compensating species are closely related to the fished species, they might provide a suitable alternative resource for parasites of the fished species (Fig. 1i). The higher the trophic level of the compensating species, the higher parasite diversity it is likely to support (see Fishery targets infected hosts). The greater the increase in abundance of the compensating species, the more host biomass there would be for parasites to exploit. But these three effects are not likely to combine in such a way as to increase parasitism. Compensation by competitors may provide similar hosts in terms of relatedness and trophic level, but may not replace biomass lost to fishing due to imperfect overlap of resource use. Furthermore, fisheries often target suites of similar species or switch to close relatives after targeted species decline (Myers & Worm 2003; Essington *et al.* 2006), making compensatory increases in competitors a transient phenomenon. Prey released from predation by fished species are unlikely to support the parasites of their predators due to their ecological dissimilarity. In addition, because they feed at a lower trophic level, prey should support a lower diversity and abundance of parasites than their fished predator. Although compensation by prey might lead to a net increase

in host biomass (due to their lower trophic level), studies conducted along spatial gradients of fishing pressure in the Northwestern Hawaiian Islands (Friedlander & DeMartini 2002a) and Northern Line Islands (Stevenson *et al.* 2007; Sandin *et al.* 2008) reveal that, as fishing pressure increases, the relative abundance of lower trophic-level species rises, but overall fish biomass declines dramatically. Though it is unclear whether insufficient compensation or fishing pressure is depressing the biomass of species at low trophic levels, the pattern strongly suggests that overall availability of habitat for parasites (i.e., fish biomass) will tend to decline as fishing pressure increases, resulting in a lower overall abundance and diversity of parasites at the community level. This suggests that compensatory increases of alternative hosts will have either neutral or negative impacts on parasites (Fig. 1i).

Empirical evidence

There are numerous empirical examples of change in the abundance of parasites of the prey and competitors of fished species. The first comes from the largest epizootic known for any marine animal – the 1983–1984 die-off of the long-spined sea urchin, *Diadema antillarum*, in the Caribbean and western Atlantic, which resulted in a 98% reduction in urchin abundance throughout the species' New World range (Lessios 1988). *Diadema antillarum* reached extremely high densities on Caribbean coral reefs before this mortality event, probably due to intense overfishing of its predators (e.g., balistid, sparid and batrachoidid fishes) and competitors (e.g., scarid and acanthurid fishes; Hay 1984; Lessios 1988; Hughes 1994). The resulting increase in urchin density may have facilitated transmission of the waterborne pathogen that caused the die-off (Jackson *et al.* 2001). However, these urchin die-offs were not locally density-dependent at the single site where density dependence was tested, making it difficult to assess the hypothesis that enhanced urchin density drove the disease outbreak (Hunte *et al.* 1986). A clearer case for fishing-mediated increases in disease comes from a study of the purple sea urchin, *Strongylocentrotus purpuratus*, in the kelp forests of the California Channel Islands. Fishing-driven declines of the predators of *S. purpuratus* (e.g., large spiny lobster *Panulirus interruptus* and sheephead *Semicossyphus pulcher*), in combination with the non-natural absence of sea otters in the Channel Islands, allowed urchin populations to grow quickly until, in the early 1990s, an unidentified pathogen caused widespread, density-dependent mortality (Lafferty 2004). In another example from the Channel Islands, the black abalone (*Haliotis cracherodii*) attained extremely high abundances over the course of the twentieth century due to elimination of predation by sea otters and reductions in the abundance of other predators (e.g., spiny lobster, sheephead; Lafferty & Kuris 1993). A *Rickettsia*-like prokaryote pathogen emerged and began to spread in 1985 through these dense populations, causing a lethal condition called withering syndrome that nearly drove the black abalone to extinction and eliminated the fishery (Lafferty & Kuris 1993; Altstatt *et al.* 1996). In other words, it seems plausible that high density was favourable to the concurrent development of the fishery and the emergence of disease.

Targeting the predator of a given host can also cause a decrease in the abundance of its parasites, if the predator serves as the definitive host for an indirectly transmitted parasite shared with the prey species (Fig. 1f). This pattern was observed in the previous example of shark fishing reducing cestodes in reef fishes (Lafferty *et al.* 2008), and is underscored by observations that increased cestode parasitism in demersal fishes can occur in areas where sharks aggregate in response to feeding by tourists (Vignon *et al.* in press). For an additional example, fishing of pike (*Esox lucius*) in a Norwegian lake reduced the abundance of the cestode parasite *Triaenophorus crassus* in the pike's primary prey, whitefish (*Coregonus* spp.; Amundsen & Kristoffersen 1990). This occurred even though pike do not transmit parasites directly to whitefish; instead, parasite larvae shed by the pike must first infect copepods, which whitefish then ingest.

VARIABILITY IN THE RESPONSE OF PARASITES TO FISHING

Though we have assumed that parasite species will generally respond to fishing pressure in similar ways, the substantial variability in life history traits among parasites is likely to influence these responses. Perhaps most importantly, degree of host specificity will determine how strongly the fate of a parasite population is tied to that of its host. Parasites specializing on fished species should be particularly vulnerable in this respect, as should specialists on the predators of fished species, which are likely to dwindle alongside their prey. Meanwhile, specialist parasites of the competitors and prey of fished species should increase alongside their hosts. Lafferty & Holt (2003) used simple SIR models to investigate the effects of reducing host abundance on parasites; they found that such reductions decreased rates of parasitism (R_0), but that this effect was more pronounced for host-specific parasites, because coupling of host and parasite abundance tends to be stronger when the parasite is exclusively dependent on one host (Lafferty & Holt 2003).

In contrast, fishing should not impact generalist parasites if suitable hosts experience a compensatory increase in abundance (Fig. 1i; see Fishery targets host's predator, competitor, or prey; Lafferty & Gerber 2002). If we consider a 'generalist' strategy to include not only parasites that can exploit alternative hosts, but also those that make

use of paratenic hosts, an excellent example comes from nematode 'sealworm' parasites (*Pseudoterranova decipiens*) of cod (*Gadus morhua*) in Norwegian coastal waters. Rates of parasitism among cod of this region have remained stable despite the fish's dramatic decline, probably due to the role of small, untargeted benthic species (e.g., sculpin) as suitable paratenic hosts for the parasite (Andersen *et al.* 1995). Therefore, the fates of specialist parasites should be tied tightly to the fates of their hosts' populations, whereas generalist parasites will be freed from this constraint if they can use hosts unaffected or positively affected by fishing.

Though compensatory increases in hosts other than the fished host can allow some parasite populations to maintain or even increase their abundance (depending on their host and degree of host specificity), fishing is predicted to negatively affect both specialist and generalist parasites at the community level. Fishing pressure depresses the mean trophic level of fish assemblages (Pauly *et al.* 1998; Myers & Worm 2003; Pauly & Watson 2005), the mean size of exploited species (Pauly & Watson 2005; Pauly *et al.* 2005), and the mean size and age of fishes within exploited species (Bianchi *et al.* 2000; Friedlander & DeMartini 2002b; Jennings & Blanchard 2004; Pauly *et al.* 2005; Sala & Knowlton 2006; McClenachan 2008). Fishers therefore seek out precisely the high trophic level (Lafferty *et al.* 2006), large-bodied (Guegan & Hugueny 1994; Arneberg 2002), old (Pacala & Dobson 1988; Hudson & Dobson 1995; Lo *et al.* 1998; Zelmer & Arai 1998; Poulin 2000) fishes and invertebrates that are the most heavily parasitized species, populations, and individuals in marine communities (see Fishery targets host based on infection status). Fishing out these individuals should reduce the availability of prime parasite habitat – and therefore parasite abundance and diversity – at the community level, for both specialist and generalist parasites. Even compensatory increases of non-target host species may fail to 'rescue' the parasites of fished species, in part because fisheries switch to non-target species in response to fishing-driven scarcity of the original target (Friedlander & DeMartini 2002a; Myers & Worm 2003; Essington *et al.* 2006; Stevenson *et al.* 2007; Sandin *et al.* 2008), resulting in reduced overall availability of habitat for parasites (i.e., fish biomass) with increasing fishing pressure.

SYNTHESIS

The effects of fishing on the abundance of marine parasites are likely to be complex. However, the existing evidence suggests that, on balance, fishing-driven change in fish community structure will act to reduce the abundance of parasites (Table 1). Even without linear density-dependent transmission, the severe reductions in host density caused by fishing should reduce transmission of parasites among remaining fish hosts. Reductions in host density will occur at some spatial scale [e.g., reduction in population density (Fig. 1a), reduction in density of populations by fragmentation (Fig. 1b)] wherever the abundance of a stock is declining due to fishing pressure; such declines have been

Table 1 Summary of studies cited herein, which were chosen by exhaustive search of the literature for papers addressing the impacts of exploitation- (fishing- and hunting-) driven changes in host community structure on parasites

		Impact on parasites			
Level	Fishery…	−	0	Equivocal	+
Population	…targets host	**7**[(1–7)]	0	**3**[(8–10)]	0
	…targets infected hosts	**2**[(11–12)]	0	0	0
	…targets uninfected hosts	0	0	0	**1**[(13)]
Community	…reduces food web complexity	**4**[(14–17)]	**1**[(18)]	0	0
	…targets host's prey	0	0	0	0
	…targets host, with compensatory increase in alternative/paratenic host	0	**1**[(19)]	0	0
	…targets host's predator or competitor	**2**[(20–21)]	0	0	**2**[(22–23)]

References: [1]**Black 1983, 1985**; [2]Dobson & May 1987; [3]**Amundsen & Kristoffersen 1990**; [4]Lafferty & Holt 2003; [5]**Ward & Lafferty 2004**; [6]McCallum *et al.* 2005; [7]**Stokesbury *et al.* 2007**; [8]Choisy & Rohani 2006; [9]Bolzoni *et al.* 2007; [10]Holt & Roy 2007; [11]**Sanders & Lester 1981**; [12]Packer *et al.* 2003; [13]Kuris & Lafferty 1992; [14]**Sasal *et al.* 1996**; [15]**Bartoli *et al.* 2005**; [16]**Loot *et al.* 2005 and Hechinger *et al.* 2008**; [17]**Lafferty *et al.* 2008**; [18]**Ternengo *et al.* 2009**; [19]**Andersen *et al.* 1995**; [20]**Lawler 1970**; [21]**Amundsen & Kristoffersen 1990**; [22]**Lafferty & Kushner 2000 and Lafferty 2004**; [23]Packer *et al.* 2003.

For empirical studies, we limited this search to marine and freshwater ecosystems. Table reports the number of studies documenting an increase, decrease, no change, or equivocal change (i.e., a change whose direction depends on the intensity of the fishery impact) in parasite intensity, prevalence, or diversity for various impacts of exploitation at the population and community levels. We consider one study to be equal to one biological example, and multiple papers on the same parasite in a single host at a single location were therefore counted as one study. Empirical studies are highlighted in bold in the references list.

documented all over the world for many fisheries (Jackson *et al.* 2001; Pauly *et al.* 2002) and can be severe (e.g., 90% reduction in density of large predatory fishes globally; Myers & Worm 2003). Another well-documented effect of fishing, removal of the largest, oldest, highest trophic level, and therefore most parasite-laden individuals, causes direct mortality of parasites and reduces the capacity of the remaining parasite population to infect new hosts (Fig. 1c). The tendency to preferentially target large, high trophic-level individuals and species is ubiquitous (Pauly & Watson 2005), resulting in substantial reductions in mean trophic level and body size, which erode habitat quality and availability for parasites. Conversely, targeting of uninfected fishes, which could increase the relative abundance of infected individuals in a population (Fig. 1d), appears to be limited to the few fisheries in which diagnosis of infection is possible before processing of the fish. Even when such selective take does occur, it still reduces the overall density of hosts, thereby eliminating any potential positive effect on parasites with density-dependent transmission (Fig. 1e).

Community-level effects of fishing also appear to have generally negative implications for marine parasites. By reducing the overall abundance and diversity of available hosts, fisheries reduce the number of pathways in marine food webs, eroding the trophic interactions required by many indirectly transmitted parasites to complete their life cycles (Fig. 1f). However, the effects of fishing on the parasites of a particular host will depend on that host's relationship to the target species. Fishing the prey of a given host is likely to reduce its population's abundance, density, and therefore the abundance of its parasites (Fig. 1h), but given the tendency to 'fish down marine food webs', it is probably unlikely for a prey species to be exploited before its predator. Released from competitive or predation pressure, alternate hosts can experience compensatory increases in abundance, providing a refuge habitat for parasites of a fished host (Fig. 1i). But even if the increase in biomass of these alternative hosts exceeds the original host biomass lost, the pattern of 'fishing down marine food webs' will still drive community-level reductions in mean trophic level, size, and age of hosts, reducing the availability of prime parasite habitat. Furthermore, in the few studies that have so far been conducted (Friedlander & DeMartini 2002a; Sandin *et al.* 2008), such compensation does not appear to counteract the trend towards lower overall fish biomass at higher levels of fishing pressure.

The one mechanism by which fisheries have been reliably shown to increase the abundance of parasites – inducing increases in host density by targeting predators and competitors of the host (Fig. 1g) – could be a transient outcome if fisheries eventually switch to the lower trophic-level hosts released from predation. The available evidence therefore suggests that fishing-induced declines in communities of free-living organisms generally go hand-in-hand with declines in parasite communities.

CONCLUSION

To determine definitively whether we are 'fishing out marine parasites' requires several lines of evidence. If the impact of fishing on parasites is strong enough to swamp other drivers of parasite community structure, a change in rates of parasitism should be detectable among fished (and possibly unfished) species as fishing's impacts accumulate through time. To test for temporal change in parasite communities, continuous records of parasitic infection in fished and unfished stocks could be sought from museum specimens or historical records; alternately, historical records of parasitic infection or museum specimens could be a 'parasite baseline' against which contemporary rates of parasitism may be compared. To tie any temporal change in parasites to the influence of fishing and test whether fishing-induced changes in fish community structure are sufficient to drive shifts in parasites, parasite communities should be compared along existing spatial gradients of fishing pressure ('space-for-time substitution'; Pickett 1989). These gradients could range between sites that have never been fished to sites that are heavily fished and from no-take marine reserves to fished areas. A large-scale, large-fish exclusion experiment would further elucidate mechanisms for the relationship between fishing-induced change in fish community structure and parasitism. If such a relationship is established, the abundance and diversity of parasites could provide a useful bioindicator of overfishing (Box 1).

Although many may rejoice at news of declining rates of parasitism among the commercially exploited fishes consumed by humans, there has been growing recognition in recent years that parasites are an influential part of biodiversity and that their loss could have wide-ranging ecological implications. A substantial body of literature suggests that parasites can regulate host populations and influence the structure of communities in which those populations are embedded (Dobson & Hudson 1986; Price *et al.* 1986; Minchella & Scott 1991; Combes 1996; Poulin 1999; Hudson *et al.* 2006). Parasites can sometimes influence marine ecosystems (Thomas *et al.* 1998; Mouritsen & Poulin 2002b; Wood *et al.* 2007), but existing examples are drawn primarily from unfished, intertidal, invertebrate hosts. Work in estuaries of the west coast of North America indicates that parasites affect the structure (nestedness and connectance) of estuarine food webs (Lafferty *et al.* 2006) and play a major role in the flow of energy in those systems (Kuris *et al.* 2008). However, the extent to which parasites regulate fish populations and thereby affect coastal and pelagic marine communities and ecosystems remains unknown. Scattered examples of strong host population regulation

Box 1 Parasites as indicators of overfishing

Can we use declines in parasites to detect overfishing? Snail–bird host combinations make particularly tractable study systems and provide much of our evidence for parasites as indicators. For example, Byers *et al.* 2008 found a strong positive correlation between local trematode prevalence in the common periwinkle, *Littorina littorea*, and the abundance of bird definitive hosts over the entire New England coast. The species richness and abundance of bird definitive hosts is a strong predictor of trematode abundance and richness in the California horn snail, *Cerithidea californica* (Huspeni & Lafferty 2004; Hechinger & Lafferty 2005). The large fishes preferentially targeted by industrial fisheries (Bianchi *et al.* 2000; Friedlander & DeMartini 2002b; Jennings & Blanchard 2004; Pauly *et al.* 2005; Sala & Knowlton 2006; McClenachan 2008) generally serve as definitive hosts in the life cycles of indirectly transmitted parasites, suggesting the bird–trematode model could be extended to monitor the effects of fishing. For instance, Lafferty *et al.* (2008) argue that the prevalence of larval cestodes in reef fishes can be a sensitive indicator of shark abundance, a relationship supported by spatial associations between sharks and cestodes in demersal fish (Vignon *et al.* in press). Increases in parasitism can also be associated with protection of formerly fished sites (see Fishery reduces food web complexity), indicating that restoration of fish communities may restore associated parasite communities. However, increases in parasitism can also occur when food webs are disrupted, with parasites filling the gaps left by overfished predators (see Fishery targets host's predator or competitor).

exist; for example, *Ichthyophonus hoferi* has caused several population crashes of Atlantic herring (*Clupea harengus*; e.g., Patterson 1996). If parasites of marine fishes play roles similar to those established for parasites of other taxa and ecosystems, their 'fishing out' could have important indirect effects on marine communities. Because the impacts of large-scale, industrial fishing have been accumulating for more than half a century, these changes may already be well underway.

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SIDE BAR: DEFINITIONS

Alternative host – a host in which a parasite can accomplish the development it typically undergoes in its regular host.

Definitive host – the host in which sexual reproduction of an indirectly transmitted parasite takes place.

Directly transmitted parasites – parasites that only require a single host species to complete their life cycle.

Indirectly transmitted parasites – parasites with life cycles involving hosts of multiple species, usually transmitted only between and not within host species.

Intermediate host – all hosts in the life cycle of an indirectly transmitted parasite other than the definitive host.

Life-cycle bottleneck – a limitation on overall parasite abundance (i.e., in all host species) arising from limited availability of one host species in the life cycle of an indirectly transmitted parasite.

Limiting host – the host whose limited availability creates a life-cycle bottleneck for its indirectly transmitted parasite.

Macroparasite – 'typical parasites', or those for which the impact of infection on the host increases in severity with the number of independent infection events; includes some parasitic helminths and copepods.

Microparasite – 'pathogens', or organisms for which the impact of infection on the host is generally unrelated to the number of independent infection events; includes some fungi, bacteria, protozoa and viruses.

Mean parasite abundance – mean number of parasites in each host individual.

Parasite intensity – number of parasites in an infected host.

Parasite prevalence – per cent of host individuals infected.

Paratenic host – a host in which a trophically transmitted parasite undergoes no development but can remain infective. Infection of paratenic hosts may allow a parasite to bridge an ecological gap between required hosts (e.g., if the life cycle requires a copepod and a large, piscivorous fish, a small, zooplanktivorous fish paratenic host may facilitate transmission) or to survive predation on an intermediate host by a non-required host.

R_0 – the basic reproductive rate of a parasite. For a microparasite, this is the expected number of new infections expected to arise from the entry of a single infected host to an otherwise uninfected host population. For a macroparasite, this is the average number of expected successful parasite offspring from a parasite in a host immigrating to an otherwise uninfected host population. R_0 is a meaningful measure of parasite success because it represents the ability

of a parasite to invade a host population. It is a convenient measure because the limited conditions it represents (hosts at carrying capacity and all hosts susceptible) lead to simplified calculations.

SI model – a mathematical model that divides the host population into two classes (susceptible and infective) and tracks the size of each class over time. Used to model diseases in which prior infection does not confer host resistance.

SIR model – a mathematical model that divides the host population into three classes (susceptible, infective, and resistant) and tracks the size of each class over time. Used to model disease in which prior infection confers host resistance.

Threshold density for parasite transmission – the minimum number of hosts necessary for a parasite to establish itself in the host population or the number of hosts at which the rate of infection of new hosts just equals the rate of loss of infected hosts due to mortality or recovery from infection.

Trophically transmitted parasites – indirectly transmitted parasites that are passed from prey to predator during predation events.

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SUPPORTING INFORMATION

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Appendix S1 Development and interpretation of Figure 2.

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