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Forum

Survival of the sickest: selective predation differentially modulates ecological and evolutionary disease dynamics

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Predators and parasites are critical, interconnected members of the community and have the potential to shape host populations. Predators, in particular, can have direct and indirect impacts on disease dynamics. By removing hosts and their parasites, predators alter both host and parasite populations and ultimately shape disease transmission. Selective predation of infected hosts has received considerable attention as it is recognized to have important ecological implications. The occurrence and consequences of preferential consumption of uninfected hosts, however, has rarely been considered. Here, we synthesize current evidence suggesting this strategy of selectively predating uninfected individuals is likely more common than previously anticipated and address how including this predation strategy can change our understanding of the ecology and evolution of disease dynamics. Selective predation strategies are expected to differentially impact ecological dynamics and therefore, consideration of both strategies is required to fully understand the impact of predation on prey and host densities. In addition, given that different strategies of prey selectivity by predators change the fitness payoffs both for hosts and their parasites, we predict amplified coevolutionary rates under selective predation of infected hosts compared to uninfected hosts. Using recent work highlighting the critical role that predators play in disease dynamics, we provide insights into the potential mechanisms by which selective predation on healthy individuals can directly affect ecological outcomes and impact long-term host–parasite coevolution. We contrast the consequences of both scenarios of selective predation while identifying current gaps in the literature and future research directions.

Keywords: host–parasite, coevolution, predation, selective predation, disease dynamics

Introduction

Parasites are a critical component of many ecological communities and can shape host populations and alter community structure (Minchella and Scott 1991, Grenfell and Dobson 1995, Zuk et al. 1997). At the same time, other community members such as competitors (Holt et al. 2003) and predators (Ives and Murray 1997, Packer et al. 2003) can limit or promote establishment and persistence of a parasite in a host

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population. Predators, in particular, can influence disease dynamics directly by reducing the abundance of both susceptible and infected hosts (Packer et al. 2003, Orlofske et al. 2014). Additionally, when consuming their prey, predators indirectly alter parasite populations by consuming their prey's parasites (Ives and Murray 1997). These direct and indirect mechanisms can result in changes in both host and parasite population dynamics and can ultimately shape disease transmission.

The effects of predation on disease transmission have been investigated using theoretical (Ostfeld and Holt 2004, Hall et al. 2005) and experimental approaches (Lafferty 2004, Duffy et al. 2005, Johnson et al. 2006). Attention has been focused on the potential for predators to alter the effects of parasites on host population dynamics by selectively predating infected hosts (Packer et al. 2003, Malek and Byers 2016, Lopez and Duffy 2021). This approach assumes that infected individuals are infectious and symptomatic and we will also make that assumption. Infected prey thus suffer from energetic, cognitive or other physiological costs that result in reduced predator avoidance or escape ability (Decaestecker et al. 2002), accordingly predators are modeled to preferentially consume infected individuals (Schaller 1972, Moore 2002). Such increased vulnerability to predation due to parasite-induced morbidity is widespread (Moore 2002, Hatcher et al. 2006, DeBlieux and Hoverman 2019). In these cases, however, predators are not actively selecting infected prey. Rather, consumption of infected prey is a consequence of the negative effect of the parasite on the host/prey.

Although selective predation on infected prey has attracted considerable attention, cases of predators preferentially selecting uninfected prey are less often considered (Sloan and Simmons 1973, Schlichter 1978). Such preferential consumption of uninfected prey is likely more common than is currently assumed. While limited, there is evidence that some predators have the ability to discriminate and avoid infected prey thus reducing their likelihood of infection (Hamilton and Zuk 1982, Jones et al. 2005, Meyling and Pell 2006). There is an array of opportunities for selective predation on healthy hosts that can modulate disease dynamics (Holt and Roy 2007, Toor and Best 2015, Vitale and Best 2019).

Disease outcomes are shaped by selective pressures imposed on the predator, parasite and host which, depend on their particular ecological strategies. For example, parasites are expected to be selected to manipulate the behavior of their hosts such that the latter become more conspicuous to predators who can serve as the next host in their lifecycle, but to conceal their hosts from predators who are a dead-end to the parasite (Moore 2002). Palatable prey, for instance, are not expected to opt for traits that increase their conspicuousness to predators, but uninfected individuals may be conspicuous as a by-product of selection in other contexts (e.g. mating). Similarly, for predators that may be infected by consuming infected prey, selective predation on healthy individuals is expected to evolve. Disease dynamics thus result from complex interactions among species specific strategies from all of those ecological players.

Here, we identify the characteristics of hosts, parasites and predators likely to yield a significant role for predators to influence disease dynamics. In particular, we discuss the mechanisms by which differential predation for infected versus uninfected individuals may occur and address the ecological and coevolutionary consequences of those strategies. While predation, in general, decreases host densities, the ecological and evolutionary outcomes of host–parasite dynamics may be quite different under different selective predation strategies. Integrating hypotheses from both disease ecology and behavioral ecology, we provide insights into the effects of predation strategies in modulating disease dynamics and highlight future research directions.

Selective predation on infected individuals

Support for predators selectively predating infected, possibly weakened and easier to catch prey, has been widely recognized in experimental studies in which parasite-induced morbidity increases the host's risk of predation (Schaller 1972, Moore 2002). For example, snowshoe hares *Lepus americanus* treated with antihelminthics are less likely to be attacked by predators than untreated, infected individuals (Ives and Murray 1997). In water fleas, *Daphnia* spp., multiple lines of evidence show that predatory fish preferentially feed on prey infected with the chytrid fungus *Polycaryum laeve*. Similarly, fungal infections make zooplankton opaque and conspicuous to certain fish predators, increasing predation risk for both the host and parasite (Duffy and Hall 2008, Johnson et al. 2010). Selective culling of infected individuals due to fish predation ultimately reduces infection prevalence in zooplankton populations (Hall et al. 2005, 2010). Experimental mesocosms, for instance, revealed that infected *Daphnia* are consumed two to five times more frequently than healthy individuals of similar size (Galbraith 1967). Field studies further validate that yellow perch impose stronger selection against infected *Daphnia* (Johnson et al. 2006). Thus, parasitic infections can decrease the effectiveness of anti-predator strategies, resulting in higher predation pressure for infected prey.

Predators can also selectively consume infected individuals due to higher encounter probabilities with such prey rather than a particular preference for attacking susceptible individuals. This pattern can arise from either host behavioral changes due to parasite manipulation (Moore 2002, Lefèvre and Thomas 2008) or pathological alterations as a result of infection. In parasites with complex life cycles, manipulation of intermediate hosts to increase predation risk is common resulting in such predation increasing transmission to the final host (Moore 2002, Poulin 2010). Likewise, parasite-induced changes in host pathology can increase the probability of predation for infected individuals. For instance, the abundance of red grouse *Lagopus lagopus* and their fox predators, is influenced by a parasitic nematode *Trichostrongylus tenuis* that exploits red grouse as their host (Hudson et al. 1992). Robust evidence that foxes selectively prey on heavily infected

grouse comes from comparisons among temporal and spatial variation in fox hunting intensity. In areas where gamekeepers control fox abundance, red grouse exhibit seasonal cyclic fluctuations in population density predominantly driven by a reduction in host fecundity resulting from the parasitic infection (Hudson et al. 1998). If the number of predators is allowed to increase by removing gamekeepers, however, the abundance of grouse is less likely to oscillate (Hudson et al. 1992). Once predator control methods are resumed, oscillations in red grouse abundance also resume. Comparisons between sites with and without gamekeepers show increases in nematode infection prevalence in grouse with decreased predation pressure due to fox hunting (Hudson et al. 1992). Similarly, infections of *Spirobacillus cienkowskii*, a bacterial parasite that infects *Daphnia dentifera*, display infectious epidemics consistent with seasonal changes in predation rates (Duffy et al. 2005). The evidence from these studies supports the role of predation in decreasing disease prevalence within host populations.

Regardless of the mechanism (parasite manipulation or pathological effects), as predators eliminate infected individuals from the prey population, they limit the spread of disease (Packer et al. 2003) (Fig. 1a). Following the robust evidence suggesting infected individuals are more likely to be predated over uninfected prey in many systems (Hatcher et al. 2006), a growing number of theoretical models and empirical studies have investigated the relationship between predation and infection prevalence in this context. Among the proposed conceptual frameworks outlining the potential effects of predation on disease dynamics, the 'healthy herds' hypothesis has received the most attention (Packer et al. 2003). Correlative evidence of predators reducing the prevalence of disease within a host population is abundant. Additionally, several models have provided insights into the role selective predation plays in modulating population densities and therefore disease outcomes in their prey population (Hall et al. 2005, Duffy and Hall 2008, Vitale and Best 2019).

Selective predation on healthy individuals

We propose that preferential predation of uninfected prey, while less often discussed and assumed to be limited, is likely common and could occur under different conditions. On one hand, it could be the result of predators who can detect and avoid consuming infected prey. On the other hand, preferential predation of uninfected prey could result from predators

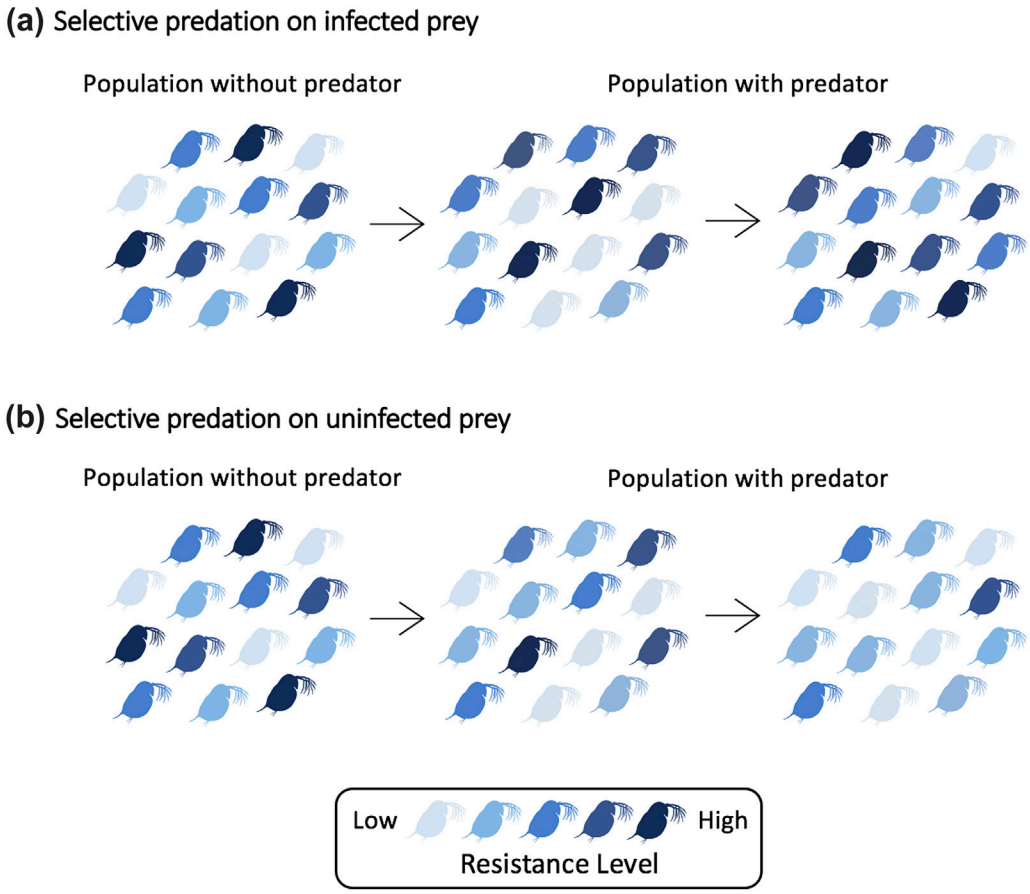

Figure 1. Representation of the effect of selection predation on infected (a) and uninfected (b) prey. In addition to a reduction in host population density, selective predation results in different changes in allele frequency of host resistance. Coevolutionary rates are expected to increase with selective predation on infected prey (a) and decrease with selective predation on healthy prey (b).

consuming individuals who, due to their healthy status, display traits that make them more likely to be detected and increase encounter rates. The outcome in both cases results in consumption of healthy prey. While in the first case predators actively avoid infected individuals, in the second one, predators preferentially attack healthy prey. Below we discuss both scenarios.

Avoidance of infected prey requires external cues of parasitism and a high cost of infection for the predators. The cues may be a non-adaptive side effects of infection or may occur when infected prey carry specialized parasites who exploit predators as the next host in their lifecycle. These parasites may be able to manipulate their intermediate host to increase transmission to final hosts. To reduce infection risk and costs, final hosts may be under selection to avoid infected prey. Along these lines, predators that have evolved with the parasite of an intermediate host consume fewer infected prey compared to a naive predator (Sheath et al. 2018). For example, *Anthocoris nemorum*, the common flowerbug, a generalist predator that can be infected by *Beauveria bassiana*, a fungal pathogen transmitted by foraging on plants with the fungus, can detect and avoid infected plants (Meyling and Pell 2006). These predation strategies can reduce infection risk and costly consequences for the predator.

Predators often use visual cues, such as prey size, to modulate their foraging strategy. For instance, red-winged blackbirds *Agelaius phoeniceus* in captive feeding trials prefer to forage on army-worms *Spodoptera frugiperda* that are not parasitized by an ectoparasitoid wasp *Euplectrus plathypenae* as non-infected worms grow larger in size compared to infected army-worms (Jones et al. 2005). Similarly, black-capped chickadees *Parus atricapillus* showed a preference for foraging upon larger galls on Canada goldenrod *Solidago canadensis* that are not parasitized by mordellid beetle larvae (Schlichter 1978). In these cases, however, it is unclear if predators are avoiding infected prey, choosing higher reward prey due to their larger size, or both. While detection of infected prey has been described mainly in systems where infection results in changes in prey-emitted visual cues, other sensory modalities are likely to provide information about infection status.

Alternatively, selective predation of healthy individuals could result from lower prey encounter rates with infected individuals due to reduction of host activity associated with infection. Vector-borne diseases, for example, often elicit symptoms that reduce host mobility (Moore 2002) and can result in increased predation rates of uninfected individuals. Consistent with this prediction, infection with avian malaria *Plasmodium* in blue tits *Cyanistes caeruleus* is associated with lower recapture rates than uninfected birds (Lachish et al. 2011). To our knowledge, however, few studies have examined the role of predators at modulating host and vector born disease dynamics (Møller and Nielsen 1997). Similarly, pathological consequences of infection can dampen traits that increase prey detectability to natural enemies. This phenomenon has been shown in Eastern tree hole mosquito larvae *Aedes triseriatus* infected with gregarines *Ascogregarina barretti* (Jones et al. 2016). While this parasite alone does not affect mortality of this mosquito larvae, in the presence of the predatory larval, elephant mosquito *Toxohrynchites rutilus*, uninfected individuals experience higher mortality rates. Compared to uninfected larvae, infected larvae are less active and thus less likely to be detected by predators. Unlike cases of host manipulation, parasite-induced changes in these scenarios are side effects of infection and not adaptive physiological changes mediated to promote the transmission of the parasite.

A common scenario that also results in selective predation of healthy individuals is the production of conspicuous mating signals. It has long been proposed that individuals with more conspicuous mating signals are less likely to be infected by parasites as a result of heritable resistance (known as the Hamilton–Zuk hypothesis (Hamilton and Zuk 1982). Although evidence supporting this hypothesis is inconclusive (Balenger and Zuk 2014), in multiple systems there is decreased infection prevalence among individuals who produce conspicuous signals and such individuals suffer higher predation rates. For example, parasitic infections reduce the degree of expression of carotenoid colors therefore reducing the conspicuousness of male sexual signals in Trinidadian guppies *Poecilia reticulata* (Houde and Torio 1992). Male guppies displaying more conspicuous signals are more likely to be approached, attacked and captured by predatory blue acara cichlid fish *Aequidens pulcher* (Godin and McDonough 2003). Given that individuals with more conspicuous signals are more easily detected by predators (Endler 1991), which may occur due to their non-infected status, this system is a prime example of selective predation on healthy individuals. Investigating similar systems with conspicuous signals, focusing on those modified by parasitic infection (Jones et al. 2016), would provide a better estimate of the frequency of selective predation on healthy individuals. The Hamilton–Zuk hypothesis has received considerable attention in behavioral ecology (Balenger and Zuk 2014), but exploration of its role in altering host–parasite dynamics has been limited. We propose that this scenario of eavesdropping predators selectively removing healthy (presumably uninfected) individuals represents a potentially important mechanism that can affect host–parasite dynamics. Theoretical and empirical approaches that assess the impact on parasite prevalence and virulence when predators eavesdrop on prey mating signals would provide valuable insights into the consequences of these interactions on disease dynamics. This type of selective predation, for instance, is likely to alter frequency-dependent host–parasite dynamics during the breeding season of the host. In some species thus, in- and out-of-breeding season periods may result in alternations between different types of selective predation ultimately shifting short-term temporal disease dynamics. One mathematical model has considered switches in selective predation during the lifetime of the host (Mukhopadhyay and Bhattacharyya 2009) but to our knowledge, no observational or experimental studies have considered the impact on parasite population dynamics.

Predators that disproportionately prey upon healthy prey, as discussed above, could ultimately increase disease prevalence of prey populations (Fig. 1b). In general, predation decreases host densities resulting in fewer opportunities for

disease transmission. However, predators that exhibit a preference for uninfected prey could alter the ratio of infected to uninfected individuals within a population, increasing the probability of susceptible individuals encountering parasites, ultimately raising transmission rates. Additionally, selective predation of healthy individuals is expected to change the cost of infection imposed on the host. Infected prey would have a lower risk of predation and thus reduced predation mortality rates compared to healthy individuals. We thus suggest that the benefit of increased survival due to reduced predation may, under some circumstances, partially offset the fitness cost of a parasitic infection.

Coevolutionary consequences of selective predation

In the previous section we highlighted how selective predation of healthy individuals versus infected individuals can result in contrasting ecological outcomes. Here we discuss the role that selective predation plays in long-term, evolutionary disease dynamics and how their disparate disease outcomes are expected to alter host–parasite coevolutionary dynamics. Selective predation may result in coevolutionary changes in host resistance and parasite virulence but the diverse consequences of removal of infected or uninfected hosts have not been fully explored (but see Best 2018). Different predation strategies have the potential to shape the evolutionary consequences of selective predation on host–parasite interactions.

Equivalent to the mechanisms proposed by the 'healthy herds' hypothesis addressing predation on infected individuals, a reduction of both host and parasite population densities would be expected. The combination of an increase in host resistance, increased predation of infected hosts and a decrease in host and parasite densities could lead to evolutionary changes in parasite populations. As result of the now-shorter host life span and thus a shorter infectious period, an increase in the selective pressure for parasites with the highest transmission potential would be expected (Poulin and Combes 1999, Combes 2001, Krist et al. 2004). Such selection for increased parasite transmission would in turn increase selection for higher host resistance, which could increase the frequency of host resistance alleles. In the same way that the prevalence of parasites selects for increased host resistance (Minchella 1985), selective predation of infected individuals is expected to lead to an increase in selection intensity in the frequency of alleles for host resistance. Ultimately, the increase selective pressures on both the parasite and host would amplify the rate at which they coevolve in response to one another (Fig. 2a). While the link between parasite virulence and disease transmission remains unclear (Williams et al. 2014, Ben-Ami 2017, Ben-Shachar and Koelle 2018), there is strong evidence for a positive relationship between within host replication and virulence, as well as between replication and transmission (Acevedo et al. 2019, Turner et al. 2021). Reduced infectious periods, as a result of selective predation on infected individuals, is thus expected to increase the replication rate of parasites, amplifying the strength of selection between parasites and their hosts. Yet, in *Daphnia*, some theoretical models suggest parasite virulence may not increase in all systems due to the joint effect of selective predation and rapid host evolution (Hall et al. 2007, Duffy and Hall 2008, Best 2018). In general, there is limited clarity about how the different direct and indirect effects of consumption of infected individuals interact with disease dynamics over evolutionary time. Experimental studies examining the effect of this predation pattern on the coevolution between hosts and parasites are needed to further clarify allele frequency dynamics associated with parasite virulence and host resistance.

Given the distinct patterns between selective predation on infected versus healthy individuals, we predict changes in rates of host–parasite coevolution. If a predator selectively removes healthy individuals from the population, the number of resistant hosts is also expected to be reduced. Meanwhile, infected prey would experience a decrease in predation-induced mortality resulting in an extended time for parasite transmission. Under these circumstances, the reduced cost of infection for the host is expected to decrease selection for host resistance alleles and therefore, reduce the selective pressure for higher virulence in parasites. Accordingly, we predict that the combination of selective predation on uninfected individuals and the subsequent relaxed selection for host resistance and parasite transmission would dampen the rate of coevolution between hosts and their parasites (Fig. 2b). The net costs of infection and evolutionary outcomes likely differ when predators selectively avoid infected prey versus when eavesdroppers are preferentially attracted to uninfected prey. Estimating the net cost of infection, however, may be difficult in systems where predation costs are linked to mating costs that may be suffered due to the production of less conspicuous mating signals (e.g. reduced mate attraction). We identify specific research avenues that can improve our understanding of the way selective predation impacts disease dynamics.

Studies investigating the impact of predators on disease have generally been conducted using systems where infected individuals experience increased predation pressure (Schlichter 1978, Ives and Murray 1997, Moore 2002, Johnson et al. 2006). Thus, the potential for predators to decrease the number of infected hosts and reduce parasite transmission has been well-documented (Combes 2001, Moore 2002). Studies focusing on host and parasite changes under selective predation of healthy individuals will be critical for a comprehensive understanding the mechanisms by which predators ultimately alter host–parasite dynamics.

Towards understanding how selective predation modulates host–parasite dynamics

Here we identify specific venues of research that need particular attention to move the field forward at understanding the way selective predation impacts disease dynamics.

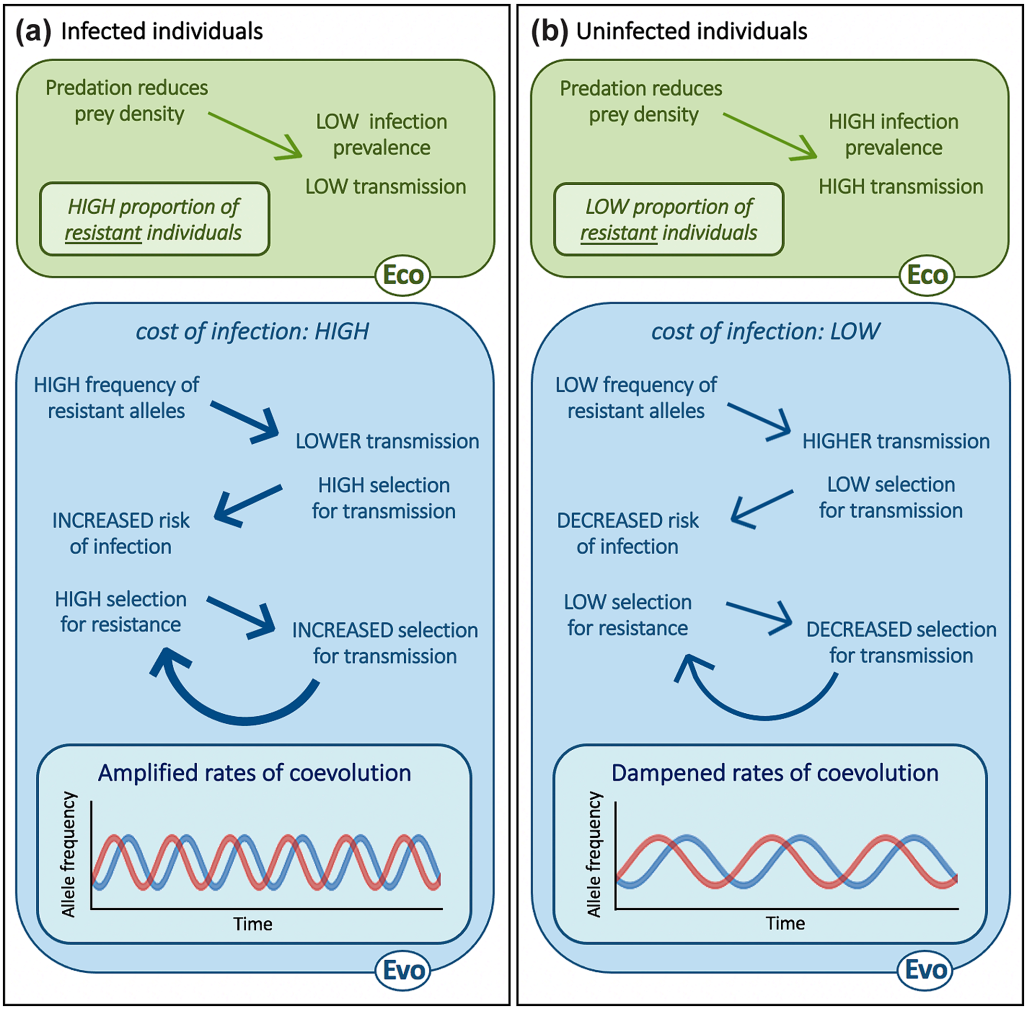

Figure 2. Illustration of frequency-dependent mechanisms that could lead to a change in the rate of coevolution between hosts and parasites as a result of selective predation pressure on infected (a) and uninfected (b) prey.

Examining the influence of predation on host–parasite interactions requires knowledge of predator, prey and parasite life history traits that can shape disease outcomes. For instance, predator feeding behavior and its effects on the behavior of their prey can be affected by the host's infection status (Decaestecker et al. 2002, Moore 2002, Hatcher et al. 2006). Under distinct predation strategies, prey could experience different levels of predation pressure, altering the cost of a parasitic infection for the prey. These changes in the virulence of such parasites can also mediate the degree and rate of changes in predator–prey and host–parasite dynamics. Similarly, seasonal changes in host behavior, predator feeding preferences or the cost of infection could modulate disease dynamics. Given that in many organisms their reproductive behavior is restricted to certain times of the year, and it requires high energetic demands, seasonality in life history traits associated with disease dynamics are likely. Additionally, the potential for genetic correlations between host resistance to parasites and resistance to predators may affect responses to selection on host–parasite coevolution (Friman and Buckling 2013). Few study systems, however, seem to have such information available across hosts, their predators and parasites. Further studies that exploit natural variation in life history traits and genetic mechanisms that influence disease outcomes and examine those traits associated with selective predation for uninfected individuals, could reveal previously unconsidered disease dynamics.

Additionally, the structure of parasitic communities can alter critical disease factors, such as the cost of infection for hosts and predators and shape the way predators interact with their prey. Co-occurrence of different species of parasites in a host population can influence the host-choice behavior of parasites. In cases where one parasite species is competitively dominant and reduces the successful establishment of another species, the subordinate parasite may alter their host-choice strategy. Free-living larval stages of subordinate trematode species detect and avoid potential snail hosts infected with dominant parasites to increase their establishment success (Allan et al. 2009, Vannatta et al. 2020). Such host-choice behavior could lead to the aggregation of the subordinate parasites' population in

a small number of hosts. Even though this type of host choice behavior has not been directly linked to aggregation of parasites in hosts, aggregation is common (Combes 2001, Poulin 2013). Changes in parasite load can alter the cost of infection through increased parasite-induced mortality or risk of predation. Interactions among species of parasites within a community through predation of infected or healthy individuals can shift the interplay between hosts, parasites and predators and influence disease transmission rates.

Investigations across multiple locations could provide instances of different selective predation strategies across populations allowing direct comparisons of these strategies on host–parasite interactions. Potential scenarios that could provide opportunities for assessing the differential role of selective predation include taking advantage of natural variation in predator abundance and distribution. For example, as species become isolated on predator-free islands (Blumstein 2002), insular systems could be used to contrast disease and prey dynamics with their mainland counterparts. Similarly, human-driven control of predators across populations, have and can offer valuable study systems to examine patterns of disease dynamics. For example, variation in fox hunting practices served as an opportunity to observe differences in red grouse infections between populations (Hudson et al. 1998). Investigating communities with natural variation in predation pressure could provide insights into the role of predators in moderating host and parasite population interactions.

Using long-term ecological studies would add to our understanding of the effects of predators on disease as ecological and evolutionary timescales differ among predators, prey and parasites. While some mathematical models have considered the disease outcomes of selective predation over multiple generations (Packer et al. 2003, Hoyle et al. 2012, Best 2018), most observational and experimental studies focus on short-term changes in disease dynamics (Duffy et al. 2005, Johnson et al. 2006; but see Ives and Murray 1997). Long-term ecological studies of prey population densities and parasite prevalence under varying conditions could uncover dynamic patterns in the cost of infection for predators and prey. For instance, as the prevalence of infected prey changes, the probability of a predator consuming infected prey and becoming infected will change, impacting the overall cost of infection. Cases in which there is a significant increase in prevalence and cost of disease could lead a predator to modify their predation strategies. Practical constraints of long-term studies including the generation time of the study systems and limitations in sampling frequency and duration may inaccurately capture natural dynamics. Despite these limitations, the conclusions of long-term studies would allow for more robust predictions of disease transmission and prevalence.

A challenge inherent to studying these complex, three player interspecific interactions is the variance associated with studies in natural settings. Systems amenable to mesocosms or laboratory experiments can simulate predation pressure by selectively removing infected or uninfected individuals under more controlled conditions. This approach can be implemented in species such as those in the genus *Daphnia* or *Drosophila* that can be readily cultured in replicated 'populations', allowing parasite infection to be manipulated and predation preference simulated. For example, the results of measuring changes in population densities and infection rates in *Daphnia*, under artificial selective predation over multiple generations, suggest that the benefits of low susceptibility in these predation treatments may be outweighed by associated tradeoffs (e.g. reduced fecundity) (Gutierrez et al. unpubl.). Similarly there is great potential in building upon recent work on *Drosophila* fruit flies that have been used to investigate changes in activity levels in response to parasite exposure (Horn et al. 2020). Expanding on this research to include a predator would provide an ideal opportunity to explore the effect of predation pressure on host–parasite interactions. Studies under controlled conditions with varying disease prevalence and population densities could reveal patterns, exceptions and factors modulating such dynamics over different timescales. Understanding the ecological and evolutionary consequences of selective predation we can begin to unravel the complex influences of predation on host–parasite interactions and the resulting outcomes.

Concluding remarks

Current evidence suggests selectively predating of uninfected individuals is likely more common than previously anticipated. Selective predation both on infected and uninfected individuals impacts host and parasite population dynamics. This ecological interaction can also result in long-term coevolutionary consequences for disease transmission. Selective predation on either healthy or infected prey is predicted to have different evolutionary outcomes on host–parasite systems. Whereas selective predation on infected individuals is predicted to intensify the coevolution between hosts and parasites, preferential consumption of healthy individuals is predicted to dampen the reciprocal selective pressures between hosts and parasites. Coevolutionary rates could vary in systems with diverse costs of mating signals. Testing these predictions in theoretical and empirical work using a diverse array of systems will enhance our understanding of the role that predation plays in host–parasite coevolution and disease dynamics.

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Author contributions

Stephanie O. Gutierrez: Conceptualization (lead); Visualization (lead); Writing – original draft (lead); Writing – review and editing (lead). **Dennis J. Minchella**: Writing – review and editing (equal). **Ximena E. Bernal**: Conceptualization (equal); Writing – original draft (equal); Writing – review and editing (equal).

Data availability statement

Data sharing is not applicable to this article as no new data were created or analyzed in this study.

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