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LETTER

Do predators keep prey healthy or make them sicker? A meta-analysis

Robert L. Richards[1,2] | **John M. Drake**[1,2] | **Vanessa O. Ezenwa**[1,2,3]

[1]Odum School of Ecology, University of Georgia, Athens, Georgia, USA

[2]Center for the Ecology of Infectious Diseases, University of Georgia, Athens, Georgia, USA

[3]Department of Infectious Diseases, College of Veterinary Medicine, University of Georgia, Athens, Georgia, USA

Correspondence
Robert L. Richards, Louisiana State University, Baton Rouge, LA, USA.
Email: robbielrichards@gmail.com

Present address
Vanessa O. Ezenwa, Department of Ecology and Evolutionary Biology, Yale University, New Haven, Connecticut, USA

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Abstract

Ecological theory suggests that predators can either keep prey populations healthy by reducing parasite burdens or alternatively, increase parasitism in prey. To quantify the overall magnitude and direction of the effect of predation on parasitism in prey observed in practice, we conducted a meta-analysis of 47 empirical studies. We also examined how study attributes, including parasite type and life cycle, habitat type, study design, and whether predators were able to directly consume prey contributed to variation in the predator-prey-parasite interaction. We found that the overall effect of predation on parasitism differed between parasites and parasitoids and that whether consumptive effects were present, and whether a predator was a non-host spreader of parasites, were the most important traits predicting the parasite response. Our results suggest that the mechanistic basis of predator-prey interactions strongly influences the effects of predators on parasites and that these effects, although context dependent, are predictable.

KEYWORDS
disease ecology, healthy herds hypothesis, meta-analysis, parasite, predator, trophic interaction

INTRODUCTION

Organisms navigate a complex set of interspecific interactions, among the most important being victimisation by natural enemies. Both predators (Krebs et al., 1995, 2018) and parasites (Hudson, Newborn, et al., 1992; Tompkins & Begon, 1999) can affect the population demography and dynamics of the species they attack. However, few organisms are victim to only a single natural enemy. Competition between predators of a single prey population (Holt & Lawton, 1994; Holt & Polis, 1997; Tallian et al., 2017) and between parasites within a single host organism (Ezenwa & Jolles, 2011; Jolles et al., 2008; Pedersen & Fenton, 2007) have both been studied for the effects that these interactions have on natural enemy and victim populations. But predators and parasites of a single victim population also interact indirectly through their prey. Parasites may weaken their hosts, making them easier to catch and consume (Hudson, Dobson, et al., 1992; Moore, 2002), and the killing and consuming of prey by predators also kills parasites (Borer et al., 2007; Hatcher et al., 2006), except when the predator itself becomes the next host (Kuris, 2003; Lafferty, 1999; Logiudice, 2003). Therefore, like other natural enemy interactions, interactions between predators and parasites are important to understanding the dynamics of natural populations.

Ecologists have long recognised the importance of predator-prey-parasite interactions (Hudson, Dobson, et al., 1992). Among the most influential hypotheses about the consequences of predator-prey-parasite interactions is Packer et al. (2003)'s prediction, based on a mathematical model, that predators reduce both prevalence (proportion of hosts infected) and mean intensity (number of parasites per host) of parasites in their prey. This Healthy Herds Hypothesis (HHH) phenomenon might be produced by multiple mechanisms. First, predators directly, and often preferentially, kill heavily infected hosts, decreasing the number of infected individuals and the mean infection intensity in the population. Second,

predators often reduce prey population sizes, which can decrease density dependent parasite transmission. However, subsequent conceptual and theoretical work has shown that under particular circumstances predators can increase parasite prevalences in prey (e.g. Holt & Roy, 2007). Recently, Duffy et al. (2019) laid out a framework of eight different mechanisms by which predators may influence parasite prevalence and intensitiy in prey, many resulting in either increases or decreases in parasitism under different circumstances. For example, if parasites are highly aggregated in certain individuals, as is the case with metazoans such as helminths and arthropods (Shaw & Dobson, 1995) and parasitoids (Chesson & Murdoch, 1986; Hassell, 1982), selective predation on heavily infected groups should decrease parasite prevalence whereas mean intensity and selective predation on less infected groups should have the opposite effect (Joly & Messier 2004, Duffy et al., 2019). Alternatively, although predator induced reductions in prey densities may decrease transmission of some density dependent parasites, this same phenomenon may increase the prevalence and mean intensity of parasites that actively seek their hosts in the environment due to there being fewer potential host targets (Rohr et al. 2015, Duffy et al., 2019). Non-host "predator-spreaders,' which, although they cannot become infected, facilitate parasite spread from their prey items by dispersing infectious agents more widely (Cáceres et al., 2009) may also increase prevalence and mean intensity by directly consuming infected hosts. In addition to the variety of post-infection (i.e. consumptive) interactions listed above, pre-exposure (i.e. non-consumptive) interactions between predators and prey can alter prey movement and space use behaviour (Brown et al., 1988; Creel et al., 2014; Jones & Dornhaus, 2011; Spieler, 2003) in ways that predictably increase or decrease parasite transmission (Duffy et al., 2019; Ezenwa, 2004; Patterson & Ruckstuhl, 2013). Finally, many of these processes and mechanisms are thought to operate differently or at different frequencies in aquatic and terrestrial systems (Lopez & Duffy 2021).

Congruent with theoretical expectations, empirical studies measuring the effect of predators on parasites in their prey, have documented a range of patterns. Some studies show a strong negative effect of predators on parasitism, however, others show strong positive effects. For example, experimentally increased bird predation on lizard hatchlings (*Acanthodactylus beershebensis*) decreased parasitic trombiculid mite loads in the lizards (Hawlena et al., 2010), but sunfish (*Lepomis gibbosus*) predators introduced into tanks with infected tadpoles (*Lithobates spp.*), increased trematode cercarial load in tadpole prey (Szuroczki & Richardson, 2012). Given this variation, we conducted a meta-analysis to quantify the overall magnitude and direction of the effect of predation on parasite prevalence and intensity, providing a synthesis of the empirical work done to date on this topic. We also examined whether the outcome of this interaction was predictable based on the underlying context and study design. We emphasise that although ours and many prior studies use the phrasing 'effect of predators on parasites in prey', actual measurements of prevalence and intensity are fundamentally prey-focused, rather than a meaningful measure of parasite fitness or population densities. In contrast to qualitative syntheses of predator-prey-parasite interactions published over the past 20 years (Ostfeld & Holt, 2004; Hatcher et al., 2006; Duffy et al., 2019; Lopez & Duffy 2021), we used an approach that allowed us to: (1) quantify the typical effect of predators on parasites in their prey and (2) identify the most important drivers of variation in this response.

To achieve our latter goal, we tested the prediction that differences among studies explain variation in observed parasite responses along multiple axes: (1) parasite type, (2) host habitat type, (3) parasite life cycle complexity, (4) whether predator consumptive effects were present, and (5) whether predators acted as non-host parasite spreaders. Although we collected data on additional factors of interest, such as transmission mode, these factors were either too limited in variation or too highly associated with another factor for inclusion in all statistical models. Specifically, we predicted that effects of predators on metazoan parasites and parasitoids would be stronger than effects on other parasites (protozoa, bacteria, viruses, fungi), because metazoans and parasitoids tend to be highly aggregated among hosts and spatial locations (Chesson & Murdoch, 1986; Hassell, 1982; Shaw & Dobson, 1995) allowing small amounts of selective predation to nearly eliminate parasite populations (Duffy et al., 2019). Parasitoids, which are not frequently considered in predator-prey-parasite theory, have free-living adult stages which may fall prey to or avoid predators of their hosts (Brodeur & Rosenheim, 2000; Heimpel et al., 1997). Thus, predation should necessarily affect parasitoids via more varied mechanisms than other parasites, including via selective predation, shifts in community structure, and behavioural effects on the adult parasitoids themselves (Duffy et al., 2019). Helminth parasite species with complex life cycles often actively quest through the environment for their intermediate hosts (Buck & Lutterschmidt, 2017) and ectoparasites such as ticks characteristically quest as well (Mejlon & Jaenson, 1997). Because complex life cycle parasites in our study were largely limited to questing arthropods and helminths in their intermediate hosts, we predicted that predators would increase parasite prevalences and mean loads in these systems as compared to parasites with simple life cycles. Although theory suggests that both consumptive and non-consumptive effects can increase or decrease parasite prevalence and intensity in prey, we follow the predictions of the healthy herds hypothesis in predicting that consumptive predatory interactions would decrease parasitism more than non-consumptive interactions, except when consumptive effects facilitate parasite spread ('predator-spreaders'). In summary, we

asked: (1) what is the average overall effect of predators on parasites in their prey, among the studies examined, and (2) does this effect vary by parasite type, parasite life cycle, host habitat, whether consumptive effects of predators were present, and whether the predator acted as a non-host parasite spreader? We expected to find that parasite prevalence and intensity decreases with increases in predation, but this effect should be stronger for metazoan parasites and parasitoids than viruses, bacteria, fungi, and protozoa, for simple life cycle parasites than for complex life cycle parasites, for terrestrial hosts than for aquatic hosts, and for interactions involving consumptive than non-consumptive interactions. We also expected that consumptive interactions involving identified 'predator-spreaders' should increase parasite prevalence and intensity. Finally, we expected to use patterns found in this meta-analysis to identify high priority areas for additional empirical study.

MATERIALS AND METHODS

Study search and screening

To identify candidate studies we performed a systematic search of the Web of Science Core Collection using the following search string: predat* AND (parasit* OR pathogen*). This search identified 11,417 candidate studies. Abstracts were subsequently screened to determine if they met three strict inclusion criteria: they must have (1) involved an animal host/prey population, a predator population that kills and consumes the host/prey, and a parasite that is described in the study as infecting the host/prey but NOT the predator; (2) observed multiple levels of predation pressure, and (3) measured at least one relevant parasite outcome (e.g. intensity or prevalence). Based on abstract screening 256 studies were identified as potentially meeting these three criteria, 49 of which were confirmed following full-text screening (Figure S1).

Effect size and study trait variable extraction

We recorded the following information from each study to allow direct comparison of effect sizes, test the effect of study features (moderators) on this effect, and control for variation between studies: host/prey taxa to test for a phylogenetic trend in our models; parasite type (helminth, arthropod, virus, bacterium, protozoan, fungus, parasitoid), host habitat type (aquatic or terrestrial), parasite life cycle type (simple or complex), study design (observational or experimental), whether consumptive effects were included (non-consumptive effects only vs. both consumptive and non-consumptive effects), and predator-spreader identity (predator spreader or not) for inclusion in mixed effects models (MEMs) testing the effect of these moderators on effect sizes. We also collected data on other potential factors of interest, such as transmission mode which was too limited in variation across studies for inclusion (nearly all parasites studied used environmental transmission). Most studies (44 of 49) included a binary comparison of a parasite response across two levels of predation, and many were analysed using multivariate statistics which makes statistical comparison of effect sizes across studies challenging (Borenstein et al., 2017). For this reason, we extracted the mean parasite response value, sample size, and measure of variation (typically SE, SD, or 95% CI) from the text or figures of each of these studies and calculated the standardised mean difference (Hedges g) using the *escalc* function in the R package metafor (Viechtbauer, 2010). A few studies (5 of 49) reported parasite responses over a range of predation pressures. We converted responses from 3 of these studies to binary effect sizes by using raw data provided to compare the mean parasite response for samples in the first quartile of predator abundance to those in the 4th quartile of predator abundance. We excluded studies from further analysis if sufficient data for this procedure were not provided. Following this protocol, we extracted 187 effect sizes from 47 studies (Table 1).

Not all effect sizes contain the same type of information because of differences in parasite biology and in the associated response metric. For our study, we grouped effect sizes into 2 broad categories based on the parasite response that was measured: (1) the number or proportion of hosts infected (quantified as prevalence, number or density of infected individuals, or disease induced mortality rate; $n = 83$ effect sizes from 21 different studies, Table 2) and (2) the number of parasites in an average individual (quantified as parasite intensity or parasite load; $n = 61$ effect sizes from 19 different studies). Because we expected that predators would have different effects on prevalence and intensity measures (for example limited selective predation on a population with highly aggregated parasites may have a large effect on mean intensity but a small effect on prevalence), we analysed these responses separately. Another distinction we made was to separate parasites from parasitoids ($n = 43$ effect sizes from 11 different studies). Parasitoids behave like both predators and parasites over the course of their life cycle. Adult parasitoids are free-living flies and wasps that lay eggs on live hosts, but the juvenile parasitoids that hatch from these eggs are obligately parasitic and typically lethal to the host. Consequently, the effect of predators on parasitoids in prey may result from different processes than the effects on typical parasites.

Statistical analysis

Main effect and publication bias

We analysed effect size data for each of the three categories of our data (prevalence, intensity, parasitoid)

TABLE 1 All studies included in meta-analysis

Hosts (common name)	Hosts (binomial)	Predators (common name)	Predators (binomial)	Parasites	Parasite type	Interaction type	Citation
American bullfrog, wood frog, green frog	*Lithobates catesbeianus, Lithobates sylvaticus, Lithobates clamitans*	Pumpkinseed sunfish	*Lepomis gibbosus*	*Echinostoma trivolvis*	Helminth	NonConsumptive	Szuroczki and Richardson, (2018)
Wood frog	*Lithobates sylvaticus*	Green darner	*Anax junius*	*Echinostoma*	Helminth	NonConsumptive	Buss and Hua, (2018)
Black bean aphid	*Aphis fabae*	Two-spotted ladybug	*Adalia bipunctata*	*Lecanicillium muscarium*	Fungus	Consumptive	Mohammed (2018)
Freshwater snail	*Biomphalaria glabrata, Bulinus truncatus*	Blue crayfish, giant waterbug	*Procambarus alleni, Belostoma flumineum*	*Schistosoma haemotobium*	Helminth	Consumptive	Halstead et al. (2018)
Tomato leafminer	*Tuta absoluta*	Plant bug	*Macrolophus pygmaeus*	*Stenomesius japonicus*	Parasitoid	Consumptive	Chailleux et al. (2017)
American toad	*Anaxyrus americanus*	Dragonfly	*Anax sp.*	*Echinoparyphium sp.*	Helminth	NonConsumptive	Koprivnikar and Urichuk, (2017)
Bank vole, wood mouse	*Myodes glareolus, Apodemus sylvaticus*	Red fox, European pine marten, beech marten, European polecat	*Vulpes vulpes, Martes martes, Martes foina, Mustela putorious*	*Ixodes ricinus*	Arthropod	Consumptive	Hofmeester et al. (2017)
Tomato leafminer	*Tuta absoluta*	Tomato bug	*Nesidiocoris tenuis*	*Bracon nigricans, Necremnus tutae*	Parasitoid	Consumptive	Naselli et al. (2017)
Potato psyllid	*Bactericera cockerelli*	Plant bug	*Dicyphus hesperus*	*Tamarixia triozae*	Parasitoid	Consumptive	Ramirez-Ahuja et al. (2017)
Water flea	*Daphnia dentifera*	Phantom midge	*Chaoborus punctipennis*	*Metschnikowia bicuspidata*	Fungus	Consumptive	Strauss et al. (2016)
Eastern oyster	*Crassotrea virginica*	Flatback mud crab	*Eurypanopeus depressus*	*Perkinsus marinus, Haplosporidium nelsoni*	Protozoa	Consumptive, NonConsumptive	Malek and Byers, (2016)
Silverleaf whitefly	*Bemisia tabaci*	Harlequin ladybeetle	*Harmonia axyridis*	*Encarsia formosa, Encarsia sophia*	Parasitoid	Consumptive	Tan et al. (2016)
Green frog	*Lithobates clamitans*	Green darner, comet darner	*Anax junius/longipes*	*Echinostoma revolutum*	Helminth	NonConsumptive	Marino et al. (2016)
Red spider mite	*Tetranychus urticae*	Predatory mite	*Phytoseiulus persimilis*	*Neozygites floridana*	Fungus	Consumptive	Trandem et al. (2016)
Mosquito	*Anopheles coluzzii*	Aquatic backswimmer	*Anisops jaczewskii*	*Plasmodium falciparum*	Protozoa	NonConsumptive	Roux et al. (2015)
Pink salmon, chum salmon	*Oncorhynchus gorbuscha, Oncorhynchus keta*	Unspecified	*Unspecified*	*Lepeophtheirus salmonis*	Arthropod	Consumptive	Peacock et al. (2015)

TABLE 1 (Continued)

Hosts (common name)	Hosts (binomial)	Predators (common name)	Predators (binomial)	Parasites	Parasite type	Interaction type	Citation
Freshwater snail	*Bulinus globosus, Bulinus truncatus*	African river prawn	*Macrobrachium vollenhovenii*	*Schistosoma hematobium*	Helminth	Consumptive	Sokolow et al. (2015)
Wood frog	*Lithobates sylvaticus*	Diving beetle	*Dytiscus spp.*	*Batrachochytrium dendrobatidis*	Fungus	NonConsumptive	Groner and Relyea, (2015)
Guppy	*Poecilia reticulata*	Killifish, blue acara, brown coscarob, Guyana leaffish, millet, wolf fish, sleeper goby	*Anablepsoides hartii, Aequidens pulcher, Cichlasoma taenia, Polycentrus schomburgkii, Crenichla alta, Hoplias malabaricus, Gobiomorous dormitor*	*Gyrodactylus spp.*	Helminth	Consumptive	Stephenson et al. (2015)
Tomato leafminer	*Tuta absoluta*	Plant bug	*Macrolophus pygmaeus*	*Stenomesius japonicus*	Parasitoid	Consumptive	Chailleux et al. (2014)
Northern pacific treefrog	*Pseudacris regilla*	Dragonfly	*Anax sp.*	*Ribeiroia ondatrae*	Helminth	NonConsumptive	Orlofske et al. (2014)
Water flea	*Daphnia dentifera*	Midge	*Chaoborus sp.*	*Metschnikowia bicuspidata*	Fungus	Consumptive	Penczykowski et al. (2014)
Green frog	*Lithobates clamitans*	Dragonfly	*Anax sp.*	*Echinostomatidae*	Helminth	Consumptive, NonConsumptive	Marino and Werner, (2013)
Water flea	*Daphnia dentifera*	Bluegill, phantom midge	*Lepomis macrochirus, Chaoborus punctipennis*	*Metschnikowia*	Fungus	NonConsumptive	Bertram et al. (2013)
Wood frog	*Lithobates sylvaticus*	Diving beetles, skimmers, darners, emerald dragonflies	*Dyticidae, Libellulidae, Aeshnidae, Cordululiidae*	*Ranavirus*	Virus	NonConsumptive	Reeve et al. (2013)
Gypsy moth	*Lymantria dispar*	Common black ant	*Formica fusca*	*Nosema lymantriae, Vairimorpha disparis*	Protozoan, fungus	Consumptive	Goertz and Hoch, (2013)
Cassava green mite	*Mononychellus tanajoa*	Predatory mite	*Typhlodromalus aripo*	*Neozygites tanajoae*	Fungus	Consumptive	Agboton et al. (2013)
Guppy	*Poecilia reticulata*	Unspecified	Unspecified	*Gyrodactylus turnbulli*	Helminth	Consumptive, NonConsumptive	Perez-Jvostov et al. (2012)

(Continues)

TABLE 1 (Continued)

Hosts (common name)	Hosts (binomial)	Predators (common name)	Predators (binomial)	Parasites	Parasite type	Interaction type	Citation
Green frog, wood frog, upland chorus frog, Cope's gray treefrog	*Lithobates clamitans, Lithobates sylvaticus, Pseudacris feriarum, Hyla chrysoscelis*	Dragonfly, giant waterbug	*Anax sp., Belostoma flumineum*	*Ranavirus*	Virus	NonConsumptive	Haislip et al. (2012)
Water flea	*Daphnia magna*	Ide	*Leuciscus idus*	*Pasteuria ramosa*	Bacteria	NonConsumptive	Jansen et al. (2011)
Water flea	*Daphnia longispina*	European bitterling	*Rhodeus sericeus amarus*	*Metschnikowia*	Fungus	NonConsumptive	Yin et al. (2011)
American toad	*Anaxyrus americanus*	Eastern newt	*Notphthalmus viridescens*	*Echinostoma trivolvis*	Helminth	NonConsumptive	Raffel et al. (2010)
Small ground finch	*Geospiza fuliginosa*	Black rat, house mouse, domestic cat, domestic dog, smooth-billed ani	*Rattus rattus, Mus musculus, Felis catus, Canis lupus familiaris, Crotophaga ani*	*Philornis downsi*	Arthropod	Consumptive	O'Connor et al. (2010)
Be'er Sheva fringe-fingered lizard	*Acanthodactylus beershebensis*	Iberian grey shrike, common kestrel	*Lanius meridionalis, Falco tinnunculus*	*Trombiculid mites*	Arthropod	Consumptive	Hawlena et al. (2010)
Northern grasshopper	*Melanoplus borealis*	Wolf spider	*Hogna spp.*	*Entomophaga grylli*	Fungus	Consumptive	Laws et al. (2009)
Water flea	*Daphnia dentifera*	Phantom midge	*Chaoborus punctipennis*	*Metschnikowia bicuspidata*	Fungus	Consumptive	Cáceres et al. (2009)
Pea aphid	*Acyrthosiphon pisum*	Seven-spotted ladybug	*Coccinella septempunctata*	*Pandora neoaphidis*	Fungus	Consumptive	Baverstock et al. (2009)
Water flea	*Daphnia magna*	Phantom midge	*Chaoborus crystallinus*	*Pasteuria ramosa*	Bacteria	NonConsumptive	Coors and De Meester (2008)
Soybean aphid	*Aphis glycines*	Unspecified	Unspecified	*Aphidius colemani*	Parasitoid	Consumptive	Chacon et al. (2008)
Guppy	*Poecilia reticulata*	Millet	*Crenicichla alta*	*Gyrodactylus spp.*	Helminth	Consumptive	Martin and Johnsen, (2007)
Green citrus aphid	*Aphis spiraecola*	Beetle	*Scymnus posticalis*	*Lysiphlebus japonicus*	Parasitoid	Consumptive	Kaneko (2007)
Aphids	*Microlophium carnosum, Uroleucon, Acyrthosiphon pisum*	Seven-spotted ladybug	*Coccinella septempunctata*	*Pandora neoaphidis*	Fungus	Consumptive	Ekesi et al. (2005)

TABLE 1 (Continued)

Hosts (common name)	Hosts (binomial)	Predators (common name)	Predators (binomial)	Parasites	Parasite type	Interaction type	Citation
Diamondback moth	*Plutella xylostella*	Red imported fire ant	*Solenopsis invicta*	*Diadegma insulare* *Cotesia plutellae,* *Chetogena scutellaris,* *Diadegma insulare* *Cotesia plutellae,* *Chetogena scutellaris*	Parasitoid	Consumptive	Harvey and Eubanks, (2005)
Cotton aphid	*Aphis gossypii*	Convergent ladybug	*Hippodamia convergens*	*Lysiphlebus testaceipes*	Parasitoid	Consumptive	Colfer and Rosenheim, (2001)
Wood frog, green frog	*Lithobates sylvaticus,* *Lithobates clamitans*	Banded killifish	*Fundulus diaphanus*	*Echinostoma sp.*	Helminth	NonConsumptive	Thiemann and Wassersug, (2000)
Pea aphid	*Acyrthosiphon pisum*	Seven-spotted ladybug	*Coccinella septempunctata*	*Erynia neoaphidis*	Fungus	Consumptive	Roy et al. (1998)
Aphid	*Uroleucon*	Spotless ladybug	*Cycloneda sanguinea*	*Aphidius floridaensis*	Parasitoid	Consumptive	Ferguson and Stiling, (1996)
Tortoise beetle	*Chalepus*	Unspecified	Unspecified	21 species	Parasitoid	Consumptive	Memmott et al. (1993)

Note: Additional information on studies available in the full dataset included in published data (see data accessibility statement).

according to the following scheme. First, we fit a random effects model (REM) to estimate the overall effect of predators on parasites in prey, within these studies. We report the size and direction of the overall effect as well as I^2, a measure of heterogeneity that can be interpreted as the proportion of total variation that is due to between study variation (Higgins & Thompson, 2002). We also used these models to diagnose publication bias in the data by visualising the relationship between effect size and variance with a funnel plot and testing for a significant correlation between these traits using a rank-order correlation test. If significant correlation was detected, we used the trim-and-fill method (Duval & Tweedie, 2000) to determine whether introducing studies to balance the diagnosed bias would alter the main effect. Trim-and-fill involves identifying identify asymmetric positive or negative outliers in the funnel plot and trimming these from the funnel to estimate the symmetrical centre of the funnel. We then restore the asymmetric outliers along with imputed counterparts on the opposite side of the funnel centre before refitting the REM to the reconstructed data. Because of shared evolutionary history, closely related host species may have similar effect sizes. Using Pagel's lambda (Pagel, 1999) we failed to detect evidence of phylogenetic dependence in any dataset (see Supplementary Methods).

Effects of moderators

Given the variation in the effect of predators on parasites in prey, we were interested in identifying attributes of the study or study system that were most important for explaining variation in effect sizes across studies. To do this, we fit mixed effects models (MEM) to the prevalence and intensity effect size data sets, including a series of binary moderators: whether consumptive effects were included (non-consumptive effects only vs. consumptive and non-consumptive effects), predator-spreader identity (predator spreader vs. not), parasite type (metazoan: helminths, arthropods vs. other: viruses, bacteria, protozoa, fungi), host habitat type (aquatic vs. terrestrial), parasite life cycle (simple vs. complex) and all two-way interactions. We note that because studies in which the predator was described as a viable host for the parasite were excluded, all predator-spreaders are non-host spreaders. Habitat type was not included in the intensity model because it was too strongly associated with consumptive effect inclusion among intensity studies ($\varphi > 0.8$). Study design (experimental vs. observational) was also included as a moderator to control for variation in responses but without a particular hypothesis. We also included study as a random effect. We note that although we were interested in the distinction between non-consumptive and consumptive effects, most consumptive effect studies technically allowed for both non-consumptive and consumptive interactions due to

TABLE 2 Enumeration of effect sizes categorized in nested subsets of moderators: Observational/Experimental, Metazoan (Helminth or Arthropod)/Other (Virus, bacteria, fungus, or protozoa), Non-Consumptive/All interactions, Aquatic/Terrestrial habitat, Complex/Simple life cycle, Predator-spreader/Non predator-spreader

Prevalence

Observational (6)

Parasite type

Metazoans (4)

Interaction type

All (4)

Habitat type: Aquatic (4) | Terrestrial (0)

Complex cycle?: Yes (0) | No (4)

Predator spreader?: Yes (0) | No (4)

Non-Consumptive (0)

Habitat type: Aquatic (0) | Terrestrial (0)

Complex cycle?: Yes (0) | No (0)

Other (2)

Interaction type

All (2)

Habitat type: Aquatic (2) | Terrestrial (0)

Complex cycle?: Yes (0) | No (2)

Predator Spreader?: Yes (2) | No (0)

Non-Consumptive (0)

Habitat type: Aquatic (0) | Terrestrial (0)

Complex cycle?: Yes (0) | No (0)

Experimental (77)

Parasite type

Metazoans (19)

Interaction type

All (18)

Habitat type: Aquatic (18) | Terrestrial (0)

Complex cycle?: Yes (18) | No (0)

Predator spreader?: Yes (0) | No (18)

Non-Consumptive (1)

Habitat type: Aquatic (1) | Terrestrial (0)

Complex cycle?: Yes (0) | No (1)

Other (58)

Interaction type

All (40)

Habitat type: Aquatic (3) | Terrestrial (37)

Complex cycle?: Yes (0) | No (40)

Predator spreader?: Yes (23) | No (17)

Non-Consumptive (18)

Habitat type: Aquatic (18) | Terrestrial (0)

Complex cycle?: Yes (4) | No (14)

Intensity

Observational (18)

Parasite type

Metazoans (18)

Interaction type

All (18)

Habitat type: Aquatic (1) | Terrestrial

Complex cycle?: Yes (16) | No (2)

Predator spreader?: Yes (0) | No (18)

Non-Consumptive (0)

Habitat type: Aquatic (0) | Terrestrial (0)

Complex cycle?: Yes (0) | No (0)

Other (0)

Interaction type

All (0)

Habitat type: Aquatic (0) | Terrestrial (0)

Complex cycle?: Yes (0) | No (0)

Predator spreader?: Yes (0) | No (0)

Non-Consumptive (0)

Habitat type: Aquatic (0) | Terrestrial (0)

Complex cycle?: Yes (0) | No (0)

Experimental (43)

Parasite type

Metazoans (23)

Interaction type

All (6)

Habitat type: Aquatic (3) | Terrestrial (3)

Complex cycle?: Yes (1) | No (5)

Predator spreader?: Yes (0) | No (6)

Non-Consumptive (17)

Habitat type: Aquatic (17) | Terrestrial (0)

Complex cycle?: Yes (16) | No (1)

Other (20)

Interaction type

All (1)

Habitat type: Aquatic (1) | Terrestrial (0)

Complex cycle?: Yes (0) | No (1)

Predator spreader?: Yes (0) | No (1)

Non-Consumptive (19)

Habitat type: Aquatic (19) | Terrestrial (0)

Complex cycle?: Yes (0) | No (0)

limitations in experimental design. Therefore we draw the distinction between studies that manipulate only non-consumptive interactions and those which include consumptive interactions. From this model, we generated candidate sets of all possible MEMs for each data set and used the Akaike information criterion corrected for sample size (AICc) to compare model fit. We calculated the importance (on a scale from 0 to 1) of each moderator as the summed model weights for all MEMs in which a given moderator occurred. We then fit univariate models for each moderator to identify the direction of the effect. Because parasitoid studies were uniformly terrestrial, experimental, and consumptive, we did not fit MEMs with moderators to these data.

RESULTS

Study patterns

We identified substantial gaps in the literature for certain combinations of moderators (Table 2). In particular, no observational studies considered non-consumptive effects, no studies that measured parasitism by intensity metrics or that studied metazoan parasites manipulated the effect of known predator-spreaders, and nearly all parasites studied had an environmental transmission mode. Both parasite taxonomic categories are represented in studies measuring both prevalence and intensity but metazoans were more common in intensity studies (n = 41/61) and other parasites (viruses, bacteria, protazoa) were more common in prevalence studies (n = 60/83).

Parasite prevalence

The REM of prevalence effect sizes showed an overall effect that was significantly different from zero (z = 2.437, p = 0.015; Figure 1a). However, there was substantial

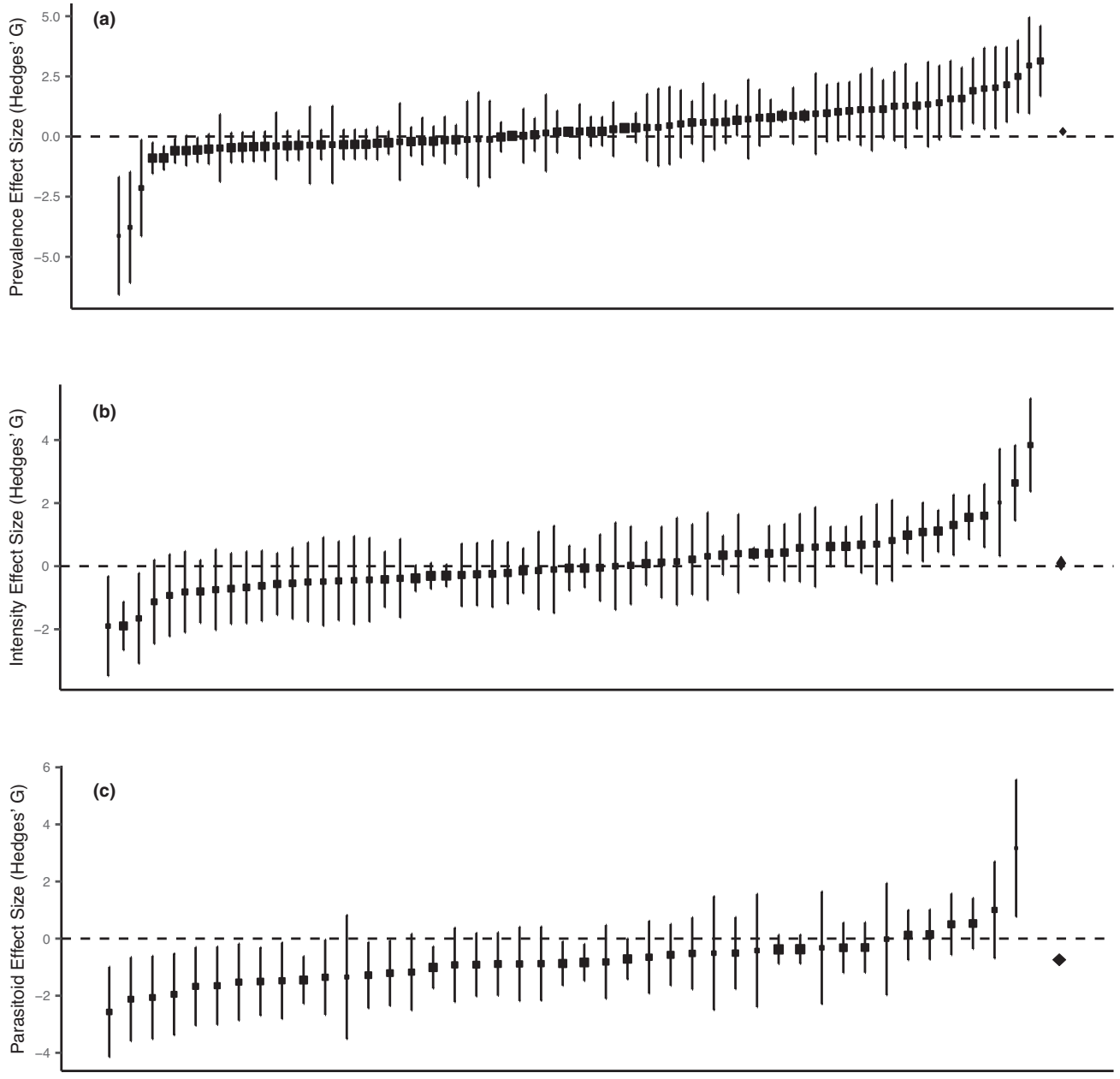

FIGURE 1 Range and grand means from random-effects meta-analysis models (REMs) for the effect of predators on parasites in prey stratified by prevalence (a) intensity (b) and parasitoid (c) data. Each square point represents a single effect size from a study in Table 1. Lines show 95% confidence intervals for effect sizes and REMs (uncorrected for publication bias). The dashed line represents no relationship between condition and infection

heterogeneity between studies (I^2 = 84.59%), and significant publication bias (τ = 0.234, p = 0.002, Figure S2). The trim-and-fill method estimated 15 missing negative studies, and inclusion of these studies eliminated the significant effect, with the modified REM showing no evidence of an effect (z = −0.054, p = 0.957). In our analysis of moderators, predator-spreader identity was included in nearly all MEMs with non-zero weights (Importance = 0.998; Figure 2a, Table 3). Host habitat type was also important (Importance = 0.922), but other main effects were less so (parasite type importance = 0.880; study design importance = 0.834; presence of consumptive effects importance = 0.834; life cycle type importance = 0.829). The most important interaction term was between whether consumptive effects were present and predator-spreader identity (Importance = 0.576). In univariate analyses, only predator-spreader identity significantly affected mean effect size (QM_1=9.692, p = 0.0019), despite significant residual heterogeneity (QE_{81} = 271.854, p < 0.001). Predator-spreaders tended to increase parasite prevalence more than non-spreader predators (Figure 3).

Parasite intensity

An REM of intensity effect sizes did not detect a statistically significant effect of increased predation on parasite intensity in prey (z = 0.829, p = 0.407; Figure 1b), with substantial heterogeneity between studies (I^2 = 75.93%), and no evidence of publication bias (τ = −0.106, p = 0.231, Figure S3). The single most important moderator was whether consumptive interactions were present (Importance = 0.667; Figure 2b, Table 3), and this was the only variable identified as a significant moderator in subsequent univariate analyses (QM_1 = 5.848, p = 0.016), despite significant residual heterogeneity (QE_{59} = 182.050, p < 0.001). Studies with just non-consumptive interactions tended to increase parasite intensity more than those which also included consumptive interactions (Figure 3).

Parasitoids

An REM of parasitoid effect sizes detected a statistically significant, negative, overall effect of predation on parasitoid abundance in prey (z = −6.919, p < 0.001; Figure 1c), with a smaller amount of heterogeneity between studies as compared to the analyses of parasite responses (I^2 = 35.47%). Although there was evidence of significant publication bias (τ = −0.227, p = 0.032, Figure S4), the inclusion of 9 missing positive effect sizes estimated by the trim-and-fill method did not eliminate the overall significant negative effect of predators on parasitoids (z = −4.630, p < 0.001).

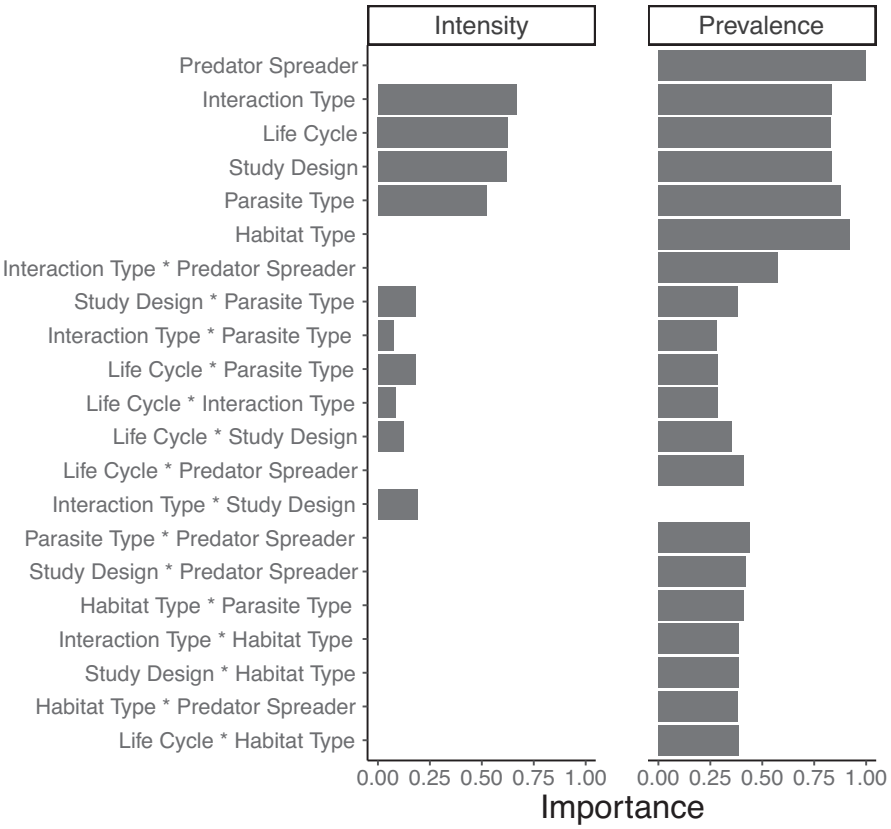

FIGURE 2 Moderator importance for mixed effects models (MEMs) of intensity and prevalence effect sizes, determined by AICc weight

TABLE 3 Ranking of mixed-effects models (MEMs) predicting effect size for the effect of predators on parasites in the prevalence and intensity data

	df	σ study	QM	QE	ΔAICc	wi
MEMs fit to prevalence data						
G ~ Habitat type + predator spreader + habitat type * predator spreader	5	0.6609	15.2042 (0.002)	270.8532 (<0.001)	0	0.002
G ~ Interaction type + predator spreader + interaction type * predator spreader	5	0.5243	17.5804 (<0.001)	241.6048 (<0.001)	0.25	0.002
G ~ Interaction type + study design + predator spreader + interaction type * predator spreader + study design * predator spreader	7	0.5198	22.1689 (<0.001)	181.4157 (<0.001)	0.5	0.001
G ~ predator spreader	3	0.5964	11.345 (<0.001)	372.8542 (<0.001)	0.63	0.001
G ~ Interaction type + habitat type + predator spreader + interaction type * predator spreader	6	0.5549	18.6789 (0.002)	240.3655 (<0.001)	0.74	0.001
G ~ Interaction type + habitat type + predator spreader + interaction type * habitat type + interaction type * predator spreader	6	0.5549	18.6789 (0.002)	240.3655 (<0.001)	0.74	0.001
G ~ Interaction type + study design + predator spreader + interaction type * predator spreader	6	0.4869	20.4384 (0.002)	183.2502 (<0.001)	0.78	0.001
MEMs fit to intensity data						
G ~ Interaction type	3	0.5049	6.5098 (0.012)	182.0501 (<0.001)	0	0.091
G ~ Interaction type + life cycle	4	0.5005	8.0635 (0.018)	180.6978 (<0.001)	0.82	0.06

Note: Models are ranked by ΔAICc with the number of model degrees of freedom (df), test statistic for the omnibus test of model coefficients (QM), test statistic for residual heterogeneity (QE), difference in corrected AIC from the best model, and Akaike weights (wi). Only MEMs with ΔAICc ≤ 1 are shown.

DISCUSSION

The healthy herds hypothesis (Packer et al., 2003) predicts that predators should have negative effects on parasites in their prey, but empirical studies testing this hypothesis have reported varied effects. This variation is unsurprising given the recent synthetic work suggesting that nearly all mechanisms by which predators can influence parasitism in prey can produce both increases and decreases in parasitism (Duffy et al., 2019). In this study, we aimed to identify patterns in both the overall effect of predators on parasites in prey across taxa and the effect of variation in predator and parasite traits on the overall effect. Using a meta-analytic approach, we found that the main effect of predators on parasites in prey differed between parasites and parasitoids but not between metazoan parasites (helminths, arthropods) and non-metazoan parasites (viruses, bacteria, fungi, protozoa), with a net negative effect only present for parasitoids. Additionally, we found that the presence of both consumptive interactions and predator-spreader interactions were most important in predicting the effect of predators on parasites in prey. These findings provide clear evidence for the growing theoretical consensus that the HHH prediction is far from universal. The degree to which the HHH holds in a given system is both parasite- and context dependent, but we were able to identify patterns in the published literature.

We observed significant heterogeneity in the magnitude and direction of the main effect of predators on parasites in prey. We, therefore, sought to determine if there were factors that explained this variation in effects. First, we found that the difference between consumptive and non-consumptive interactions can explain variation in the effect of predators on parasites, but specific mechanisms are also very important. In studies that measured intensity, the effect size significantly differed between those involving consumptive interactions and those in which only non-consumptive interactions were present, with non-consumptive interactions generally increasing parasite intensity more. This result aligns with our prediction that consumptive interactions will have more negative effects on parasites compared with non-consumptive interactions. We note that our studies involving consumptive interactions typically were open to all interactions including non-consumptive, suggesting that this result may, in fact, be conservative. Conversely, in the prevalence response model studies involving consumptive interactions and those with

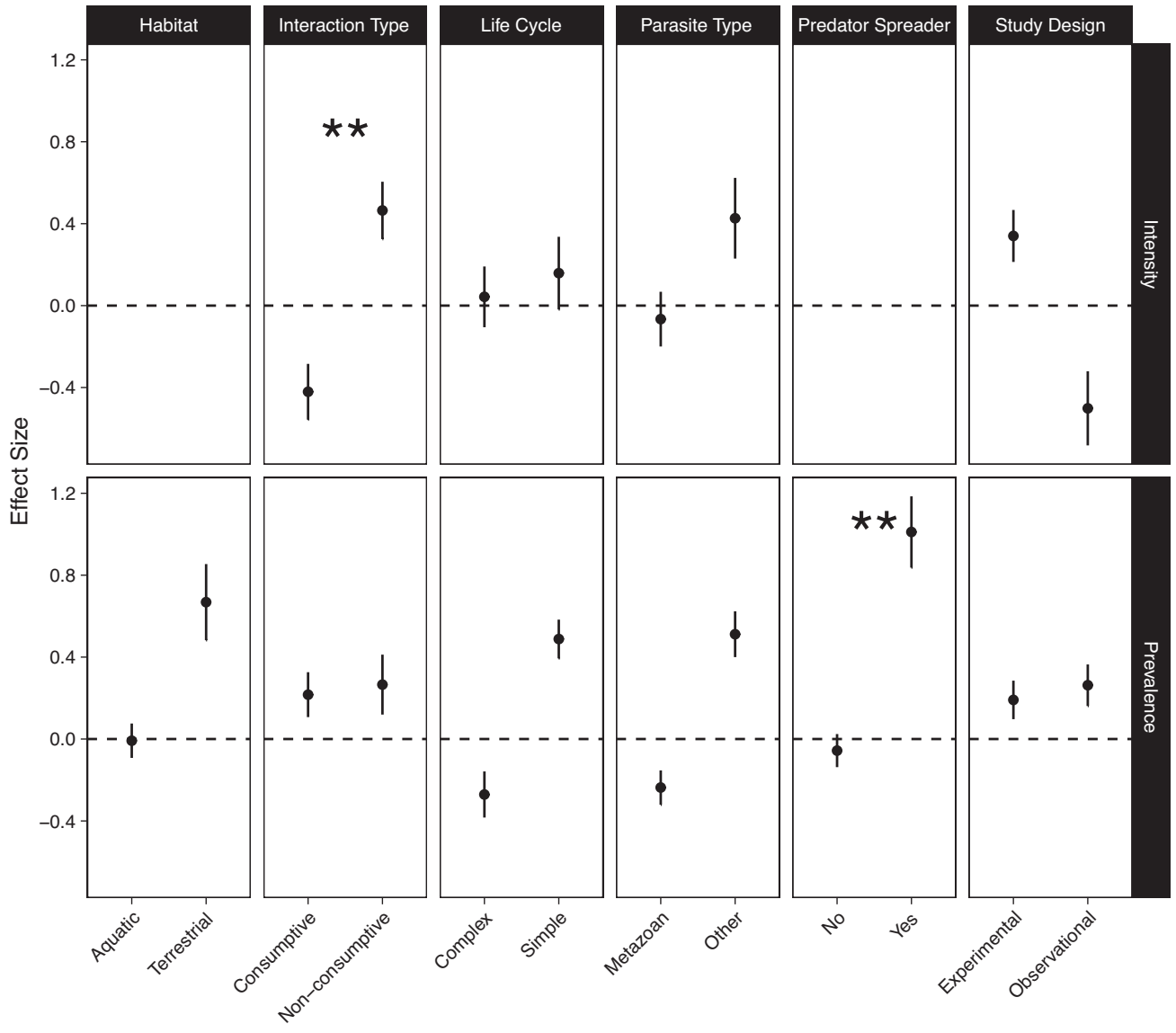

FIGURE 3 Modelled univariate relationships (means and 95% confidence intervals) for the five most important moderators of effect size across all intensity data (top row), and prevalence data (bottom row). Results from mixed-effects models are sorted by study traits. Consumptive effects allowed: yes or no. Life cycle: complex or simple. Parasite type: metazoan (helminth or arthropod) or other (virus, bacteria, fungi, protozoa). Predator-spreader identity: identified as a predator spreader or not. The dashed line represents no relationship between condition and infection and asterisks denote patterns that were significant in univariate analysis

only non-consumptive interactions were estimated to be nearly identical on average. We suggest that the difference between these two response variables is an artefact of the significant residual heterogeneity even in our best fit models. Most of this variation is likely explained by unexplored mechanisms within these studies. Duffy et al. (2019) outlined 7 independent mechanisms whereby consumption can directly or indirectly increase disease in prey. For example, predators can selectively prey on uninfected individuals, shift host population structure toward more susceptible or heavily infected classes, and suppress competition between hosts allowing them to support more parasites. Unfortunately few studies provide sufficient information to assess which mechanisms are at play. Nonetheless, we were able to directly test this idea by including one of these mechanisms (predator-spreaders; Cáceres et al., 2009) as a moderator variable since researchers typically identified this attribute of predators in studies. As expected, predator-spreader identity was highly important for predicting the parasite outcome in the prevalence dataset (predator-spreaders were absent from the intensity dataset), generally increasing parasite prevalence. Ultimately, the lack of clear support for the hypothesis that consumptive interactions should decrease parasite prevalence and intensity more than non-consumptive interactions is a result of the conflicting negative effects in studies of typical consumptive interactions versus positive effects in studies of

consumptive predator-spreader interactions and certain non-consumptive interactions.

Second, we failed to detect a clear effect of parasite traits in our analysis. We hypothesised that differences in the aggregation patterns of metazoan parasties (helminths, arthropods) and other parasites (viruses, bacteria, fungi, protozoa) would result in metazoans having a stronger and more negative response to predator pressure than non-metazoans, but found no evidence for a difference between parasite types and this variable was generally less important for explaining variation. This lack of an effect may be due to multiple factors. Although one might expect random predation, or predation on infected individuals, to decrease parasite intensity more when parasites are aggregated (Packer et al., 2003), the opposite is also true. Gape limited predators, such as many piscivorous fish and carnivorous snakes (King, 2002; Nilsson & Brönmark, 2000) that selectively prey on smaller and younger individuals may cause population demographics to shift towards larger, older and more heavily infected hosts (Byers et al., 2015; Dobson, 1989; Duffy et al., 2019; Nilsson & Brönmark, 2000). Alternatively, our assumption that high aggregation among metazoans makes them more vulnerable to predation may be countered by the existence of significant aggregation in non-metazoan parasite systems as well (Lord et al. 1999; Grogan et al. 2016). We also found limited evidence for an effect of parasite life cycle in moderating predator effects on parasite prevalence and intensity despite our prediction that complex life cycle parasites which actively seek hosts would increase with increased predator pressure. These predictions were based on the idea that predator induced decreases in host density should increase the number of parasites that attack each individual host, at least over the short time scales considered in most studies. However, complex parasites are often also metazoans and display substantial aggregation (Shaw & Dobson, 1995), as such depredation of the few heavily infected individuals may balance the increased parasite attack rate of the remaining individuals. We see therefore that the dominant mechanisms which we ascribed a priori to metazoan parasites and complex life cycle parasites may interact to obscure an overall effect of either.

Similarly, although we predicted that predators should be more likely to increase parasitism in prey in aquatic habitats than terrestrial habitats, we did not find a clear role for host habitat on the effects of predators on parasites in prey. Our ability to test this hypothesis was limited by the strong association, among intensity studies which include most non-consumptive interactions, between habitat type and whether consumptive effects were present in the study. Although we found that habitat type was an important variable explaining variation in parasite prevalence in our multivariate analysis, there was no significant relationship between habitat type and mean effect size in our univariate analysis. Furthermore, the non-significant pattern of association in the univariate analysis was the opposite of our prediction. Although studies performed in terrestrial habitats showed a mean positive effect of predators on parasite prevalence, for studies performed in aquatic habitats the mean effect of predators on prevalence was indistinguishable from zero. In combination, these results suggest that parasites in aquatic habitats may not be more sensitive than those in terrestrial habitats to predation pressure. Nonetheless, common mechanisms of predator-parasite interactions may differ substantially between aquatic and terrestrial systems (McCallum et al., 2004; Lopez & Duffy, 2021). For example, predator signs may travel more easily and persist for long periods in water enhancing predator induced behavioural and physiological changes in prey (Lopez & Duffy, 2021). But, this may have been difficult to detect in the existing literature due to the fact that terrestrial and aquatic studies are not otherwise equivalent. For example aquatic studies are much more variable than terrestrial studies, spanning parasite types, life cycle types, and study designs, whereas there were no studies of non-consumptive interactions in terrestrial systems in our entire dataset (Table 2). Therefore we propose that a wider, more mechanistically diverse, study of predator-prey-parasite interactions in terrestrial systems is required to appropriately estimate how the outcomes of these diverse mechanisms actually differ by habitat.

Third, although there may not be a significant difference between parasite taxonomic groups we saw a clear difference between parasites and parasitoids. When controlling for publication bias, predators significantly decreased parasitoids with no overall effect on parasites. Our ability to detect a strong directional effect for parasitoids is perhaps partly due to the uniformity across the studies in the parasitoid analysis, also supported by the more limited heterogeneity in the parasitoid REM. The decrease in parasitoid abundance with increased predation may be due to the fact that consumptive effects of predators on parasitoids rarely include mechanisms that could produce positive effects. Predators rarely act in a 'spreader' role for parasitoids in their prey because the larval life cycle of the parasitoid is typically interrupted by predation (Naselli et al., 2017). Perhaps most non-consumptive effects of predators on parasitoids concern free-living adult life stages, which may avoid areas with predators due to direct intraguild predation of predators on adult parasitoids (Brodeur & Rosenheim, 2000; Heimpel et al., 1997). As a result, it is conceivable that parasitoids would display the stronger response to predator addition than other parasitic organisms.

One of the main limitations of this study, as with all quantitative synthesis, is the selection bias in the field being synthesised. We detected significant publication bias in the literature in multiple directions. Particularly, our analysis of prevalence showed a significant bias towards publication of positive effect sizes (predators increasing prevalence), probably due to the

abundant predator-spreader associated effect sizes. For parasitoids, however, there was significant evidence of publication bias for negative effect sizes. Besides publication bias in effect sizes, we noted important imbalances in study characteristics, particularly the lack of non-environmentally transmitted parasites, the focus on invertebrate and amphibian hosts, and the strong association between habitat type and whether consumptive effects were present. Although many animal parasites are environmentally transmitted, directly transmitted parasites are also common and may behave very differently in response to predation pressure. For example, directly transmitted parasites may be more sensitive to predator induced decreases in prey density because there is no environmental reservoir of parasites. Therefore, we encourage testing whether solely or primarily directly transmitted parasites follow the patterns we find here. Small animals, especially invertebrates, are commonly used to study ecological patterns due to tractability, but cannot represent the wealth of mechanisms and behaviours possible in predator-prey-parasite interactions and testing whether our findings hold true in other taxa is necessary. Aquatic systems are where non-consumptive interactions are commonly studied using caged predators and predator kairomones but this bias in the literature makes it difficult to extrapolate to terrestrial systems. As such, we encourage testing whether non-consumptive effects of predators on parasites in terrestrial systems behave similarly to those in aquatic systems. Based on the existing literature the empirical dissection of consumptive effect mechanisms is largely limited to predator-spreading. In order to truly understand these systems, we need to test whether other specific consumptive mechanisms (Duffy et al., 2019) increase or decrease parasitism in prey. Finally, we note that the severity of disease inflicted by a parasite on its host is likely important to determine the effect of predators on parasite transmission but could not be reasonably extracted from the published literature for many systems, and directed empirical study across varying levels of parasite harm would greatly improve our understanding. For example, parasites that cause substantial harm are predicted to both induce more preferential predation and restrict host behavioural responses to predators (Buss & Hua, 2018; Packer et al., 2003). We hope that our identification of these gaps in the literature provides motivation for future empirical work.

Overall, we found that the healthy herds hypothesis is not broadly supported by the current empirical literature. Instead, as suggested by recent synthetic and theoretical work (Duffy et al., 2019), the average effect of predators on parasites in prey varies significantly according to the presence and type of consumptive interactions and whether the focus is on parasites or parasitoids. Our findings provide the first quantitative synthesis supporting the theoretical consensus (Choisy & Rohani, 2006; Duffy et al., 2019; Hethcote et al., 2004; Holt & Roy, 2007; Roy & Holt, 2008) that predator effects on parasites are context dependent. Our results further suggest that the mechanistic basis of predator-prey interactions strongly influences parasite outcomes and that these effects are predictable.

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AUTHORSHIP

RLR, JMD, and VOE conceived of the study. RLR performed literature search, collected and analysed data, and wrote the first draft of publication. All authors contributed substantially to revisions.

PEER REVIEW

The peer review history for this article is available at https://publons.com/publon/10.1111/ele.13919.

OPEN RESEARCH BADGES

This article has earned Open Data and Open Materials badges. Data and materials are available at: https://doi.org/10.6084/m9.figshare.13363268.v1.

DATA AVAILABILITY STATEMENT

Data and code used in these analyses will be published on figshare upon acceptance of the manuscript and a do will be included in the article. Prior to acceptance data and code can be accessed at this private figshare link: https://figshare.com/s/ae13262817c42e4e82d9

ORCID

Robert L. Richards https://orcid.org/0000-0003-4131-2732
John M. Drake https://orcid.org/0000-0003-4646-1235
Vanessa O. Ezenwa https://orcid.org/0000-0002-8078-1913

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