An Investigation of The Healthy Herd Hypothesis In The Trinidadian Guppy

by

Ben K Sniegowski

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Approved by

Ronald Bassar, Associate Professor of Biological Sciences
Michael Smith, Assistant Professor of Biological Sciences
Moises Bernal, Assistant Professor of Biological Sciences

Abstract

Predators can influence host–parasite dynamics through density-mediated effects, behavioral modification, and selective removal of infected individuals, as explained by the Healthy Herd Hypothesis (HHH). Despite its conceptual prominence, empirical support for the HHH remains taxonomically and methodologically narrow, with limited tests in vertebrate freshwater systems. This thesis integrates a systematic review of the literature on predator–parasite–prey interactions with a replicated mesocosm experiment using Trinidadian guppies (*Poecilia reticulata*) and the nematode *Camallanus cotti*. The review identifies substantial biases toward invertebrate systems, directly transmitted parasites, and field studies lacking mechanistic resolution, that is to say tests of what parasites changed in their hosts. In the mesocosm experiment, predator presence reduced overall guppy survival particularly of parasitized individuals, with these having 25.3% lower survival than unparasitized fish when predators were present. Parasite presence was significantly lower in predator treatments, supporting selective predation. These findings provide rare experimental evidence for the HHH in a vertebrate system and clarify the ecological conditions under which predators reduce parasite prevalence through targeted predation.

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Chapter 1. Linking Infection Dynamics to Ecological Outcomes in Predator–Host–Parasite Systems: A Review

Introduction

Some of the most widespread and influential interactions in the field of ecology are those between parasites and their hosts. Parasites are capable of altering species interactions, regulating host population size, and even of driving evolutionary change in host life history, behavior, and physiology (Hasik et al., 2025; Schmid-Hempel, 2001; Anderson and May 1979). Past work has focused on simplified host-parasite interactions and considered broader effects only in passing, but recent research has emphasized that parasite dynamics are embedded within ecological networks containing hosts, competitors, predators, and other environmental drivers (Johnson et al., 2010; Lafferty et al., 2008).

Two conceptual frameworks have been proposed to explain how ecological interactions may shape parasite prevalence in host populations. The first is the Healthy Herd Hypothesis, which proposes that predators can reduce disease prevalence when they selectively remove infected or otherwise weakened prey from the population (Packer et al. 2003). In this framework, reduced disease prevalence in a population results from selective mortality imposed by predators on high-risk hosts due to parasite infection (Packer et al. 2003). The second framework is the dilution effect, which proposes that greater biodiversity can reduce disease prevalence by decreasing the relative abundance of highly competent hosts or by disrupting transmission pathways among susceptible hosts (Ostfeld and Keesing 2000). Unlike the Healthy Herd Hypothesis, dilution effects are typically mediated through changes in host density, species composition, competition, or encounter rates rather than selective predation

itself (Keesing et al. 2006). Although both mechanisms can reduce parasite prevalence, they operate through fundamentally different ecological pathways, that is to say changes in host population density for the dilution effect and direct predation for The Healthy Herd Hypothesis. Both frameworks have experimental support, although their effects depend strongly on transmission mode, host community composition, and the ecological context in which host–parasite interactions occur (Halliday and Rohr 2019).

Foundations

Classic Models

The work of Robert May and Robert Anderson founded the field of disease ecology with their development of theoretical models describing the population biology of infectious diseases and their cycles (Anderson & May, 1979). This theoretical foundation was based on earlier experimental work in population ecology, in particular that of Thomas Park and his long-term *Tribolium* beetle competition studies, which demonstrated that population dynamics are strongly shaped by density dependence and interspecific interactions, and that these processes can generate stable cycles, coexistence, or extinction under controlled conditions (Park 1948; Park 1954; Edmunds et al. 2003). More recent syntheses of Park's work have emphasized its foundational role in establishing population regulation as a central problem in ecology and in the basis of later mathematical approaches to population dynamics (Edmunds et al. 2003; Pointer et al. 2021).

Building on this broader foundation of ecology and evolutionary theory and experimentation, Anderson and May formalized mathematical frameworks for infectious

disease dynamics in host populations by explicitly incorporating host population dynamics into general mathematical models of transmission processes (Anderson & May 1979a, 1979b). Prior to their work, little integration of host density, transmission processes, and disease prevalence into a unified theoretical structure had been achieved. In their 1979 *Nature* papers, Anderson and May developed key models that linked host–pathogen interactions to population-level outcomes. Anderson and May later synthesized these ideas into their formal definition of the basic reproductive number R_0, defined as the expected number of secondary infections produced by a single infected individual in a fully susceptible host population (Anderson & May 1979). Their work also contributed to broader ecological debates on the importance of top-down regulation in natural systems, particularly whether parasites and pathogens, like predators, can regulate host population sizes and shape community structure across trophic levels (Anderson & May 1978; May & Anderson 1979).

Subsequent theoretical and empirical work has supported their work by demonstrating that parasites can influence not only individual host fitness but also host survival, reproduction, population stability, and interspecific interactions (Hudson et al., 1998; Lafferty, 2008). The work of Anderson and May helped establish parasites as integral components of ecological communities, capable of shaping biodiversity patterns, trophic interactions, and ecosystem processes in ways that extend classical predator–prey theory (Dobson & Hudson 1986; Poulin 1999).

Drivers of Evolution

Parasites have been recognized not only as infectious disease agents, but also as drivers of ecological and evolutionary processes in natural communities. Because parasites can reduce host growth, survival, reproductive success, and general condition, they are able to exert strong selective pressures on host populations (Price, 1980; Hasik et al. 2025). These selective pressures can generate opposing evolutionary responses between hosts and parasites, thereby creating continuous coevolutionary arms races in which as hosts evolve defenses against infection, parasites evolve mechanisms to overcome those defenses (Dybdahl & Lively 1998). This process is commonly described by the Red Queen hypothesis, which proposes that species must constantly evolve to maintain their relative fitness as they coevolve with other species (Lively et al. 1990; Solé 2022). Host–parasite systems are among the clearest examples of Red Queen dynamics due to the short generation times of many parasites and their ability to rapidly adapt to the current most common host genotypes (defense strategies), thereby favoring genetic variation within host populations (Lively, 2010).

Parasite-mediated coevolution has also been proposed as one of the primary evolutionary explanations for the persistence of sexual reproduction (Lively 2010). Sexual reproduction may allow hosts to generate novel genetic combinations faster than asexual reproduction, thereby reducing parasite success and slowing parasites' adaptation to common host genotypes in the community (Hamilton et al., 1990; Lively & Dybdahl, 2000). Experimental work in freshwater snail–trematode systems showed that parasites often adapt most quickly to the most common host genotypes, therefore selection favors relatively rare host genotypes and maintains genetic diversity within

populations (Lively & Dybdahl, 2000). These studies helped establish parasites as important evolutionary forces capable of shaping host life histories, reproductive strategies, and the genetic structure of host populations.

Parasites may also influence the evolution of secondary sexual traits and mating behavior, as explained by the Hamilton–Zuk hypothesis. The Hamilton–Zuk hypothesis proposes that sexually selected traits may function as honest signals of parasite resistance and overall genetic quality (Hamilton & Zuk, 1982). This framework proposes that individuals displaying brighter coloration, more elaborate ornaments, or more vigorous courtship behaviors may be signaling superior parasite resistance to potential mates. In fishes, parasitic infections frequently alter host coloration, activity, body condition, and behavior, traits that are key to predator avoidance and reproductive success (Barber et al. 2000). Guppies are particularly well suited for examining these interactions because coloration and behavioral displays are important components of their sexual selection, and predators have been shown to exert a strong influence on these traits (Endler, 1980; Magurran & Phillip 2001).

Parasites frequently alter host phenotype and behavior in ways that increase susceptibility to predation, linking parasitism and predation as interacting ecological forces (Lafferty & Morris 1996; Poulin 2010). Infected individuals may exhibit reduced swimming performance, impaired escape responses, altered behavior, or decreased body condition, all of which can increase the likelihood of capture (Hudson et al., 1992; Lafferty, 1999). In some cases, parasites may even manipulate host behavior in ways that increase trophic transmission to final or definitive hosts (Lafferty & Morris 1996). However, when predators selectively remove infected prey without themselves

becoming effective hosts for the parasite, predation may reduce parasite prevalence within prey populations (Lopez et al. 2023; Lopez & Duffy 2021). This mechanism forms the basis of the Healthy Herd Hypothesis, which predicts that predators can decrease disease prevalence by disproportionately consuming infected individuals (Packer et al., 2003).

Although parasite-mediated selection has long been recognized as an important evolutionary force, the ecological consequences of predators selectively removing infected individuals remain insufficiently understood across many host–parasite systems (Vitale & Best 2019). In Trinidadian stream communities, guppies experience strong predation pressure while simultaneously suffering infection by the invasive nematode *Camallanus cotti*. This system therefore provides an opportunity to examine how predator–parasite interactions may influence not only individual survival, but also broader patterns of parasite prevalence across ecological communities.

Biodiversity and The Dilution Effect

Concepts

The dilution effect occurs when higher biodiversity reduces disease transmission by lowering the proportion or impact of highly competent hosts within a community (Keesing and Ostfeld 2021). When a pathogen encounters a diverse host community, it is more likely to interact with species that are poor or incompetent hosts, thereby reducing the overall probability of transmission to a competent host and decreasing disease prevalence or risk (Keesing, Holt, & Ostfeld, 2006; Ostfeld & Keesing, 2017).

Biodiversity can thus serve as a natural form of disease regulation: as the number and variety of species in an ecological community increase, the likelihood that any given pathogen encounters a high-competence host decreases, potentially reducing transmission at the community level (Ostfeld & Keesing, 2017).

Dilution effects can arise through multiple mechanisms. Reduced encounter rates: in diverse communities, vectors or pathogens encounter a greater number of non-competent hosts, lowering the chance of successful transmission to competent hosts (Halliday & Rohr, 2019). Increased prevalence of low-competence hosts: when diversity includes species that are less likely to support infection or transmit parasites, overall transmission potential declines (Downs et al. 2019). Interference with transmission pathways: additional species can act as ecological “buffers”, competing with or preying on competent hosts or vectors, consuming free parasite stages, or otherwise disrupting the cycle of infection (Halliday & Rohr, 2019; Johnson et al. 2010).

Different mechanisms may dominate in different systems. Dilution via encounter reduction is often important in vector-borne diseases, whereas in directly transmitted disease systems, changes in host community composition may be more influential (Halliday & Rohr, 2019).

Evidence

Experimental and observational studies have provided evidence that biodiversity can reduce disease prevalence. Increased host richness has been shown to reduce parasite infection rates in freshwater communities (Lagrue and Poulin 2015). Amphibian

communities with higher species diversity had lower trematode infection rates (Johnson et al. 2008). Conversely some studies show that under certain conditions, increased diversity may instead amplify disease (Wood et al. 2014; Keesing et al. 2010). Amplification is most often seen in systems where anthropogenic biodiversity loss has occurred rather than natural systems. The actual drivers of these differences, however, remain unclear.

Synthesis

Despite empirical support, the dilution effect remains a subject of active debate. Meta-analyses have suggested that biodiversity–disease relationships are context-dependent and influenced by ecological and epidemiological factors (Rohr et al. 2019). Other studies have indicated that dilution effects are most consistent when diversity gradients result from anthropogenic loss, whereas natural diversity gradients may show weaker or more variable effects (Halliday et al. 2020).

Healthy Herd Hypothesis

Conceptual Framework

The Healthy Herd Hypothesis proposes that predators can and will reduce disease prevalence in hosts that they prey upon by selectively removing infected individuals over non-infected individuals (Packer et al. 2003). This hypothesis explains the decreased prevalence of infection observed in the presence of predators compared to their absence in many host-parasite-predator systems.

Mechanisms

Predators can reduce disease prevalence in their prey through multiple mechanisms. Prey that are infected with a parasite often exhibit impaired locomotion and or altered behavior (Møller and Nielsen 2007). Predators also lower prey density, thereby reducing transmission rates between hosts in density-dependent systems where parasites are transmitted directly between hosts (Anderson and May 1979; Ostfeld and Holt 2004). Finally, predators may directly consume parasites at some stage before the parasite has reached its definitive host, or predators may consume intermediate hosts, thereby disrupting transmission (Johnson et al. 2010; Rohr et al. 2015).

These mechanisms when taken and understood together suggest how predators can both reduce disease prevalence and shape host population dynamics.

Evidence

The Healthy Herd Hypothesis has received mixed, but often positive support from experimental studies. Studies in amphibian host-parasite-predator systems have shown that predators can reduce parasite prevalence through both consuming infected hosts and consuming parasites directly (Orlofske et al., 2012). In a zooplankton-parasite-predator system, predators were shown to reduce infection prevalence by altering species interactions and lowering overall host density (Duffy et al., 2005). Similar results were also found for a tadpole-ranavirus-dragonfly larvae system, where the presence of predatory dragonfly larvae reduced tadpole host density and therefore ranavirus infection (Gallagher et al. 2019).

Patterns in Empirical Tests of the Healthy Herd Hypothesis

Although numerous studies have examined predator–parasite interactions, support for the Healthy Herd Hypothesis has proven highly context dependent, varying with host and parasite identity, transmission mode, and ecological system (Lopez & Duffy, 2021; Lopez et al., 2023). A more obvious pattern in the literature is that predator-mediated reductions in disease prevalence are most commonly observed when predators selectively remove infected individuals over uninfected ones and when the parasites do not rely on trophic transmission to complete their life cycles (Lopez & Duffy, 2021; Lopez et al., 2023). In systems involving complex parasite life cycles, particularly those that depend on trophic transmission to reach a definitive host, support for the Healthy Herd Hypothesis is often weaker because predation may facilitate rather than disrupt parasite transmission by completing the parasite's life cycle (Lafferty & Morris, 1996; Lafferty, 1999; Poulin, 2010).

A second clear pattern in the literature is a strong taxonomic and methodological bias in the systems used to study predator–parasite interactions. Across the 69 studies used to make the figures in this review, invertebrate systems constitute the vast majority of the empirical foundation of the Healthy Herd Hypothesis, accounting for 29% of all studies. Mammalian (17.4%), fish (13%), and bird (10.1%) systems appear less frequently, and amphibian systems account for only 5.8% of studies (Figure 1). The remaining 24.6% fall into the “Other” category, which includes multi-taxon syntheses, conceptual or theoretical work, and studies that do not focus on a single host group. These patterns indicate that the evidence base for predator-mediated disease

regulation is shaped disproportionately by a narrow set of host taxa, with invertebrate and mammalian systems contributing the bulk of empirical tests.

A similar pattern emerges when the study designs used to evaluate Healthy Herd mechanisms are considered. Nearly half of all studies (47.8%) were conducted in natural field settings, only 10.1% were laboratory experiments and 5.8% were mesocosm studies. Mixed field–lab (5.8%) and mixed mesocosm–field (1.45%) designs were comparatively rare most likely due to their more involved nature (Figure 2). The remaining 29% of studies fell into an “Other” category, consisting primarily of theoretical models, comparative analyses, and conceptual syntheses. Although field studies dominate numerically, the mechanistic evidence—particularly studies capable of isolating selective predation, density-mediated effects, or parasite-induced vulnerability—still comes from controlled experimental systems, where predator density, parasite exposure, and host condition can be manipulated independently. In contrast, field studies often involve simultaneous variation in environmental conditions, community structure, and host density, making it difficult to attribute changes in infection risk to specific predator-mediated processes.

Together, these patterns show that current support for the Healthy Herd Hypothesis is built on a methodologically broad but taxonomically narrow literature. The predominance of invertebrate systems and the reliance on controlled experiments highlight important gaps in our understanding of how predator-mediated disease regulation operates in many host taxa, particularly fish and mammal systems under natural ecological conditions. Expanding empirical tests into these less-represented systems—and incorporating parasites with diverse life histories—will be essential for

determining the ecological contexts under which Healthy Herd effects emerge and for evaluating whether mechanisms identified in these systems are generalizable to more complex natural communities.

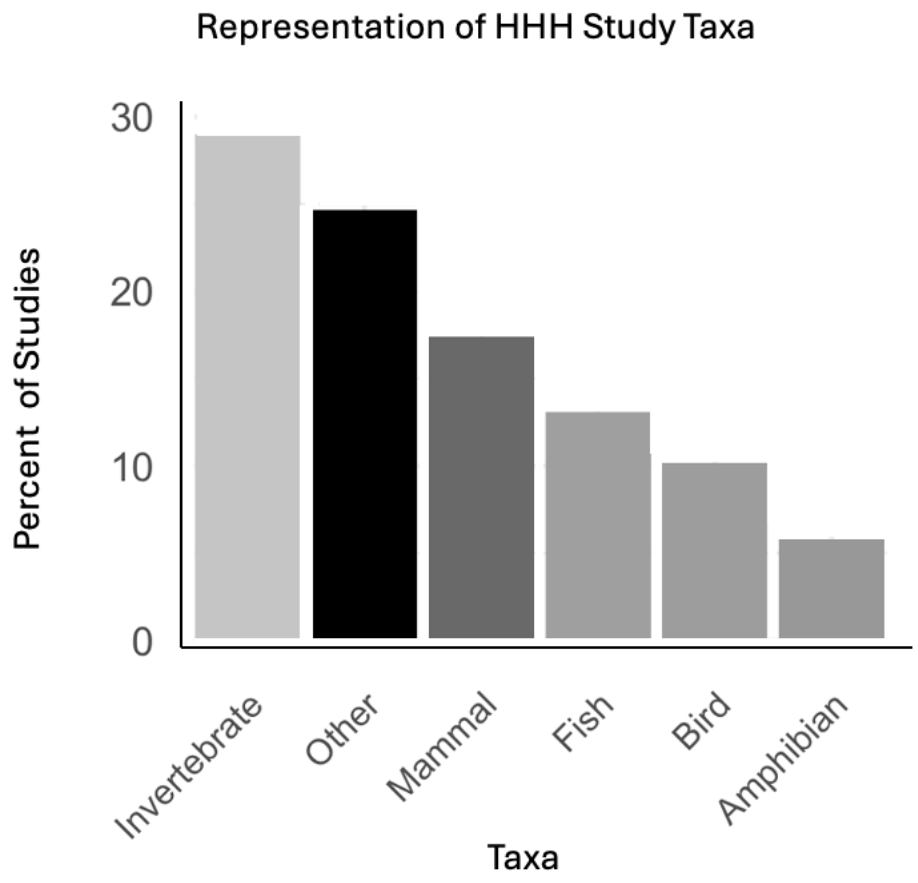

Figure 1 Representation of HHH Study Taxa

Distribution of biological taxa represented in studies relevant to the Healthy Herd Hypothesis. Invertebrate taxa dominated the literature (29%), with substantial representation from "Other" nonliving systems such as models and theory (24.6%) and mammalian hosts (17.4%). Fish (13%) and bird (10.1%) taxa were moderately represented, while amphibian taxa were comparatively rare (5.8%).

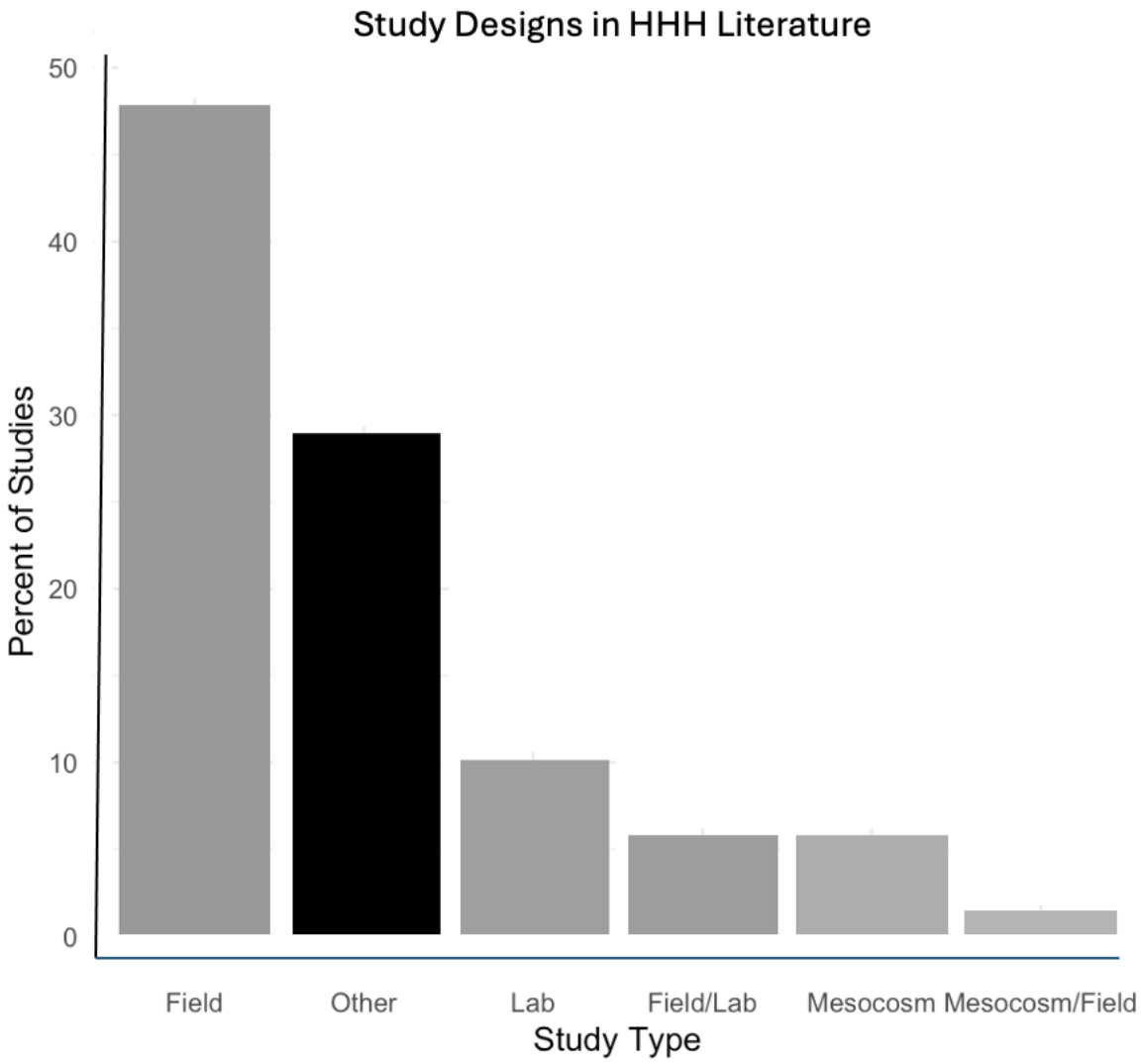

Figure 2 Study Designs in HHH Literature

Proportion of study designs used in Healthy Herd Hypothesis research. Field studies were the dominant approach (47.8%), with "Other" designs—primarily theoretical or modeling approaches—representing 29%. Laboratory studies (10.1%), mesocosm experiments (5.8%), and mixed field–lab (both observational studies in natural systems and controlled experiments in a laboratory) studies (5.8%) were less frequently employed and mixed mesocosm–field studies were rare (1.45%).

Knowledge Gaps and Future Directions

Despite growing evidence that predators can influence disease dynamics, several important gaps remain in our understanding of the Healthy Herd Hypothesis. As previously stated, one of the most notable patterns emerging from the literature is the concentration of empirical studies within relatively few aquatic model systems, particularly amphibian–trematode and zooplankton–parasite interactions (Duffy et al., 2005; Orlofske et al., 2012; Gallagher et al., 2019). Aquatic systems are particularly suited to disease research because in aquatic systems diseases and their hosts can be separated more easily and effectively than in many terrestrial systems. Previous reviews of predator–parasite interaction studies similarly note that the bulk of the available evidence has been found in a narrow range of host taxa and ecological systems, therefore limiting the generalizability of their findings across broader ecological contexts (Lopez & Duffy, 2021). Few studies have examined the dynamics of healthy herd effects in fish systems despite the widespread importance of both predation and parasitism in freshwater ecosystems and the economic and biological importance of these systems (Lafferty et al., 2008; Poulin, 2010).

A second limitation is that the majority of studies are field experiments. While field studies are invaluable for identifying patterns, relatively few studies have investigated Healthy Herd predictions under controlled conditions where multiple biotic and abiotic factors may be simultaneously manipulated (Werner & Peacor, 2003; Carpenter, 1996). Highly controlled experimental systems allow investigators to manipulate predator density, parasite exposure, and host abundance independently, thereby allowing mechanistic inferences to be made, but the extent to which these

findings translate to natural populations remains an important question (Lopez & Duffy, 2021). Therefore, a need remains for studies that bridge the gap between highly controlled experiments and complex natural ecosystems.

There is also a bias in the literature toward systems involving directly transmitted pathogens or parasites with relatively simple life cycles. Less attention has been given to parasites with indirect transmission pathways, including many nematodes, in which transmission depends upon interactions among hosts, intermediate hosts, and predators (Anderson & May, 1979; Poulin, 2010). In these systems where predators may either disrupt or facilitate transmission, such as the toxoplasma-rodent-felid system where the parasite relies on the final felid host to reproduce, predictions regarding Healthy Herd effects are often less straightforward than those for directly transmitted diseases (Lafferty & Morris, 1996; Lafferty, 1999; Cáceres et al. 2009). Such complexity may also contribute to the non-uniform support for predator-mediated disease reduction observed across host–parasite systems.

Finally, much of the disease ecology literature has focused on infection prevalence, transmission rates, and other epidemiological metrics, and not the broader ecological consequences of infection. These broader consequences include effects on host growth, survival, population structure, and community dynamics. They have been understudied despite their importance in understanding the role of parasites in natural ecosystems (Dallas and Cornelius 2015). Some researchers have highlighted the need to better understand how infectious diseases influence demographic processes and population persistence as a bridge across the gap between studies of infection dynamics and studies of ecological outcomes (Johnson et al. 2015; Tompkins et al.

2011). Evaluation of these broader dynamics is particularly important in the context of the Healthy Herd hypothesis because predators may reduce infection prevalence while simultaneously altering population structure and dynamics through selective mortality of infected individuals (Packer et al., 2003; Johnson et al. 2010). Additionally, predator-induced changes in host behavior, physiology, and life-history traits are known to influence disease dynamics in ways that may not be captured by prevalence estimates alone (Hawlena & Schmitz, 2010; Clinchy et al., 2013). A clearer understanding of Healthy Herd effects will therefore require studies that integrate epidemiological, behavioral, and demographic responses to predation and parasitism.

Unhealthy Herds

Through indirect effects predators may conversely increase disease prevalence in hosts. Predator presence can increase physiological stress thereby reducing immune function (Clinchy et al. 2013) which in turn can lead to increased susceptibility to infection. Prey may also change their behavior in the presence of predator cues, thereby increasing their susceptibility to infection (MacLeod and Luong 2025). In the presence of predators prey may group more closely for defense, spend more time in environments that facilitate transmission such as dense vegetation, or they may feed on non ideal food sources that are vectors for parasite transmission (Richards et al. 2023). While predators can reduce disease through selective removal of infected hosts, they can also increase disease risk through indirect effects or by facilitating trophic transmission, particularly in parasites with complex life cycles.

Trophic Transmission

In systems where parasites rely on trophic transmission — that is, where successful transmission to the next host requires that an infected intermediate host is eaten by a predator — the role of predation is fundamentally different from the assumptions of the healthy herd hypothesis. Classic models of The Healthy Herd Hypothesis assume that predation always reduces parasite prevalence by removing infected individuals from the population. However, for trophically transmitted parasites, predation acts as the transmission mechanism itself: parasites exploit predator–prey interactions to move between hosts and complete their life cycles.

Early conceptual work has highlighted that many parasite taxa have evolved strategies that increase the likelihood of trophic transmission, a phenomenon referred to as parasite-increased trophic transmission (PITT), which underscores the pervasive ecological and evolutionary importance of this transmission strategy in parasites that require multiple hosts (Lafferty 1999).

Empirical examples in which parasites manipulate the behavior or phenotype of their intermediate hosts in ways that make them more likely to be consumed by the appropriate predator, thereby facilitating trophic transmission and increasing parasite fitness are found in the literature as well (Lafferty & Morris, 1996). This inversion of the typical predator-removes-disease narrative illustrates a key limitation of the healthy herd hypothesis: predators do not always act to reduce transmission risk but can instead be essential in parasite life cycles.

Trophic transmission dependency emphasizes the importance of parasite life-history strategies in shaping host–parasite–predator interactions. Whether predation

reduces or enhances parasite success depends critically on the parasite's transmission mode and life history, with trophically transmitted parasites representing a clear example where predation can facilitate, rather than mitigate, transmission.

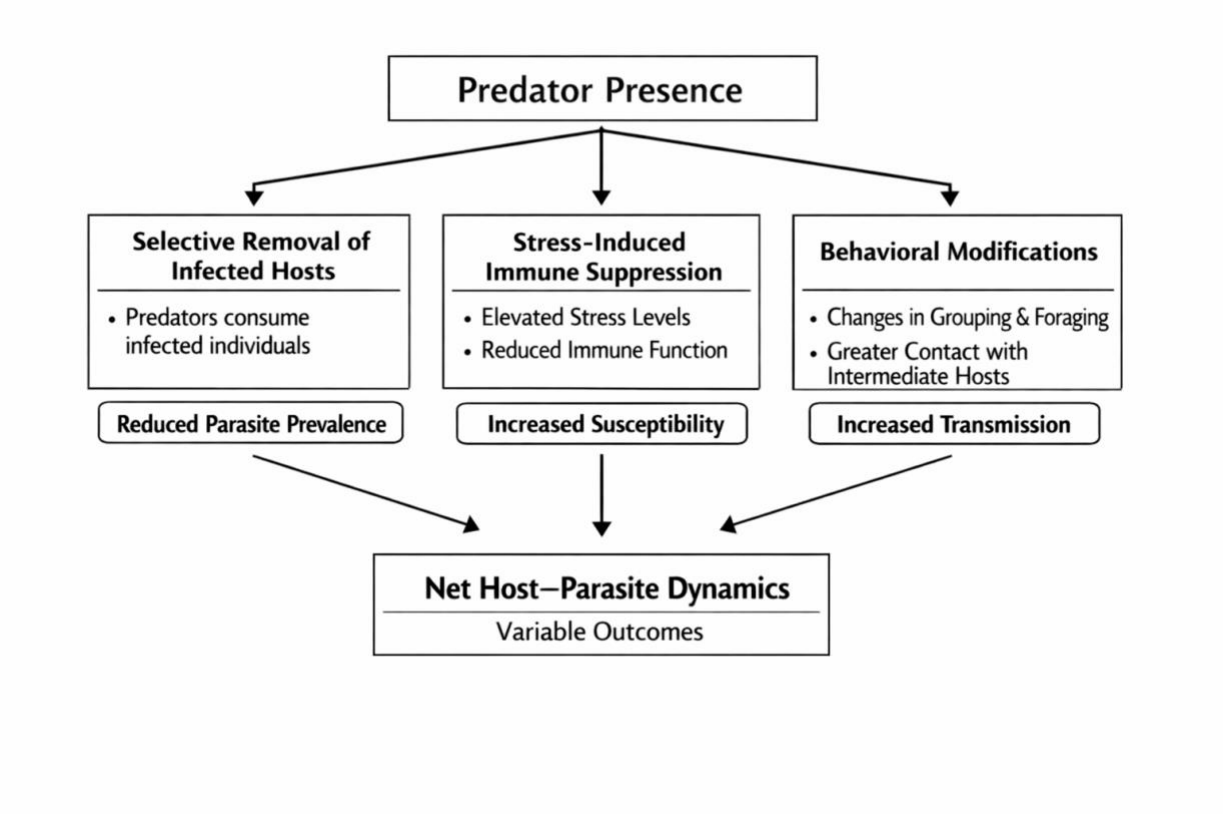

Figure 3 Variable Outcomes of Predator Presence

Predator presence can influence infection outcomes through three primary pathways: (1) selective removal of infected hosts, reducing parasite prevalence; (2) stress-induced immune suppression, increasing host susceptibility; and (3) behavioral modifications that alter exposure and transmission potential. The net effect on parasite dynamics depends on the relative strength and interaction of these pathways under natural or experimental conditions.

Application To Guppy-Camallanus-Cichlid System

Guppies (Poecilia reticulata) have long been used for studies focused both on predators and parasites in ecological communities due to well documented variation both in both predator and parasite species along elevational gradients within their stream communities (Magurran, 2005; Reznick et al., 1996). Guppies are known hosts

for a range of parasites both external and internal such as Gyrodactylus spp. And Camallanus spp. respectively (Cable & van Oosterhout 2007; Stephenson et al. 2015).

Parasites such as *Camallanus cotti* can substantially reduce host fitness in guppies (*Poecilia reticulata*) by impairing growth, reproductive output, and overall condition (Menezes et al. 2006). Infected individuals often exhibit reduced vigor and altered behavior, which may increase their vulnerability to predation by visually oriented predators such as the pike cichlid (*Crenicichla alta*). This creates conditions under which predators may selectively remove infected individuals from the population, consistent with the healthy herd hypothesis (Lafferty & Morris, 1996; Packer et al., 2003).

Conversely, predator presence can create conditions under which parasite exposure risk and subsequent infection may increase (Macleod and Luong 2025). Exposure to predation risk may elevate physiological stress in guppies, potentially suppressing immune responses and increasing susceptibility to *Camallanus* infection as has been demonstrated in House Sparrows (*Passer domesticus*) and their predators (Navarro et al. 2004). Predators may also induce behavioral changes such as tighter shoaling, increased use of refuge habitats, or reduced activity which may in turn increase host-host contact and or concentrate individuals in environments more conducive to parasite transmission (Walsman et al. 2022; Thieltges et al. 2024). Due to the transmission mode of Camallanus, through intermediate hosts such as freshwater copepods, predator induced changes in foraging behavior may have an impact on exposure rates for prey species that feed on copepods like guppies (Lima & Dill 1990; Levsen & Berland 2002).

Experimental manipulations of parasite prevalence and predator presence in mesocosms are useful for understanding the exact nature of these interactions in a simulated wild setting (Strasburg and Boone 2022). By integrating predator presence and parasite prevalence manipulations in a mesocosm design it is possible to assess whether The Healthy Herd Hypothesis can be considered applicable in the guppy-Camallanus-cichlid system.

Conclusion

A systematic review of the Healthy Herd Hypothesis literature shows that the field is conceptually rich but empirically narrow. Although research in the field spans a wide range of host–parasite systems, the evidence is disproportionately shaped by invertebrate studies, which account for 29 percent of all papers. Mammalian, fish, and bird systems appear with moderate frequency, but amphibian systems account for only a small fraction of studies. Nearly a quarter of the literature consists of theoretical, conceptual, or multi-taxon work that does not directly test Healthy Herd mechanisms but instead describes conditions under which support may be found. This distribution indicates that direct empirical tests remain concentrated in a narrow subset of host–parasite interactions, limiting the generality of current conclusions.

The methodological landscape shows a similar imbalance. Field studies represent the largest share of published work, yet many of these studies document correlations rather than isolating causal pathways linking predation to infection outcomes as it is difficult to track outcomes in large scale field experiments. Laboratory and mesocosm experiments, which are best suited for manipulating predator density,

parasite exposure, and host condition independently, remain comparatively rare possibly due to the difficulty of keeping large predators and prey in captivity. Mixed designs that bridge controlled and natural settings are rarer still. As a result, the strongest mechanistic evidence for Healthy Herd effects continues to come from simplified experimental systems, while field studies often involve simultaneous variation in environmental conditions, community structure, and host density that obscures the processes driving infection patterns.

These patterns reveal that current support for the Healthy Herd Hypothesis rests on a methodologically diverse but mechanistically narrow foundation. Important gaps remain in understanding how predator-mediated disease regulation operates in understudied host taxa, particularly fishes and mammals, and in systems involving parasites with complex or trophic transmission, especially those that manipulate host behavior to increase transmission. Addressing these gaps will require expanding empirical tests into a broader range of host–parasite systems, incorporating parasites with varied life histories, and designing experiments that more closely approximate natural ecological complexity.

The guppy–Camallanus–cichlid system stands out as a uniquely powerful model for advancing Healthy Herd research. It combines a well-characterized host–parasite interaction (guppies and Camallanus), and a naturally occurring predator (pike cichlid) that exerts strong selective pressure. Unlike many systems in the existing literature, guppies experience predation and parasitism simultaneously in the wild across a range of community types, allowing researchers to evaluate Healthy Herd mechanisms under ecologically realistic conditions. By situating this system within the broader patterns

identified here, future work can clarify when and why predators reduce disease in prey populations, when they may instead amplify infection risk, and how these dynamics unfold in a vertebrate system that is both tractable and ecologically meaningful.

Chapter 2. Predators Disproportionately Remove Parasitized Hosts in a Trinidadian Stream System

Abstract

Predators can influence disease prevalence in ecological communities by reducing host density and by selectively removing infected individuals, a mechanism known as the Healthy Herd Hypothesis. We tested whether predator presence selectively reduced survival of parasitized hosts in a replicated mesocosm experiment using Trinidadian guppies (*Poecilia reticulata*) and their invasive nematode parasite *Camallanus cotti*. Mesocosms contained mixed groups of treated (parasite-free) and untreated (potentially infected) hosts, and contained either one or no predators. Predator presence reduced overall guppy survival by 19.2%, but disproportionately affected parasitized individuals, which experienced 25.3% lower survival than unparasitized individuals in predator treatments, whereas little difference occurred in predator-free mesocosms. These results provide evidence that predators selectively remove infected hosts and suggest that predator-mediated selective mortality may contribute to negative relationships between biodiversity and pathogen prevalence observed in natural communities.

Introduction

Predators can influence pathogen prevalence in ecological communities through multiple pathways, including reducing host density and selectively removing infected individuals. This latter mechanism, known as the Healthy Herd Hypothesis, predicts that predators disproportionately consume infected or otherwise compromised hosts,

thereby lowering pathogen prevalence in the remaining population (Packer et al. 2003). Evidence for this process has been documented across diverse systems, including red grouse (Hudson et al. 1992), amphipods (Knudsen et al. 2001), and small mammals hunted by raptors (Temple 1987). However, predator–parasite–prey interactions are often complex, and not all studies have detected selective predation on infected hosts (Richards et al. 2021). A major challenge in evaluating the Healthy Herd Hypothesis is that predators simultaneously alter host density, behavior, and demography, making it difficult to isolate the contribution of selective removal from broader density-mediated effects and interactions.

Trinidadian stream communities provide a powerful system for testing how predators influence parasite dynamics in both natural and semi-natural experimental setups. Guppies (*Poecilia reticulata*) inhabit a diversity of stream communities across the island, ranging from low-predation headwater sites dominated by the killifish *Anablepsoides hartii* to high-predation lowland sites containing piscivores such as pike cichlids (*Crenicichla alta*) and wolf fish (*Hoplias malabaricus*) (Reznick 1982). These communities were recently invaded by the exotic nematode *Camallanus cotti*, a parasite common in the tropical fish trade that attaches to the intestinal lining of its hosts and feeds on blood and tissue (Menezes 2006). First detected in Trinidadian guppies in 2016 (Rogowski et al. 2020), *C. cotti* now infects multiple species across the watersheds. Surveys show that parasite prevalence tends to be lower in high-diversity, predator-rich communities than in low-diversity, predator-poor ones (Mohammed, Potter, and Bassar, unpublished data), suggesting that predators reduce infection through selective predation.

We used a replicated mesocosm experiment to test whether predators selectively remove parasitized hosts over unparasitized hosts. Guppies from a high-predation community were introduced into mesocosms with or without predators, and each mesocosm contained a mixture of treated (parasite-free) and untreated (potentially infected) individuals. As per the Healthy Herd Hypothesis, we expected little difference in survival between parasitized and unparasitized guppies in predator-free mesocosms, but reduced survival of parasitized individuals when predators were present.

Methods

Predator and host fish were collected from the Aripo River, Trinidad in May 2025 and transported to the field laboratory in the Arima Valley. We collected 200 female and juvenile guppies (*Poecilia reticulata*) and nine pike cichlids (*Crenicichla alta*), one of the dominant piscivores in these streams (Botham et al. 2006; Endler 1980). Pike cichlids were housed individually, and guppies were randomly split into groups of ten and maintained in group tanks.

To generate parasite-free and potentially infected host groups, we treated all pike cichlids and half of the guppies with the dewormer Fritz Expel-P (Fritz Aquatics) following manufacturer instructions: one packet per 10 gallons of water, a 25% water change the following day with removal of expelled worms, and repeated treatment after one week. For three weeks before the experiment, we kept treated fish in parasite-free conditions. We used only female and juvenile guppies to avoid confounding effects of male coloration on predator choice. During this period, we fed guppies freshly hatched brine shrimp daily and fed pike cichlids treated stock guppies to prevent new infections.

After three weeks, all guppies were individually marked using subcutaneous visible elastomer implants (Northwest Marine Technologies) to distinguish treated (parasite-free) and untreated (potentially infected) individuals. Fish were anesthetized in a buffered solution of 15 mg MS-222 per 50 mL of water, marked on either the left or right caudal peduncle according to treatment, and measured for standard length (nearest 0.01 mm) and mass (nearest 0.001 g). Black elastomer was used to minimize visibility to predators. Across treatments, guppies were randomly assigned to ensure similar size and mass distributions.

We used ten outdoor mesocosms constructed from cinder block and concrete troughs, each holding 125 L of stream-supplied water. Water flowed continuously through each mesocosm at approximately 0.3 L s^{-1}, maintaining a depth of 20–25 cm. Each mesocosm contained gravel substrate, natural rocks, and bricks to provide cover and foraging structure. Mesocosms were established 18 days before the experiment to allow natural algal and invertebrate communities to develop (Bassar et al. 2010). We introduced guppies one day before adding predators to allow them time to acclimate and establish refugia.

Each mesocosm contained 20 guppies: 10 treated (parasite-free) and 10 untreated (potentially infected). Predators were added to seven of the ten mesocosms, leaving three predator-free controls. We used more predator mesocosms than predator-free controls to buffer against potential predator loss (e.g., escape, or predation on the predators). Mesocosms were monitored daily, and the experiment was terminated after 14 days, when predator mesocosms contained approximately half of their initial guppy

numbers. All fish were then removed, euthanized with an overdose of MS-222, measured again for length and mass, and preserved in 7% buffered formalin for later dissection. Dissections followed Rogowski et al. (2020) and involved the removal of the intestine and the visual assessment of *Camallanus cotti* presence or absence.

We analyzed survival using generalized linear mixed-effects models (GLMMs) implemented with the glmer function in the *lme4* package in R (Bates et al. 2015; R Core Team 2020). Survival was treated as a binomial response (1 = survived, 0 = died) with a logit link. Fixed effects included predator presence, parasite treatment (treated vs. untreated), and their interaction. Mesocosm identity was included as a random effect to account for the split-plot design. We evaluated fixed effects using Type III Wald χ^2 tests.

We measured guppy body size before and after mesocosm occupation to evaluate how predator presence shapes size-specific survival and alters the final size distribution of guppies. Predators often target smaller or compromised individuals, so comparing average sizes across the predator-present and predator-absent mesocosms enables us to detect whether predation disproportionately eliminated particular size classes. This approach provides a mechanistic assessment of how predator presence filters individuals over the course of the experiment and clarifies whether observed size differences reflect differences in growth driven by parasites or selective mortality.

Results

Predator presence strongly influenced guppy survival. Across all mesocosms, adding a predator reduced overall survival by 19.2% ($\chi^2 = 6.13$, $p = 0.013$; Figure 1). However, the effect of predators depended on parasite status. We detected a marginally significant interaction between predator presence and parasite treatment ($\chi^2 = 3.78$, $p = 0.052$), indicating that parasitized and unparasitized guppies differed in survival only when predators were present. In predator-free mesocosms, survival did not differ between parasitized and unparasitized individuals ($\chi^2 = 1.43$, $p = 0.231$). In contrast, in predator treatments, parasitized guppies experienced substantially higher mortality, with 25.3% lower survival than unparasitized guppies, corresponding to a 1.34-fold increase in mortality risk (Figure 1).

Body length changed significantly over the course of the experiment (Time effect: $p = 0.014$). In addition, predator presence and parasite treatment interacted to influence overall body length (Cichlid × Treated: $p = 0.028$). However, the three-way interaction testing whether changes in body length differed across predator × parasite treatments was not significant (Time × Cichlid × Treated: $p = 0.188$), and all other main effects and interactions were non-significant.

Predator presence and parasite treatment independently determine whether guppies are found to be parasitized. In the additive model, predator-present mesocosms show significantly lower parasite presence ($p = 0.019$), additionally untreated fish exhibit significantly higher parasite presence ($p = 0.018$). Because the Cichlid × Treatment interaction is non-significant and produces extreme standard errors in the full model,

thus indicating a lack of statistical support for the significance of the interaction, we treat these effects as independent in our analysis.

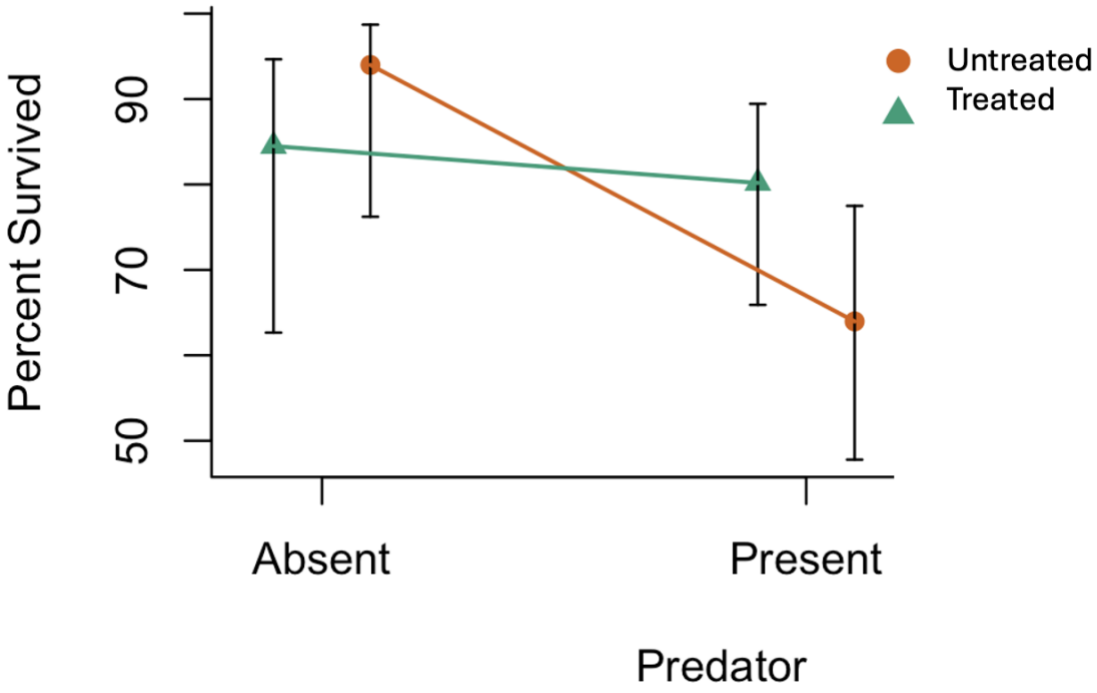

Figure 4 Guppy Survival By Treatment

Percentage of guppies surviving by treatment. "Absent" is those mesocosms not containing pike cichlids and "Present" is those where a pike cichlid was present.

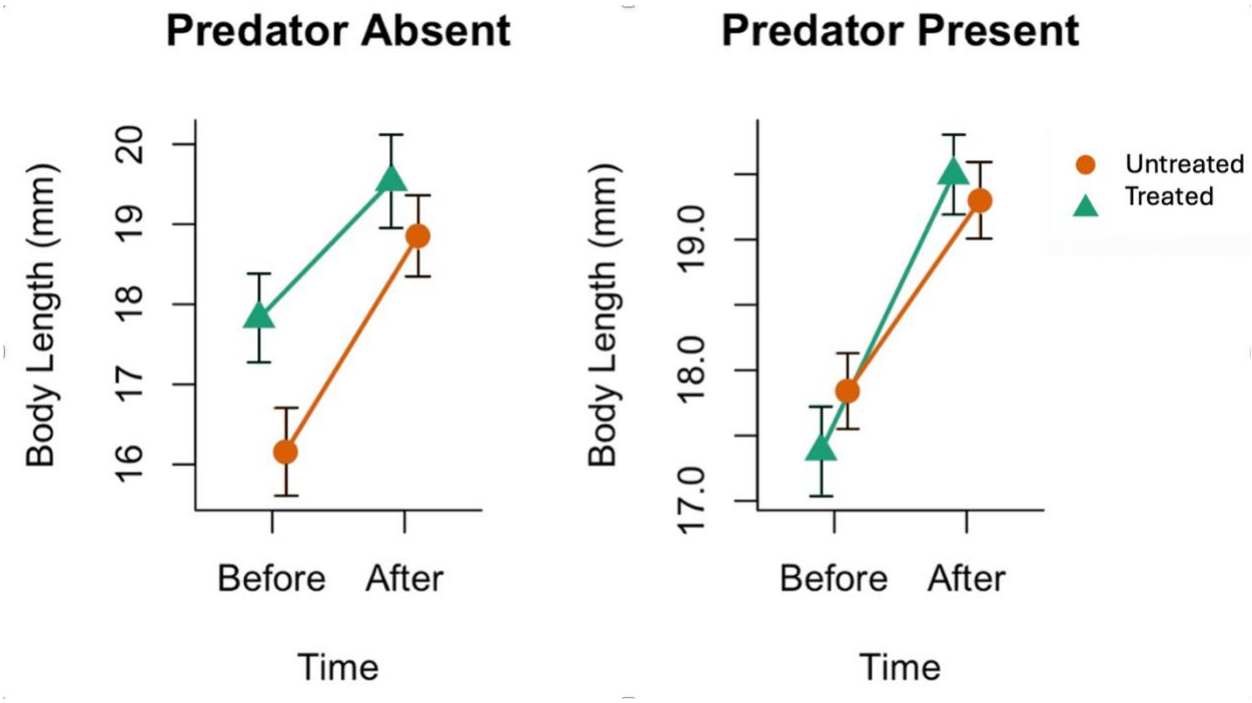

Figure 5 Guppy Size Change By Treatment

Average body length (mm) of unparasitized and parasitized guppies measured before and after the experimental period under predator absent and predator present conditions.

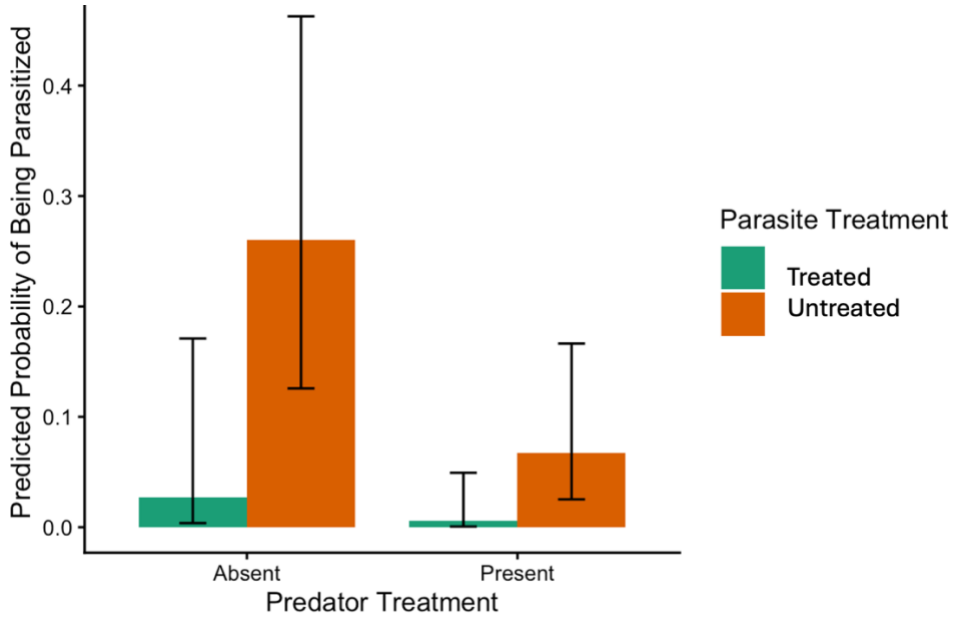

Figure 6 Guppy Parasite State By Treatment

Predicted probability of guppies being parasitized across predator and parasite treatments from the additive logistic model. Predator present mesocosms show lower parasite presence, while untreated fish show higher parasite presence. Error bars show 95% confidence intervals.

Discussion

Our experiment tested whether predators reduce *Camallanus cotti* prevalence in Trinidadian stream communities by selectively removing parasitized hosts. Consistent with this prediction, parasitized guppies experienced substantially higher mortality than unparasitized guppies, but only in the presence of predators. In predator-free mesocosms, survival did not differ significantly between parasitized and unparasitized individuals, indicating that infection alone did not reduce survival in the absence of predation. Body-size patterns further support selective removal: in mesocosms without predators, unparasitized guppies remained larger than parasitized fish throughout the experiment, whereas in mesocosms with predators this size difference grew after the experiment, consistent with predators disproportionately eliminating smaller and or parasitized individuals. The magnitude of selective mortality (25.3% lower survival for parasitized individuals) exceeded the average effect of predators on overall survival (19.2%), reinforcing the conclusion that selective predation is a strong driver of mortality for infected guppies.

These results support the Healthy Herd Hypothesis, which proposes that predators can reduce pathogen prevalence by disproportionately consuming infected hosts. In our system, predators reversed the survival pattern between parasitized and unparasitized guppies: infected individuals suffered higher mortality only when predators were present. This pattern aligns with previous studies demonstrating selective removal of infected hosts across diverse ecological systems, including daphnia-bacteria-fish (Duffy et al. 2005), frog–ranavirus–dragonfly larvae systems

(Gallagher et al. 2018), and rodent–helminth–carnivore communities (Richards et al. 2023). In each of these systems, predators disproportionately consumed infected individuals, reducing potential pathogen transmission. Our results are consistent with these patterns but provide a rare experimental test in a vertebrate freshwater system under semi-natural conditions, where selective predation has been hypothesized but seldom quantified. Unlike some studies that report weak or inconsistent selective predation (Richards 2021), our experiment detected a clear interaction between predator presence and parasite status, suggesting that the strength of selective predation may depend on ecological context, predator identity, or parasite traits such as host behavior manipulation. By demonstrating selective removal in a natural predator–prey–parasite assemblage, our study helps bridge the gap between observational patterns in Trinidadian streams and experimental evidence for the mechanisms underlying them.

However, predation is not the only ecological interaction capable of influencing pathogen prevalence. In Trinidadian streams, high-diversity lowland communities contain more predators but also more competitors. Thus, although selective predation may contribute to lower parasite prevalence in these communities, competition-driven reductions in host density may also play a role. Future studies that manipulate both predation and competition could help disentangle their relative contributions to disease dynamics.

Our interpretation relies on two assumptions about host infection status. First, although we could not directly verify that all treated fish were parasite-free at the start of

the experiment, dissections of surviving treated fish confirmed that the deworming protocol was effective. Second, we could not determine the exact proportion of untreated fish that were infected at the beginning of the experiment. Surveys from the source population indicate that approximately one-third of guppies are parasitized (Mohammed, personal communication), suggesting that many untreated fish were uninfected. This uncertainty makes our test conservative: including uninfected individuals in the untreated group would reduce the apparent difference in survival between parasitized and unparasitized fish.

Finally, our results raise the possibility that predators themselves may act as hosts for *C. cotti*. One pike cichlid kept in the laboratory and fed untreated guppies throughout the experiment was found to be infected upon dissection, indicating that predators may acquire the parasite through the consumption of infected prey. Whether predators contribute meaningfully to parasite transmission in natural communities remains unclear, but this possibility warrants further investigation.

Overall, our study adds to growing evidence that predators can influence disease outcomes not only by reducing host density but also by selectively removing infected individuals. Understanding how these pathways interact will be essential for predicting how biodiversity, community structure, and species interactions shape pathogen prevalence in freshwater and other ecosystems, particularly in the face of novel pathogens.

Conclusion

This thesis examined how predators shape host–parasite interactions across ecological, epidemiological, and evolutionary scales, using Trinidadian guppies, their predators, and the nematode *Camallanus cotti* as an experimentally tractable model system. Through a synthesis of decades of theory and empirical work, I showed that predators have the potential to influence parasite dynamics through multiple pathways: by selectively removing infected individuals, by altering host behavior and physiology, and by restructuring community composition in ways that affect transmission. The literature review highlighted that although predator effects on disease have been widely discussed, empirical tests remain uneven across systems, and mechanistic clarity is often lacking. In particular, the Healthy Herd Hypothesis — the idea that predators can reduce disease by disproportionately removing infected prey — has strong theoretical support but limited experimental validation, especially in aquatic environments. This gap motivated the mesocosm experiment that forms the core of this thesis.

Across replicated mesocosms, predator presence significantly altered guppy survival and parasite prevalence. Predators reduced overall guppy survival, but this effect depended strongly on parasite status: parasitized individuals suffered substantially higher mortality only when predators were present. This pattern provides direct experimental evidence that predators selectively remove infected prey. These results align with predictions from the Healthy Herd Hypothesis and demonstrate that predator selectivity can suppress parasite prevalence by removing infected hosts before they contribute to transmission.

Patterns of parasite presence reinforce the conclusion that predators reduce parasite burden independently of parasite treatment. Predator-present mesocosms contained fewer parasitized individuals, while untreated fish showed higher parasite presence. Because the predator × parasite interaction was statistically unsupported, these effects appear to operate additively. This suggests that predators can reduce parasite prevalence even when hosts differ in exposure history, emphasizing the robustness of predator-mediated parasite suppression.

Taken together, the results of this thesis demonstrate that predators play a central role in shaping host–parasite dynamics in freshwater systems. By disproportionately removing infected individuals, predators reduce parasite prevalence thereby influencing both ecological and epidemiological processes. These findings contribute to a growing body of work showing that predators can stabilize host–parasite interactions, reduce disease transmission, and generate healthier — though sometimes smaller — prey populations. More broadly, this research underscores the importance of integrating predation into models of parasite transmission, host fitness, and community stability, particularly in multi-host systems where trophic interactions structure infection pathways.

Future research should build on these findings by quantifying predator selectivity across parasite load gradients, incorporating multiple predator species, and examining long-term eco-evolutionary consequences of predator-mediated parasite suppression. Expanding this work into natural streams would help determine how environmental complexity and natural predator diversity shape disease outcomes. Finally, integrating

empirical results with mathematical models of predator–parasite–host dynamics would provide predictive insight into how community structure influences disease risk across ecosystems. Together, these efforts will deepen our understanding of how predators influence disease dynamics, host evolution, and ecosystem stability, advancing both theoretical and applied perspectives in host–parasite ecology.

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Appendix A Supplementary Data
Table of Papers Used for Literature Review Figures

Paper 1 Title: A healthy but depleted herd: Predators decrease prey disease and density Taxon: Invertebrate Host Species: Invertebrate Parasite: Fungus Setting: Mesocosm
Paper 2 Title: Unhealthy herds and the predator–spreader: Understanding when predation increases disease incidence and prevalence Taxon: Other Host Species: Other Parasite: Other Setting: Other
Paper 3 Title: Keeping the herds healthy and alert: implications of predator control for infectious disease Taxon: Other Host Species: Other Parasite: Other Setting: Other
Paper 4 Title: An ecoepidemic model with prey herd behavior and predator feeding saturation response on both healthy and diseased prey Taxon: Other Host Species: Other Parasite: Other Setting: Other
Paper 5 Title: Healthy herds in the phytoplankton: the benefit of selective parasitism Taxon: Invertebrate Host Species: Algae Parasite: Fungus Setting: Field
Paper 6 Title: Season and prey identity mediate the effect of predators on parasites in rodents: a test of the healthy herds hypothesis Taxon: Mammal Host Species: Mammal Parasite: Invertebrate Setting: Field
Paper 7 Title: Regulation and Stability of a Free-Living Host-Parasite System: Trichostrongylus tenuis in Red Grouse. I. Monitoring and Parasite Reduction Experiments Taxon: Bird Host Species: Bird

Parasite: Invertebrate Setting: Field
Paper 8 Title: A further cost for the sicker sex? Evidence for male-biased parasite-induced vulnerability to predation Taxon: Fish Host Species: Fish Parasite: Invertebrate Setting: Lab
Paper 9 Title: Non-native parasite enhances susceptibility of host to native predators Taxon: Invertebrate Host Species: Invertebrate Parasite: Invertebrate Setting: Mesocosm/Field
Paper 10 Title: Predators reduce Batrachochytrium dendrobatidis infection loads in their prey Taxon: Amphibian Host Species: Amphibian Parasite: Fungus Setting: Lab
Paper 11 Title: Time-lagged effect of predators on tadpole behaviour and parasite infection Taxon: Amphibian Host Species: Amphibian Parasite: Invertebrate Setting: Lab
Paper 12 Title: Parasite susceptibility in an amphibian host is modified by salinization and predators Taxon: Amphibian Host Species: Amphibian Parasite: Invertebrate Setting: Lab
Paper 13 Title: Do predators keep prey healthy or make them sicker? A meta-analysis Taxon: Other Host Species: Other Parasite: Other Setting: Other
Paper 14 Title: Healthy herds or predator spreaders? Insights from the plankton into how predators suppress and spread disease Taxon: Invertebrate Host Species: Invertebrate Parasite: Fungus Setting: Field
Paper 15 Title: Harvesting can increase severity of wildlife disease epidemics

Taxon: Other Host Species: Other Parasite: Other Setting: Other
Paper 16 Title: Predation Can Increase the Prevalence of Infectious Disease Taxon: Other Host Species: Other Parasite: Other Setting: Other
Paper 17 Title: Cascading effects of predator activity on tick-borne disease risk Taxon: Mammal Host Species: Mammal Parasite: Invertebrate Setting: Field
Paper 18 Title: Parasites of Trinidadian guppies: evidence for sex- and age-specific trait-mediated indirect effects of predators Taxon: Fish Host Species: Fish Parasite: Invertebrate Setting: Field
Paper 19 Title: Impact of prey herd behavior and predator hunting cooperation on eco-epidemic dynamics with impulsive harvesting control: a real-data approach Taxon: Other Host Species: Other Parasite: Other Setting: Other
Paper 20 Title: Unhealthy herds: indirect effects of predators enhance two drivers of disease spread Taxon: Invertebrate Host Species: Invertebrate Parasite: Fungus Setting: Lab
Paper 21 Title: Predator–spreaders: Predation can enhance parasite success in a planktonic host–parasite system Taxon: Invertebrate Host Species: Invertebrate Parasite: Fungus Setting: Field/Lab
Paper 22 Title: Predator–pathogen interactions: synergy between mortality causes and failure of the healthy herds hypothesis Taxon: Other Host Species: Other

Parasite: Other Setting: Other
Paper 23 Title: The Impact of Selective Predation on Host-Parasite SIS Dynamics Taxon: Other Host Species: Other Parasite: Other Setting: Other
Paper 24 Title: Mechanisms by which predators mediate host–parasite interactions in aquatic systems Taxon: Other Host Species: Other Parasite: Other Setting: Other
Paper 25 Title: Predator diversity, intraguild predation, and indirect effects drive parasite transmission Taxon: Invertebrate Host Species: Invertebrate Parasite: Invertebrate Setting: Mesocosm
Paper 26 Title: Predation Can Increase the Prevalence of Infectious Disease Taxon: Other Host Species: Other Parasite: Other Setting: Other
Paper 27 Title: Predation risk, host immune response, and parasitism Taxon: Bird Host Species: Bird Parasite: Other Setting: Field
Paper 28 Title: Selective predators and their parasitized prey: Are epidemics in zooplankton under top-down control? Taxon: Invertebrate Host Species: Invertebrate Parasite: Other Setting: Field
Paper 29 Title: THE IMPACT OF PARASITE MANIPULATION AND PREDATOR FORAGING BEHAVIOR ON PREDATOR–PREY COMMUNITIES Taxon: Other Host Species: Other Parasite: Other Setting: Other

Paper 30
Title: Adding parasites to the guppy-predation story: insights from field surveys
Taxon: Fish
Host Species: Fish
Parasite: Invertebrate
Setting: Field

Paper 31
Title: Parasite-mediated predation between native and invasive amphipods
Taxon: Invertebrate
Host Species: Invertebrate
Parasite: Fungus
Setting: Field/Lab

Paper 32
Title: Plasticity of predation behaviour as a putative driving force for parasite life-cycle dynamics: the case of urban foxes
and Echinococcus multilocularis tapeworm
Taxon: Mammal
Host Species: Mammal
Parasite: Invertebrate
Setting: Field

Paper 33
Title: The effect of predation on the prevalence and aggregation of pathogens in prey
Taxon: Other
Host Species: Other
Parasite: Other
Setting: Other

Paper 34
Title: Cross-continental comparison of parasite communities in a wide-ranging carnivore suggests associations with prey diversity and host density
Taxon: Mammal
Host Species: Mammal
Parasite: Invertebrate
Setting: Field

Paper 35
Title: Fish farms, parasites, and predators: implications for salmon population dynamics
Taxon: Fish
Host Species: Fish
Parasite: Invertebrate
Setting: Field

Paper 36
Title: Recolonizing gray wolves increase parasite infection risk in their prey
Taxon: Mammal
Host Species: Mammal
Parasite: Invertebrate
Setting: Field

Paper 37
Title: MALARIA AND RISK OF PREDATION: A COMPARATIVE STUDY OF BIRDS
Taxon: Bird
Host Species: Bird

Parasite: Other Setting: Field
Paper 38 Title: The impacts of predators and parasites on wild bumblebee colonies Taxon: Invertebrate Host Species: Invertebrate Parasite: Other Setting: Field
Paper 39 Title: Influence of the transmission of parasites from prey fishes on the composition of the parasite community of a predatory fish Taxon: Fish Host Species: Fish Parasite: Invertebrate Setting: Field
Paper 40 Title: Prey, predators, parasites: intraguild predation or simpler community modules in disguise? Taxon: Other Host Species: Other Parasite: Other Setting: Other
Paper 41 Title: Insect predation reduces the abundance of a nidicolous ectoparasite Taxon: Invertebrate Host Species: Invertebrate Parasite: Invertebrate Setting: Field
Paper 42 Title: Selective Predation and Rapid Evolution Can Jointly Dampen Effects of Virulent Parasites on Daphnia Populations Taxon: Invertebrate Host Species: Invertebrate Parasite: Other Setting: Field
Paper 43 Title: Increased susceptibility to predation and altered anti-predator behaviour in an acanthocephalan-infected amphipod Taxon: Invertebrate Host Species: Invertebrate Parasite: Invertebrate Setting: Field
Paper 44 Title: The distribution of Echinococcus granulosus in moose: evidence for parasite-induced vulnerability to predation by wolves? Taxon: Mammal Host Species: Mammal

Parasite: Invertebrate Setting: Field
Paper 45 Title: Bird predation alters infestation of desert lizards by parasitic mites Taxon: Reptile Host Species: Reptile Parasite: Invertebrate Setting: Field
Paper 46 Title: Predator effects on host−parasite interactions in the eastern oyster Crassostrea virginica Taxon: Invertebrate Host Species: Invertebrate Parasite: Invertebrate Setting: Field/Lab
Paper 47 Title: Heteroxenous coccidia increase the predation risk of parasitized rodents Taxon: Mammal Host Species: Mammal Parasite: Other Setting: Other
Paper 48 Title: Habitat, predators, and hosts regulate disease in Daphnia through direct and indirect pathways Taxon: Invertebrate Host Species: Invertebrate Parasite: Fungus Setting: Other
Paper 49 Title: Does malaria infection increase the risk of predation-related mortality during bird migration? Taxon: Bird Host Species: Bird Parasite: Other Setting: Field
Paper 50 Title: Effects of the parasitic dinoflagellate Hematodinium perezi on blue crab (Callinectes sapidus) behavior and predation Taxon: Invertebrate Host Species: Invertebrate Parasite: Other Setting: Field/Lab
Paper 51 Title: Predators mediate the effects of a fungal pathogen on prey: an experiment with grasshoppers, wolf spiders, and fungal pathogens Taxon: Invertebrate Host Species: Invertebrate Parasite: Fungus Setting: Field

Paper 52
Title: Synergistic effects of predation and parasites on the overwinter survival of root voles
Taxon: Mammal
Host Species: Mammal
Parasite: Other
Setting: Mesocosm

Paper 53
Title: Impacts of Selective Predation on Infection Prevalence and Host Susceptibility
Taxon: Invertebrate
Host Species: Invertebrate
Parasite: Fungus
Setting: Lab

Paper 54
Title: Sarcocystis cernae: A parasite increasing the risk of predation of its intermediate host, Microtus arvalis
Taxon: Mammal
Host Species: Mammal
Parasite: Other
Setting: Field

Paper 55
Title: Ecological impacts of the microsporidian parasite Pleistophora mulleri on its freshwater amphipod host Gammarus duebeni celticus
Taxon: Invertebrate
Host Species: Invertebrate
Parasite: Other
Setting: Field

Paper 56
Title: Effects of Wildfire on Interactions Among Nematode Parasites, Mayfly Hosts and Trout Predators
Taxon: Invertebrate
Host Species: Invertebrate
Parasite: Invertebrate
Setting: Field

Paper 57
Title: Parasite infection impairs the shoaling behaviour of uninfected shoal members under predator attack
Taxon: Fish
Host Species: Fish
Parasite: Invertebrate
Setting: Lab

Paper 58
Title: Risk of parasite-induced predation: an experimental field study on Townsend’s voles (Microtus townsendii)
Taxon: Mammal
Host Species: Mammal
Parasite: Invertebrate
Setting: Field

Paper 59
Title: Reef Fishes Have Higher Parasite Richness at Unfished
Palmyra Atoll Compared to Fished Kiritimati Island
Taxon: Fish
Host Species: Fish

Parasite: Invertebrate
Setting: Field

Paper 60
Title: Estimates of daily mortality from a neascus trematode in age-0 shortnose sucker (Chasmistes brevirostris) and the potential impact of avian predation
Taxon: Fish
Host Species: Fish
Parasite: Invertebrate
Setting: Field

Paper 61
Title: Parasitized Grouse are More Vulnerable to Predation as Revealed by a Dog-Assisted Hunting Study
Taxon: Bird
Host Species: Bird
Parasite: Invertebrate
Setting: Field

Paper 62
Title: Do Predators Always Capture Substandard Individuals Disproportionately From Prey Populations?
Taxon: Mammal
Host Species: Mammal
Parasite: Other
Setting: Field

Paper 63
Title: Can Sublethal Parasitism Destabilize Predator-Prey Population Dynamics? A Model of Snowshoe Hares, Predators and Parasites
Taxon: Mammal
Host Species: Mammal
Parasite: Other
Setting: Other

Paper 64
Title: PARASITISM, PREDATION AND SURVIVAL OF HEN RED GROUSE LAGOPUS LAGOPUS SCOTICUS IN SPRING
Taxon: Bird
Host Species: Bird
Parasite: Invertebrate
Setting: Field

Paper 65
Title: Do helminths increase the vulnerability of released pheasants to fox predation?
Taxon: Bird
Host Species: Bird
Parasite: Invertebrate
Setting: Field

Paper 66
Title: Susceptibility of Prussian carp infected by metacercariae of Posthodiplostomum cuticola (v. Nordmann, 1832) to fish predation
Taxon: Fish
Host Species: Fish
Parasite: Invertebrate
Setting: Mesocosm

Paper 67 Title: Parasite-induced vulnerability to predation in larval anurans Taxon: Amphibian Host Species: Amphibian Parasite: Other Setting: Mesocosm
Paper 68 Title: Foraging on Prey that are Modified by Parasites Taxon: Other Host Species: Other Parasite: Other Setting: Other
Paper 69 Title: The visual ecology of selective predation: Are unhealthy hosts less stealthy hosts? Taxon: Other Host Species: Other Parasite: Other Setting: Other