THE EFFECTS OF PREDATORS ON PARASITES IN THEIR PREY

by

ROBERT LUNDELL RICHARDS

(Under the Direction of Vanessa Ezenwa and John Drake)

ABSTRACT

Organisms navigate a complex set of interspecific interactions, among the most important of these being victimization by predators and parasites. Ecological theory suggests that predators should keep prey populations healthy by reducing parasite burdens. However, empirical studies show that predators often have minimal effects on, or even increase, parasitism in prey. In this dissertation I used a combination of meta-analysis, macro-ecological scale analysis, experimental field manipulation, and predictive spatial modeling to (i) rigorously assess the generality of predictions of negative effects of predators on parasites in prey and (ii) evaluate the importance of heterogeneities in natural systems to the strength and direction of these effects. I found, across these studies, that the effects of predators on parasites in their prey were varied, and frequently positive rather than negative. I also found that this variation in the strength and direction of effects of predators on parasites in prey was driven by a variety of factors, including parasite traits, host/prey species identity, and predator interaction types. In particular, I find strong evidence of the importance of both non-consumptive interactions and the sharing of parasites between predators and prey contributing to the unexpected positive effects of predators on parasites in prey, while negative effects of predators on parasites tend to be

associated with consumptive interactions between predators and prey. More broadly, while I identify some useful general patterns, my dissertation provides strong support for the importance of the specifics of a system to predicting the outcome of predator-prey-parasite interactions.

INDEX WORDS: disease ecology, wildlife disease, trophic interaction, predator-prey interactions

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CHAPTER 1

INTRODUCTION AND LITERATURE REVIEW

diseases desperate grown
By desperate appliance are relieved,
Or not at all.

Claudius
Act IV, Scene III
The Tragedy of Hamlet, Prince of Denmark
William Shakespeare

Understanding species interactions, and the results of disrupting them, is a core motivation of population and community ecology. Among the most important of these interactions is victimization by natural enemies. Both predators (Krebs et al. 1995, 2018) and parasites (Hudson et al. 1992b, Tompkins and Begon 1999) can affect the population demography and dynamics of the species they attack. However, organisms are typically victims of many, varied, natural enemies. Competition between predators of a single prey population (Holt and Lawton 1994, Holt and Polis 1997, Tallian et al. 2017) and between parasites within a single host organism (Pedersen and Fenton 2007, Jolles et al. 2008, Ezenwa and Jolles 2011) can have remarkable consequences for both types of organisms. But predators and parasites of a single victim population also interact in a variety of potentially important ways.

The healthy herds hypothesis suggests that predators keep herds healthy by decreasing parasitism in their prey, both by culling infectious individuals and decreasing the density of host/prey populations (Packer et al. 2003). For example, declines in lobster

populations result in larger and more frequent epidemics in their sea-urchin prey (Lafferty 2004). This prediction has worrying implications for anthropogenic predator loss (Sih et al. 1985, Estes et al. 2011) and deliberate predator control (Packer et al. 2009), as increased parasitism in prey populations can both negatively impact prey populations and increase the possibility of spillover of parasites to other organisms, including humans (Ostfeld and Holt 2004, Han et al. 2015, 2016). Thus, given the frequency of predator loss, and the ubiquity of parasitism in prey populations, it is critical to gain a more detailed and nuanced understanding of the effects that predators can have on parasitism in their prey.

The observed effects of predators on parasites in prey frequently diverge from the basic expectation that predators should decrease parasitism. For example, despite the fact that wolves appear to prey preferentially on moose heavily infected with tapeworms, moose populations under high levels of predation suffer more than triple the mean parasite loads of populations under low predation (Joly and Messier 2004). Researchers have theoretically explored potential drivers for some of this heterogeneity (Holt and Roy 2007, Roy and Holt 2008), and tested the underlying prediction across a wide variety of predator-prey-parasite systems (Hudson et al. 1992a, Joly and Messier 2004, Duffy et al. 2011, 2019, Stephenson et al. 2015, Koprivnikar and Urichuk 2017). But only recently have efforts turned to the identification of generalizable patterns with which to predict the strength and direction of effects of predators on parasites in their prey (Duffy et al. 2019). This dissertation will use a combination of field manipulation and computational analysis to: (i) rigorously assess the generality of the predictions of the healthy herds hypothesis for the effect of predators on parasites in prey and (ii) evaluate the importance of

heterogeneities in natural systems in shaping the outcome of predator-prey-parasite interactions. In particular, I explore the importance of the type of predator-prey interaction (Chapter 2), parasite traits (Chapter 2, Chapter 3), seasonality (Chapter 4), prey species identity (Chapter 4, Chapter 5), and prey space use (Chapter 5).

In Chapter 2, I used a meta-analysis of prior empirical studies to estimate the average effect of predation on parasitism in prey and to test the influence of predator interaction type and parasite group on the strength and direction of this effect. While consumptive interactions between predators and prey are hypothesized to decrease parasitism in prey populations, there are a variety of circumstances under which direct killing of prey organisms by predators can actually increase population-level parasitism (Duffy et al. 2019). For example, some “predator-spreaders" facilitate transmission by consuming prey (e.g. chaoborus larvae shred infected *Daphnia* spreading infectious fungal spores (Cáceres et al. 2009)). Predators also influence the populations of their prey via non-consumptive effects (Schmitz et al. 2004, Creel et al. 2007, Peckarsky et al. 2008). Predators can affect parasitism in prey by modifying prey behavior in ways that increase or decrease parasite transmission between hosts (Brown et al. 1988, Ezenwa 2004, Szuroczki and Richardson 2012, Patterson and Ruckstuhl 2013, Creel et al. 2014) or by altering prey physiology in ways that increase individual susceptibility to parasitism (Navarro et al. 2004, Buss and Hua 2018). For example, Trinidadian guppies shoal in larger groups when under high predation and as a result those same guppies experience higher burdens of the ectoparasite *Gyrodactylus* (Stephenson et al. 2015). I, also, expected that differences among parasites themselves would alter the effect of predators. Particularly, I predicted that that predators would have more negative effects on

macroparasites and parasitoids than on microparasites, because the former groups are highly aggregated in certain hosts and spatial locations (Hassell 1982, Chesson and Murdoch 1986, Shaw and Dobson 1995) allowing small amounts of selective predation to eliminate their populations. This chapter aimed both to extract consensus from a messy body of empirical work and to begin to identify the drivers of heterogeneity in predator-prey-parasite interactions.

Parasite traits are, however, far more complex than the comparison of macroparasites, parasitoids, and microparasites in our meta-analysis would suggest and we expected finer-scale distinctions to influence predator-prey-parasite interactions. First, we expected parasite transmission mode to significantly influence the effect of predators on parasites in prey. For example, transmission of vector-borne parasites is typically considered to be independent of the host population size (frequency-dependent) because of the way that vectors actively search for hosts (Thrall et al. 1993). Therefore, vector-borne parasites are less likely to respond to changes in host/prey density due to predation pressure. Alternatively, parasites transmitted through close contact are likely to be highly responsive to predator induced changes in host/prey behavior that can substantially alter contact rates (Creel et al. 2014, Stephenson et al. 2015). Second, we expected that parasites which can infect both the predator and prey should respond differently to predation than those which are not shared, with shared parasites being more likely to increase with predation pressure as predators are also contributing to transmission. In Chapter 3, I used a global-scale macroecological analysis of parasites of ungulates (orders: Artiodactyla and Perissodactyla), recorded in the Global Mammal Parasite

database, to test the importance of parasite traits to the effect of predation pressure on local parasite prevalence and host species level parasite diversity.

Chapter 4 addresses three common heterogeneities in natural systems that are rarely examined in the existing literature: seasonality, disturbance, and prey species identity. Despite the fact that the importance of each of these factors is well studied in the predator-prey literature, predator-prey-parasite studies typically ignore them. This creates problems for the generalizability of empirical findings if these factors prove similarly important to predator-prey-parasite interactions. For example, prey population size and behavior often vary seasonally and after a major disturbance, such as fire, due to changes in food availability, reproduction, and torpor or hibernation (Merritt et al. 2001, Morris et al. 2011c, 2011a, 2011b). Variation such as this can alter the relative availability of prey to different predator species and the behavioral response of prey to predation pressure, directly affecting parasite distributions in prey populations. These, responses both to seasonality and disturbance and to predation pressure typically vary starkly between even closely related host/prey species (Morris et al. 2011c, 2011a, 2011b). As a result we might expect that any understanding based on observations of one host/prey species may generalize poorly to other species. I used a large-scale exclusion experiment (Conner et al. 2011, Morris et al. 2011c) to test how the effect of mammalian mesopredators on the gastro-intestinal parasites of rodents differed by season, fire disturbance, and prey species.

In Chapter 5, I investigate the way that predators can change the landscape of community-level disease transmission by influencing prey space-use. Most studies of predator-prey-parasite interactions, including those in chapters 2-4, operate at the

population level. But many parasites are transmitted among a community of interacting species (Dallas et al. 2017), and predator induced changes in the way that hosts/prey use space can have large effects on parasitism (Stephenson et al. 2015). Therefore, I expected predator-prey-parasite interactions to influence spatial disease transmission across a host community. In particular, within host species ranges, we expect areas with high levels of predator use to have lower transmission potential, because of behavioral avoidance by prey/hosts (Laundré et al. 2001, Ripple and Beschta 2012). In this way, predation might influence community-level parasite transmission at a much finer grain than previously considered. In this chapter, we report on a study of transmission and space use heterogeneity in a community of six sympatric ungulate species at the National Bison Range (Montana, USA), in which we tested the effect of carnivore space-use on community-level spatial transmission potential.

Collectively, this body of work aimed to both assess the generality of the healthy herds hypothesis and identify important heterogeneities in natural systems that drive the strength and direction of the effects of predators on parasites in their prey. My research spans a wide range of taxa and leverages a diverse array of methodological approaches to accomplish these goals. As anthropogenic forces continue to alter predator densities and disrupt their ranges (Prugh et al. 2009, Estes et al. 2011), while simultaneously drastically altering the patterns of parasite transmission in wildlife populations (Gottdenker et al. 2014, Becker et al. 2015), it is ever more important to understand the effects of predators on parasites in their prey.

CHAPTER 2

DO PREDATORS KEEP PREY HEALTHY OR MAKE THEM SICKER? A META-ANALYSIS[1]

[1] Richards R.L, Drake J.M., and V.O. Ezenwa. Submitted to *Ecology Letters*

ABSTRACT

Ecological theory suggests that predators should keep prey populations healthy by reducing parasite burdens. However, empirical studies show that predators often have minimal effects on, or even increase, parasitism in prey. To quantify the overall magnitude and direction of the effect of predation on parasitism in prey, we conducted a meta-analysis of 50 empirical studies. We also examined how key attributes of these studies, including parasite type, study design, and predator interaction type (consumptive vs. non-consumptive) 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 predator interaction type, 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, while context dependent, are predictable.

INTRODUCTION

Organisms navigate a complex set of interspecific interactions, among the most important of these being victimization by natural enemies. Both predators (Krebs et al. 1995, 2018) and parasites (Hudson et al. 1992b, Tompkins and 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 and Lawton 1994, Holt and Polis 1997, Tallian et al. 2017) and between parasites within a single host organism (Pedersen and Fenton 2007, Jolles et

al. 2008, Ezenwa and Jolles 2011) 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 in a variety of potentially important ways. Parasites may weaken their hosts, making them easier to catch and consume (Hudson et al. 1992a, Moore 2002), while the killing and consuming of prey by predators also kills parasites (Hatcher et al. 2006, Borer et al. 2007), except when the predator itself becomes the next host (Lafferty 1999, Kuris 2003, Logiudice 2003). Therefore, like other natural enemy interactions, interactions between predators and parasites are likely critical to understanding the dynamics of natural populations.

Ecologists have long recognized the importance of predator-prey-parasite interactions (Hudson et al. 1992a). 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 parasitism in their prey. This Healthy Herds Hypothesis (HHH) phenomenon might be produced by multiple mechanisms. First, predators directly, and often preferentially, kill infected individuals, decreasing the number of infected individuals in the population. Second, predators often reduce prey population sizes, which can decrease the spread of parasites with density dependent transmission. Empirical studies have tested the underlying predictions of the HHH in a variety of systems, but results are conflicting. Some studies show a strong negative effect of predators on parasites, while 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), while sunfish (*Lepomis gibbosus*) predators introduced into tanks with infected

tadpoles (*Lithobates spp.*), increased trematode cercarial load in tadpole prey (Szuroczki and Richardson 2012). Interestingly, these empirical studies differ along multiple axes, including the transmission traits of the parasite (Holt and Roy 2007, Roy and Holt 2008) and the type of predator or predatory interaction manipulated (Cáceres et al. 2009, Strauss et al. 2016, Duffy et al. 2019), which may help explain the variation in outcomes.

In this study, we used a meta-analytical approach to quantify the overall magnitude and direction of the effect of predation on parasitism, providing a synthesis of the empirical work on this topic. We also tested the prediction that differences among studies along two key axes, (i) parasite type, and (ii) type of predatory interaction, explain variation in observed parasite responses. Specifically, we predicted that effects of predators on macroparasites and parasitoids would be more negative than effects on microparasites, because macroparasites and parasitoids tend to be highly aggregated among hosts and spatial locations (Hassell 1982, Chesson and Murdoch 1986, Shaw and Dobson 1995) allowing small amounts of selective predation pressure to nearly eliminate their populations. Parasitoids in particular have free-living adult stages which may fall prey to or avoid predators of their hosts (Heimpel et al. 1997, Brodeur and Rosenheim 2000). We also predicted that consumptive predatory interactions would have more negative effects on parasites than non-consumptive interactions, except when consumptive effects facilitate parasite spread. In this case, consumptive interactions should actually increase parasitism. The HHH predicts that, on average, consumptive interactions decrease parasitism as infected individuals are removed from populations (Packer et al. 2003). However, this average effect of consumption on parasites should not apply in all circumstances (Duffy et al. 2019). In particular, “predator-spreaders” may

facilitate the spread of parasites from their prey items by dispersing infectious agents more widely (e.g. Cáceres et al. 2009). On the other hand, non-consumptive interactions can alter prey movement and space use behavior (Brown et al. 1988, Spieler 2003, Jones and Dornhaus 2011, Creel et al. 2014) in ways that can either increase or decrease parasite transmission (Ezenwa 2004, Patterson and Ruckstuhl 2013), meaning that the effects of non-consumptive interactions on parasites should be less consistently negative than those of consumptive interactions. While multiple syntheses of predator-prey-parasite interactions have been published over the past 20 years (Ostfeld and Holt 2004, Hatcher et al. 2006, Duffy et al. 2019), these studies take a qualitative approach. Here we use an approach that explicitly quantifies the typical effect of predators on parasites in their prey and the most important drivers of variation in this response. We ask: (i) what is the average overall effect of predators on parasites in their prey and (ii) does this effect vary by parasite or interaction type? We expect to find a negative overall effect of predation on parasitism, but this effect should be more negative for macroparasites and parasitoids than microparasites and for interactions involving consumptive than non-consumptive interactions. We also expect that consumptive interactions including identified "predator-spreaders" should have more positive effects than those with non-spreaders.

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 (i) involved an animal host/prey population, a predator population that kills and consumes the host/prey, and a parasite that infects the host/prey; (ii) observed multiple levels of predation pressure, and (iii) 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, 50 of which were confirmed following full-text screening (Figure 2.4).

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 (macroparasite, microparasite, or parasitoid), study design (observational or experimental), predator interaction type (all or non-consumptive), and predator spreader identity (predator spreader or not) for inclusion in mixed effects models (MEMs) testing the effect of these moderators on effect sizes. The majority of studies (45 of 50) were composed of a binary comparison of a parasite response across two levels of predation. Most studies were analyzed 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 standardized mean difference (Hedges g) using the *escalc*

function in the R package metaphor (Viechtbauer 2010). A small minority of studies (5 of 50) 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 193 effect sizes from 48 studies.

Not all effect sizes contain the same type of information because of differences in the biology of parasites 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: (i) the number or proportion of hosts infected (quantified as prevalence, number or density of infected individuals, or disease induced mortality rate; n = 89 effect sizes from 22 different studies, Table 1, Table 2) and (ii) 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 a small amount of selective predation on a population with highly aggregated parasites may have a large effect on mean intensity but a small effect on prevalence), we analyzed these responses separately. Another distinction we made was to separate parasites from parasitoids. 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. For this reason, we analyzed parasitoids (n = 43 effect sizes from 11 different studies) separately from parasites.

Statistical Analysis

Main Effect and Publication Bias

We analyzed effect size data for each of the three categories of our data (prevalence, intensity, parasitoid) according to the following scheme. First, we fit a random effects model (REM) to estimate the overall effect of predators on parasites in prey. 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 and Thompson 2002). We also used these models to diagnose publication bias in the data by visualizing 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 and Tweedie 2000) to determine whether introduction of studies to balance the diagnosed bias would alter the main effect.

Effects of Moderators

Given the level of 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 moderators: predator effect type manipulated (non-consumptive vs. all interaction types), predator-spreader identity (identified as predator-spreader or not), and parasite type (macro vs. micro) and all two-way interactions. 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 while 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 limitations in experimental design. Therefore, we draw the distinction between studies which manipulate only non-consumptive interactions and those which include consumptive interactions (all interaction types). 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. When reporting the results of univariate models for the most important variables, we provided the direction of the effect of the moderator and the results of a test for residual variation. Because parasitoid studies were uniformly experimental and consumptive, we did not fit MEMs with moderators to these data.

Assessing Phylogenetic Signal

Because of shared evolutionary history, closely related host species may have similar effect sizes. We use Pagel's lambda (Pagel 1999) to estimate phylogenetic signal in the distribution of effect sizes across taxa. When phylogenetic non-independence was detected, we included a phylogenetically structured random effect of species in our final models. For both steps, we obtained a phylogeny of relevant prey/host species from the Open Tree of Life using the *ROTL* package (Hinchliff et al. 2015, Michonneau et al.

2016); then we used the *ape* package to prune the tree to our host species, to resolve polytomies, and to generate branch lengths (Paradis and Schliep 2018). For host species with multiple effect sizes, we calculated the average effect size for each species weighted by the sample size of each component study. We then used the *pgls* function of the *caper* package to estimate Pagel's lambda by maximum likelihood for each of our datasets (Orme et al. 2018). We statistically tested the difference between this estimate and two alternative possibilities: phylogenetic independence (lambda = 0) and phylogenetic dependence as characterized by Brownian motion models (lambda = 1). We failed to detect evidence of phylogenetic dependence in any dataset using this method.

RESULTS

Study Patterns

We identified substantial gaps in the literature reviewed for certain combinations of moderators (Table 1, Table 2). In particular, no observational studies considered non-consumptive effects, and no studies that measured parasitism by intensity metrics or that studied macroparasites manipulated the effect of predator spreaders. Both micro- and macroparasites are represented in studies measuring both prevalence and intensity but macroparasites were more common in intensity studies (n = 41/61) and microparasites more common in prevalence studies (n = 66/89). Both macro- and microparasites had the effects of both interaction types studied in fairly even proportions, but non-consumptive effects were more represented in macroparasite prevalence studies while they were more common in microparasite intensity studies.

Parasite Prevalence

A REM of prevalence effect sizes showed an overall effect that was not significantly different from zero ($z = 1.818$, $p = 0.069$; Figure 2.1a), with a large amount of heterogeneity between studies ($I^2 = 91.00\%$), and significant publication bias ($\boldsymbol{\tau} = 0.200$, $p = 0.005$). The trim-and-fill method estimated 18 missing negative studies, but inclusion of these studies did not change the outcome, with the modified REM still showing no evidence of an effect ($z = -0.795$, $p = 0.427$). In our analysis of moderators, predator spreader identity was included in nearly all MEMs with non-zero weights (Importance = 0.993; Figure 2.1a, Table 3). Interaction type was also important (Importance = 0.771), but other main effects were less so (parasite type importance = 0.620; study design importance = 0.547). The most important interaction term was between interaction type and predator spreader identity (Importance = 0.665). In univariate analyses, only predator spreader identity significantly affected mean effect size ($QM_1 = 11.278$, $p = 0.0008$), despite significant residual heterogeneity ($QE_{87} = 630.561$, $p < 0.001$). Predator spreaders had more positive effects than non-spreader predators (Figure 2.3).

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 2.1b), with a large amount of true heterogeneity between studies ($I^2 = 75.93\%$), and no evidence of publication bias ($\boldsymbol{\tau} = -0.106$, $p = 0.231$). The single most important moderator was interaction type (Importance = 0.775; Figure 2.2b, 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). Non-consumptive interactions had more positive effects than all interactions (Figure 2.3).

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 2.1c), with a smaller amount of heterogeneity between studies as compared to the analyses of parasite responses (I^2 = 35.47%). While there was evidence of significant publication bias ($\boldsymbol{\tau}$ = -0.227, p = 0.032), 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).

DISCUSSION

The healthy herds hypothesis (HHH) (Packer et al. 2003) predicts that predators should have negative effects on parasites in their prey, but empirical studies testing this hypothesis have reported a variety of different effects. We hypothesized that this variation is a result of nuances in predator-prey-parasite interactions, including transmission strategy of the parasite studied and the type of predator interaction manipulated. Specifically, we hypothesized that the negative effect predicted by the HHH would be larger for macroparasites and parasitoids than for microparasites and would only hold when consumptive interactions are manipulated and when those predators are not "predator spreaders". Using a meta-analytic approach that accounted for potential sources of variation in observed predator-prey-parasite interaction outcomes, we found that the main effect of predators on parasites in prey differed between parasites and

parasitoids but not between conventional macro- and microparasites, with a net negative effect only present for parasitoids. Additionally, we found that interaction type (all vs. non-consumptive), and its subset of predator spreader interactions, were most important in predicting the effect of predators on parasites in prey. These findings provide clear evidence that the HHH prediction is not universal. The degree to which it holds in a given system is both parasite- and context-dependent, but also predictable with limited information.

We observed significant heterogeneity across studies of the HHH resulting from substantial variation 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 of those interactions are also very important. In studies that measured intensity variables, the effect size significantly differed between interactions involving consumptive and non-consumptive interactions, with non-consumptive interactions having generally more positive effects. 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 sorts of interactions including non-consumptive, suggesting that this result may, in fact, be conservative. Our result for studies measuring prevalence variables contradicts this finding as consumptive and non-consumptive interactions were estimated to be nearly identical on average. We suggest that the difference between these two response variables is an artifact of the significant residual

heterogeneity even in our best fit models. Most of this variation is likely hidden in unexplored mechanisms within these studies. Duffy et al. (2019) outlined 7 independent mechanisms whereby consumption can directly or indirectly impact 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 (Duffy et al. 2019). 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 their studies. As expected, predator-spreader identity was highly important for predicting the parasite outcome in the prevalence dataset, generally increasing parasite prevalence. The difference in the number of predator-spreader effect sizes between prevalence (n = 25) and intensity (n = 0) responses explains why we saw this effect emerge in the prevalence but not intensity dataset. Ultimately, the lack of universal support for the HHH 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, unlike predator interaction type, we failed to detect an effect of parasite type in our analysis. We hypothesized that differences in the aggregation patterns of micro- and macroparasites would result in macroparasites having a stronger and more negative response to predator pressure than microparasites but found no evidence for a difference between parasite types in either intensity or prevalence effect sizes and this

variable was generally of less importance for explaining variation. This lack of an effect may be due to a number of factors. While one might expect random predation, or predation on infected individuals, to decrease parasitism 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 (Nilsson and Brönmark 2000, King 2002) that selectively prey on smaller and younger individuals may cause population demographics to shift towards larger, older and more heavily infected hosts (Dobson 1989, Nilsson and Brönmark 2000, Byers et al. 2015, Duffy et al. 2019). Alternatively, our assumption that high aggregation among macroparasites makes them more vulnerable to predation may be countered by the existence of significant aggregation in microparasite systems as well (Lord et al. 1999, Grogan et al. 2016).

Third, while there may not be a significant difference between micro- and macroparasites we saw a clear difference between parasites and parasitoids. Even when controlling for publication bias, predators had a significant negative effect on parasitoids as compared to the lack of any 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 negative direction of the effect 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 (Heimpel et al. 1997, Brodeur and Rosenheim 2000). As a result, it is conceivable that parasitoids would display a stronger negative 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 synthesized. 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, probably due to the abundance of predator-spreader associated effect sizes. In the case of parasitoids, however, there was significant evidence of publication bias for negative effect sizes. While correction for these biases did not influence qualitative conclusions, their presence does suggest the need for additional attention to the types of results published. Besides publication bias in effect sizes, we noted a number of important imbalances in study characteristics, particularly the lack of observational studies that inspected non-consumptive effects. We also found that studies which identified predators as predator spreaders were largely limited to studies of microparasite prevalence. This finding suggests that the empirical dissection of consumptive effect mechanisms is not only limited to cases that are easy to characterize (like predator-spreaders), but also limited in taxonomic coverage. Given these gaps in the literature, we suggest the following priorities for future work: (i) examining the effect of non-consumptive predator interactions on parasites in non-manipulative field observations and (ii) further dissecting the effect of predator-spreaders and other types of consumptive interactions on both micro- and macroparasites.

Overall, we found that the healthy herds hypothesis was not broadly supported by the current literature. Instead, the average effect of predators on parasites in prey varies significantly according to the type of interaction being studied and whether the focus is on parasites or parasitoids. Our findings support the general conceptual consensus (Hethcote et al. 2004, Choisy and Rohani 2006, Holt and Roy 2007, Roy and Holt 2008, Duffy et al. 2019) that predator effects on parasites are context dependent, and provides the first quantitative analysis supporting this view. Our results further suggest that the mechanistic basis of predator-prey interactions strongly influences parasite outcomes and that these effects are predictable.

Acknowledgements

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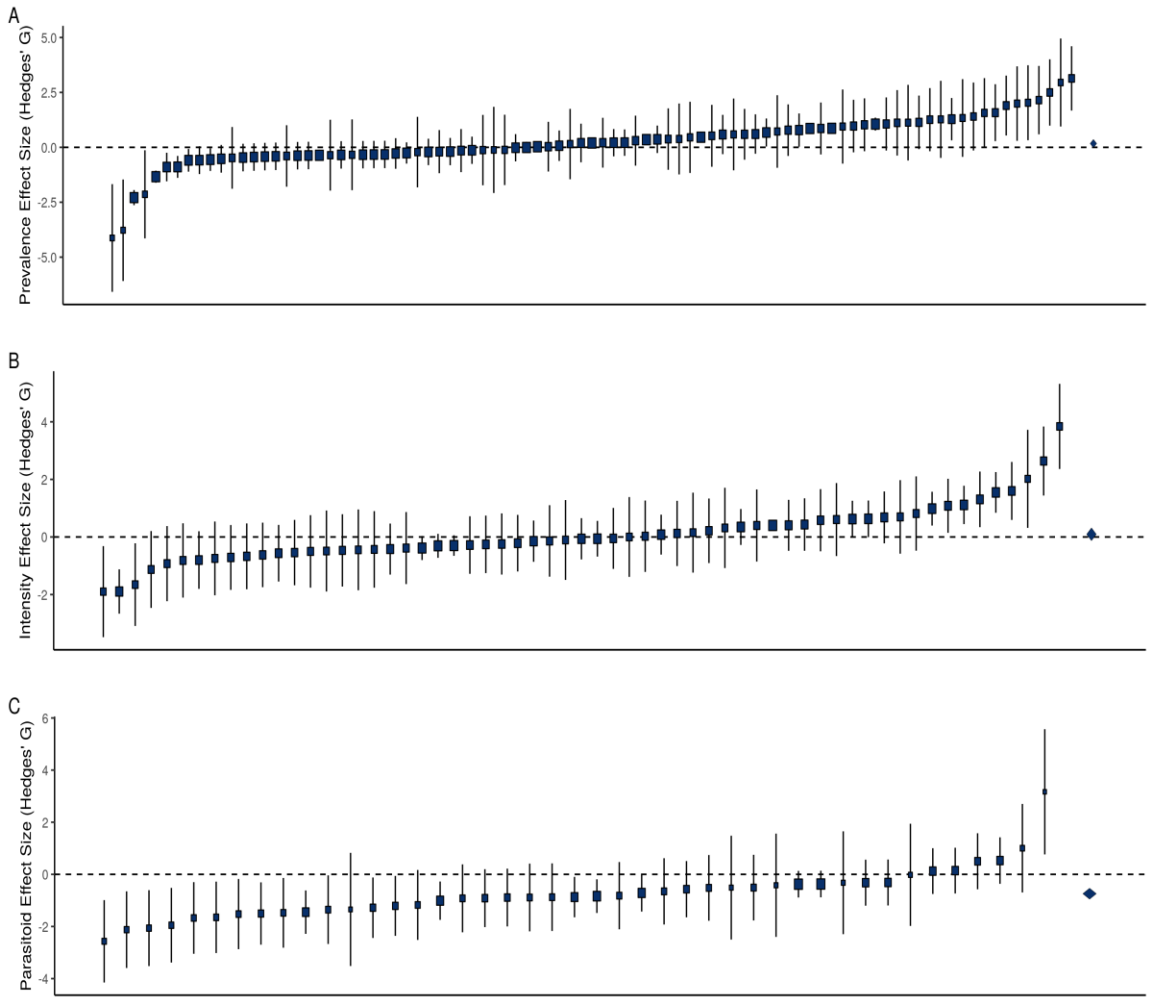

Figure 2.1. Range and grand means from random-effects meta-analysis models (REMs) for the effect of predators on parasites in prey stratified by prevalence (a) intensity (b) and parasitoid (c) data. Lines show 95% confidence intervals for effect sizes and REMs (uncorrected for publication bias). The dashed line represents no relationship between condition and infection.

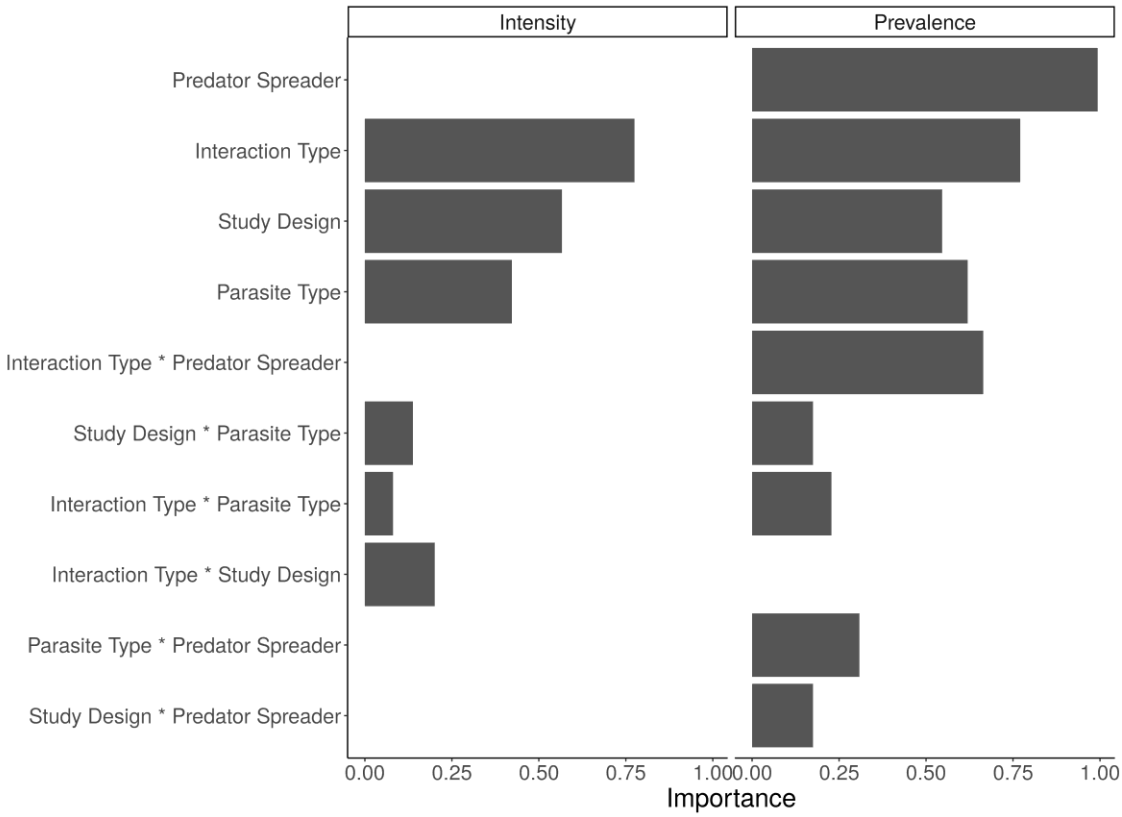

Figure 2.2. AICc weight based importance of moderators for MEMs of intensity and prevalence effect sizes.

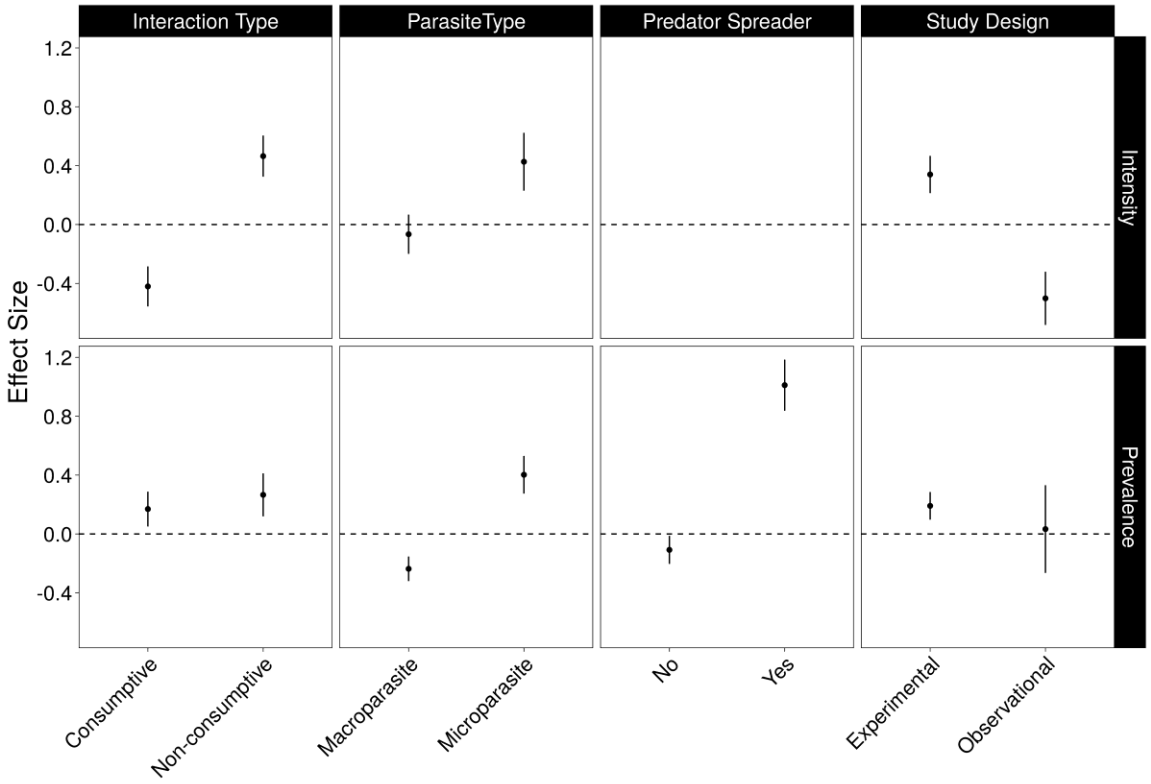

Figure 2.3. Modeled univariate relationships (means and 95% confidence intervals) for the four most important moderators of effect size across all intensity data (top row), and prevalence data (bottom row). Results from mixed-effects models are sorted by study traits. Interaction type: consumptive or non-consumptive. Parasite type: macroparasite or microparasite. Predator spreader identity: identified as a predator spreader or not. The dashed line represents no relationship between condition and infection.

Figure 2.4 PRISMA diagram of systematic literature search process.

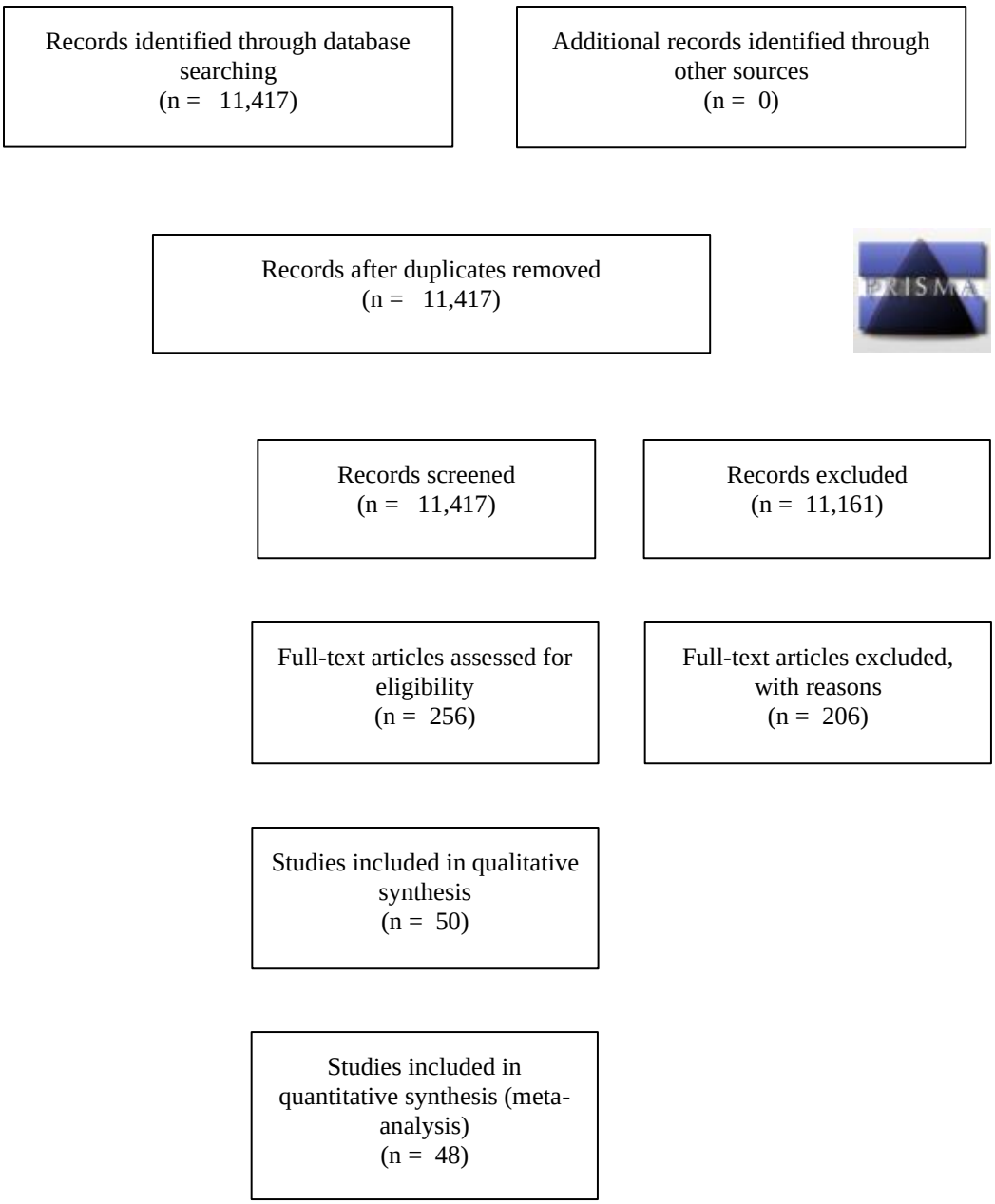

Table 2.1. Enumeration of effect sizes categorized in nested subsets of key moderators for prevalence effect sizes.

Study Design												
Observational (12)						Experimental (77)					
Parasite Type						**Parasite Type**					
Macroparasites (4)			Microparasites (8)			Macroparasites (19)			Microparasites (58)		
Interaction Type			**Interaction Type**			**Interaction Type**			**Interaction Type**		
All (4)		Non-Consumpt. (0)	All (8)		Non-Consumpt. (0)	All (18)		Non-Consumpt. (1)	All (40)		Non-Consumpt. (18)
Predator Spreader?			**Predator Spreader?**			**Predator Spreader?**			**Predator Spreader?**		
Yes 0	No 4		Yes 2	No 6		Yes 0	No 18		Yes 23	No 17	

Table 2.2. Enumeration of effect sizes categorized in nested subsets of key moderators for intensity effect sizes.

Study Design												
Observational (18)						Experimental (43)					
Parasite Type						**Parasite Type**					
Macroparasites (18)			Microparasites (0)			Macroparasites (23)			Microparasites (20)		
Interaction Type			**Interaction Type**			**Interaction Type**			**Interaction Type**		
All (18)		Non-Consumpt. (0)	All (0)		Non-Consumpt. (0)	All (6)		Non-Consumpt. (17)	All (1)		Non-Consumpt. (19)
Predator Spreader?			**Predator Spreader?**			**Predator Spreader?**			**Predator Spreader?**		
Yes 0	No 18		Yes 0	No 0		Yes 0	No 6		Yes 0	No 1	

Table 2.3. Ranking of mixed-effects models (MEMs) predicting effect size for the effect of predators on parasites in the prevalence and intensity data. Models are ranked by ΔAICc with the number of parameters (*k*), test statistic for the omnibus test of model coefficients (Q_M), estimated variance components (σ_i), and Akaike weights (w_i). Only MEMs with ΔAICc ≤ 2 are shown.

MEMs fit to prevalence data	df	σ_{Study}	QM	QE	ΔAICc	weight
G ~ Interaction Type + Predator Spreader + Interaction Type * Predator Spreader	5	0.47	20.554 (< 0.001)	586.230 (< 0.001)	0	0.108
G ~ Interaction Type + Study Design + Predator Spreader + Interaction Type * Predator Spreader	6	0.452	22.656 (< 0.001)	546.857 (< 0.001)	0.84	0.071
G ~ Predator Spreader	3	0.556	12.824 (< 0.001)	630.5613 (< 0.001)	0.86	0.07
G ~ Interaction Type + Study Design + Predator Spreader + Interaction Type * Predator Spreader + Study Design * Predator Spreader	7	0.49	22.930 (< 0.001)	545.762 (< 0.001)	1.62	0.048
G ~ Interaction Type + Parasite Type + Predator Spreader + Interaction Type * Parasite Type + Interaction Type * Predator Spreader	7	0.533	21.69 (< 0.001)	543.791 (<0.001)	1.65	0.047
G ~ Interaction Type + Parasite Type + Predator Spreader + Interaction Type * Parasite Type + Interaction Type * Predator Spreader + Parasite Type * Predator Spreader	7	0.533	21.69 (< 0.001)	543.791 (<0.001)	1.65	0.047
MEMs fit to intensity data	df	σ_{Study}	QM	QE	ΔAICc	weight
G ~ Interaction Type	3	0.5049	6.5098 (0.011)	182.050 (< 0.001)	0	0.243
G ~ Interaction Type + Parasite Type	4	0.494	7.175 (0.028)	180.870 (<0.001)	1.83	0.097
G ~ Interaction Type + Study Design	4	0.5126	6.831 (0.033)	176.171 (<0.001)	1.84	0.097
G ~ Interaction Type + Study Design + Interaction Type * Study Design	4	0.5126	6.831 (0.033)	176.171 (<0.001)	1.84	0.097
G ~ Study Design	3	0.565	3.905 (0.048)	197.867 (<0.001)	1.87	0.095

CHAPTER 3

PARASITE TRAITS AND THE MACROECOLOGY OF PREDATOR-PREY-PARASITE INTERACTIONS[2]

[2] Richards R.L., Drake J.M., V.O. Ezenwa. To be submitted to *Proceedings of the Royal Society B.*

ABSTRACT

Predators are predicted to impact parasitism in their prey but the strength and direction of this effect varies across studies. While much attention has been given to the effect of variation in the predator-prey interaction on predator effects on parasites in prey, heterogeneity in parasite traits might be important, too. For example, parasites transmitted by close contact may be more susceptible to predator-induced behavioral changes in prey than those transmitted by vectors. Here we combine a global database of parasitism rates in ungulates with independently sourced measures of local predation pressure to quantify the role of parasite traits in driving the effect of predation pressure on population-level parasite prevalence and species-level parasite richness in hosts. In particular, we ask whether the effect of predation pressure on parasite prevalence varies across: (i) parasite taxonomic identity, (ii) parasite transmission mode, and (iii) whether the parasite is shared between carnivores (predators) and ungulates (prey). We also ask whether there is an effect of predation pressure on parasite richness and whether this pattern varies according to parasite sharing. We found that parasite taxon, transmission mode, and sharing all influenced the effect of predation pressure on parasite prevalence in prey. In particular, viruses and helminths were most responsive to predation pressure, parasites with intermediate hosts significantly differed in their response to predation from those without intermediate hosts, and the patterns of these interactions differed between shared and unshared parasites. However, we found no evidence for effects of predation on parasite richness in prey populations. These findings support the hypothesis that variation in parasite traits is important to the outcome of predator-prey-parasite interactions.

INTRODUCTION

Predators influence not just their prey but entire communities of interconnected organisms, including parasites (Estes et al. 2011, Thieltges et al. 2013). Theory suggests that predators can limit parasitism in prey both by culling infected individuals from the population and reducing prey population densities (Packer et al. 2003). However, predators can also increase parasitism in prey by modifying prey behavior in ways that increase parasite transmission between hosts (Szuroczki and Richardson 2012), or by altering prey physiology in ways that increase individual susceptibility to parasitism (Navarro et al. 2004, Buss and Hua 2018). Predators and prey also frequently share parasites (Johnson et al. 2010, Thieltges et al. 2013, Stephens et al. 2019), implying that contact between them might be a cause of transmission. In some cases, predation is even required for the parasite to complete its life-cycle (Lafferty 1999, Kuris 2003). These diverse mechanisms result in a range of outcomes of predator-prey-parasite interactions that can vary across contexts (Duffy et al. 2019). For example, predators which have primarily consumptive effects on their prey tend to decrease parasitism while those with more non-consumptive, behavioral, effects tend to increase it (Chapter 2). There has been extensive study of the way variation in predator-prey interactions can produce differences in parasite outcomes (Hatcher et al. 2006, Duffy et al. 2019), but it is unclear whether variation in parasite traits is similarly important.

A number of parasite traits likely influence predator effects on parasitism in prey. In particular, parasites with certain transmission modes might be more or less susceptible to the effects of predation. For example, transmission of vector-borne parasites is

typically considered to be independent of the host population size (frequency-dependent) because of the way that vectors actively search for hosts (Thrall et al. 1993). Therefore, we hypothesized that vector-borne parasites would be less likely to respond to changes in host/prey density or space-use behavior due to predation pressure. Alternatively, parasites transmitted through close contact are likely to be highly responsive to predator-induced changes in host/prey behavior as these behavioral changes can substantially alter contact rates (Creel et al. 2014, Stephenson et al. 2015). Parasites also vary substantially in their host-range with some parasites being shared by prey and their predators and others being prey-specific (Dallas et al. 2017, Stephens et al. 2019). Shared parasites are expected to have a more positive response to predator pressure as predators contribute to their transmission to prey organisms in addition to other predator effects. Parasite taxonomic groups may also differ in fundamental ways that can alter the effect of predators. Most notably, macroparasites, such as helminths and arthropods, which are typically highly aggregated in their hosts (Shaw and Dobson 1995), are expected to be more susceptible to consumptive effects of predation because predators can cull much of the parasite population by removing a few heavily infected individuals (Packer et al. 2003). Given these distinct attributes of parasites, we suggest that the effect of predation on parasitism will vary according to parasite transmission mode, parasite taxa, and whether the parasite is shared between predators and prey.

The effects of predation on parasite abundance and prevalence are well studied (Hatcher et al. 2006, Duffy et al. 2019, Chapter 2), but few studies have tested for the effects of predation on parasite diversity in prey. Based on broader work on the relationship between parasite prevalence and richness we make the following hypotheses.

First, broadly, interactions that decrease average parasite prevalence/abundance, such as the culling of infected hosts or the depression of prey/host density, are expected to also decrease richness as low prevalence, rare, parasites are lost (Altizer et al. 2007, Farrell et al. 2015). Likewise, interactions that increase average parasite prevalence/abundance, such as changes in behavior that increase contact rates between hosts/prey in high predation environments compared with low predation environments, should generally increase parasite richness as parasites with low transmission rates are better able to persist (Anderson and May 1986, 1992). However, the strength of this correlation is predicted to depend on the number of rare parasites and their prevalence relative to common parasites. We suggest that if there are many rare parasites then parasite species richness will change more rapidly with respect to predation pressure than mean parasite prevalence but with few rare species the inverse should be true. For free-living species, community-level metrics typically change more slowly than population level metrics in response to disturbance (Supp and Ernest 2014), suggesting that shifts in parasite richness may lag behind shifts in prevalence. Parasite sharing between predators and prey could also result in increased predator pressure increasing richness of parasitism in prey more rapidly than average prevalence. For example, a larger local predator species richness is expected to expose prey organisms to more shared parasites compared with areas with fewer predator species, elevating parasite richness (Lafferty 2012, Kamiya et al. 2014a), while also increasing overall predation pressure on the population, lowering mean prevalence. Therefore, we predict that effects of predators on parasite richness will be smaller than effects on parasite prevalence, except in the case of shared parasites where we expect predator richness to strongly increase parasite richness.

Here, we report on a global scale macroecological analysis examining how the effect of predators on parasitism varies with parasite traits. Focusing on ungulates (orders: Artiodactyla and Perissodactyla), a well-studied group of mammals for which both predators (Poelen et al. 2014) and parasites (Hoberg et al. 2001, Stephens et al. 2017) are well-described, we tested the effects predation pressure on the prevalence and richness of five distinct taxonomic groups of parasites: arthropods, bacteria, helminths, protozoa, and viruses. We predicted that (i) predator pressure will decrease parasite prevalence, (ii) predator pressure will decrease richness of unshared parasites but increase richness of shared parasites, and (iii) these effects will vary by parasite transmission mode and taxon.

METHODS

Parasite Data

We collected host-parasite-location records for 104 ungulate (Artiodactyla and Perissodactyla) species in the Global Mammal Parasite Database, Version 2.0 (GMPD) (Stephens et al. 2017). The GMPD contains over 24,000 records from over 2700 primary sources spanning all continents except Antarctica and has been used regularly to study the macroecology and evolution of parasites (Dallas et al. 2017, Teitelbaum et al. 2018, Byers et al. 2019, Cohen et al. 2020, Pappalardo et al. 2020). We cleaned the data of duplicate records and host species with no recorded predators. Parasites were identified to the species level where possible as described in (Stephens et al. 2017) and parasites not identified to species level were excluded from further analysis. Parasite species were classified into broad taxonomic groups: helminths, bacteria, viruses, arthropods,

protozoa, prions, and fungi. Fungi, and prions were excluded from analyses due to extremely small sample sizes. A subset (~81%) of parasites species in the GMPD are also classified according to their transmission modes (Stephens et al. 2017)**.** These are binary classifications of whether the parasite is transmitted by (i) close contact, (ii) non-close contact, (iii) vectors, and (iv) intermediate hosts. Finally parasites were classified as shared if the parasite species was also recorded in carnivores in the GMPD and unshared if there was no such record (Stephens et al. 2019). We counted the number of unique parasites recorded in each host as the measure of parasite species richness (Ezenwa et al. 2006, Teitelbaum et al. 2018). To account for uneven sampling of host species across the database we estimated the true parasite species richness for each host using the Chao2 estimator (Chao 1984, Teitelbaum et al. 2020). To clean prevalence data of repeated measurements of the same population, we took the abundance-weighted mean prevalence across all records from a study with the same recorded location and then excluded any records with fewer than 10 hosts sampled.

Predator Data

To estimate predator pressure, we first collected a list of known mammal predators for all ungulate host species in the GMPD by combining records of species that "prey on" the host from the Global Biotic Interactions database (GloBI) (Poelen et al. 2014), and recorded predators from the University of Michigan's Animal Diversity Web ("Animal Diversity Web" n.d.) and UltimateUngulate.com ("ultimateungulate.com" n.d.). We then used species presence polygons for known predators produced by IUCNRedlist ("The IUCN Red List of Threatened Species" n.d.) to identify which predators a host

species likely experienced in each location where it was recorded in the GMPD. From this list of predators we calculated three predation pressure metrics: mean predator density, mean body mass of predators, and predator species richness. For analyses of parasite richness, we summarized each of these predator values at the host species level by averaging across all recorded locations. Data on predator densities, and host and predator adult body mass were obtained from PanTHERIA, a database of life-history and ecological traits of extant mammals (Jones et al. 2009) or from IUCN Redlist species summary reports ("The IUCN Red List of Threatened Species" n.d.).

Data Analysis

Prevalence Analysis

We used generalized linear (beta-distribution) mixed effect models (GLMMs) to test the effect of predation pressure on parasite prevalence. We fit two models with different predictor variables: for the first model, predictor variables included parasite taxonomic group, mean predator body mass, mean predator density, predator species richness, mean host adult body mass and all two-way interactions. For the second model, predictor variables included all those from the first model as well as the binary indicators of transmission mode: (i) close contact, (ii) non-close contact, (iii) vectors, and (iv) intermediate hosts. This second set of models was fit to a subset of the dataset with recorded parasite transmission mode classifications. Host adult body mass was included as a fixed effect in all models because it tends to predict risk of predation for ungulate host species (Sinclair et al. 2003, Owen-Smith and Mills 2008). The number of hosts sampled in a population was also included as a fixed effect to control for effects of study

size on prevalence estimates. Host species, parasite species, and study were included as random effects. Each model was also fit to subsets of the data representing parasites that were shared between ungulates and carnivores in the GMPD and those that were not shared. When significant interactions were detected marginal trends were estimated with p-values adjusted for multiple comparisons using the Holm method (Holm 1979). We tested for phylogenetic signal in the residuals of our models using a phylogenetic generalized least squares approach, but did not detect any signs of phylogenetic structure (Pagel's lambda not significantly different from 0). Phylogenetic relatedness for the phylogenetic model was derived from an established mammal supertree (Fritz et al. 2009).

Richness Analysis

We used linear models to test the effect of average predation pressure on estimated parasite species richness (ln(Chao2)) in a host species. Predictor variables included: mean predator density, mean body mass of predators, mean adult body mass of host, the number of separate studies of the host species, and all two-way interactions. Mean predator density was excluded from these analyses due to a strong positive correlation with mean predator richness at the host species level (Pearson's $r = 0.63$). Host adult body mass was included for reasons described above and because it is typically a strong predator of parasite species richness (Nunn et al. 2003, Kamiya et al. 2014b). We controlled for sampling bias using the Chao2 estimator as described above but the number of studies on each host was also included as a fixed effect in models as a secondary method to control for this bias. We tested for phylogenetic signal in the

residuals of our models using a phylogenetic generalized least squares approach, but similar to prevalence analyses did not detect any signs of phylogenetic structure. Models were also fit to subsets of the data representing parasites that were shared between ungulates and carnivores in the GMPD and those that were not shared. Additional sub-models separating parasites by transmission mode and taxonomic group were not included due both to the small sample size of some groups and to limit the number of statistical tests conducted.

All data processing and analysis was conducted in R version 3.6.3 (R Core Team 2020). Chao2 values were estimated with a modified version of the *ChaoSpecies* function from the *SpadeR* package (Chao et al. 2016, Teitelbaum et al. 2020). GLMMs were fit using the *glmmTMB* function in the *glmmTMB* package (Brooks et al. 2017) while linear models were fit using the *lm* function in the *stats* package (R Core Team 2020). Nakagawa R^2 values, an R^2 equivalent for GLMMs, were calculated for GLMMs using the *r2* function from the *performance* package. Marginal R^2 values consider only fixed effects, while conditional R^2 values take into account both random and fixed effects (Nakagawa and Schielzeth 2013, Lüdecke et al. 2020). Phylogenetic generalized least squares models were fit using the *pgls* function of the *caper* package allowing all phylogenetic hyperparameters to be fit using maximum likelihood (Orme et al. 2018).

Results

Our dataset included 104 ungulate host species which were host to 840 parasite species (Figure 3.1). Raw parasite species richness per host species ranged from 1 to 144, while estimated parasite species richness (Chao2) ranged up to 409. Host species in the dataset

were associated as prey to 39 mammal predator species, and sampled host populations overlapped with as few as 0 and as many as 12 species of predators per location.

Effects of parasite taxonomy and transmission mode

The effect of predation pressure on parasite prevalence depended on parasite taxon and transmission mode. Only the interactions between parasite taxon and both mean predator body mass ($\chi^2_4 = 14.894$, $p = 0.005$) and predator richness ($\chi^2_4 = 18.849$, $p < 0.001$) were significant (Table 3.1). Virus prevalence was most responsive to predation pressure when controlling for the effects of other covariates, significantly increasing with predator richness ($t = 4.299$, $p < 0.001$; Figure 3.2a) and decreasing with mean predator mass ($t = -3.420$, $p = 0.003$). Overall, the mixed effects model fit the prevalence data very well (Conditional $R^2 = 0.789$), however, the fixed effects explained a limited amount of variation in prevalence (Marginal $R^2 = 0.194$).

When models were fit to the subset of prevalence data for which parasite transmission mode was available, the interaction between predator richness and parasite taxon remained significant ($\chi^2_4 = 10.930$, $p = 0.027$, Table 3.2, Figure 3.2b), although none of the taxon marginal slopes differed from each other or from zero in post-hoc tests. Additionally we found significant interactions between predator richness and the intermediate host transmission mode ($\chi^2_1 = 3.917$, $p = 0.048$, Figure 3.3a) and between mean predator population density and the non-close contact transmission mode ($\chi^2_1 = 5.124$, $p = 0.024$). This subset model fit similarly well to the parasite taxon only model (Conditional $R^2 = 0.810$, Marginal $R^2 = 0.212$).

We failed to detect an effect of predation pressure on parasite richness. Main and interaction effects of all measures of predation pressure on parasite richness were not statistically distinguishable from zero (Table 3.7). The only variable that emerged as a significant predictor of parasite richness was sampling effort (number of studies per host species), although overall the model explained a moderate amount of the variation in parasite richness (Adjusted $R^2 = 0.5116$).

Differences between shared and unshared parasites

Different parasite taxa determine the effect of predator richness on the prevalence of parasites shared and unshared between ungulates and carnivores. Among parasites shared with carnivores, there was a significant interaction between parasite taxon and predator species richness ($\chi^2_4 = 12.797$, $p = 0.012$; Conditional $R^2 = 0.808$, Marginal $R^2 = 0.167$, Table 3.3, Figure 3.2c). This interaction was caused by a significant increase in the prevalence of helminths with increasing predator richness ($t = 2.943$, $p = 0.016$). Likewise, for parasites not shared with carnivores, there was a significant interaction between richness and parasite taxa ($\chi^2_4 = 15.343$, $p = 0.004$; Conditional $R^2 = 0.747$, Marginal $R^2 = 0.217$; Table 3.4, Figure 3.2e). For non-shared parasites, the interaction was driven by significant increases in virus prevalence with predator richness ($t = 3.978$, $p = 0.001$).

In models including parasite transmission mode, covariate interactions differed between shared and unshared parasites. Among shared parasites, there was a significant interaction between parasite taxon and both predator richness ($\chi^2_4 = 10.174$, $p = 0.038$) and mean predator body mass ($\chi^2_1 = 4.676$, $p = 0.031$; Table 3.5, Figure 3.2d). Among

unshared parasites, there was a significant interaction between intermediate host transmission mode and predator richness ($\chi^2_1 = 6.00$, p = 0.014, Figure 3.3c) and between parasite taxon and mean predator body mass ($\chi^2_4 = 12.085$, p = 0.017; Conditional R^2 = 0.801, Marginal R^2 = 0.227; Table 3.6).

We failed to detect an effect of predation pressure on parasite richness in either shared or unshared parasites (Table 3.8, Table 3.9).

Discussion

We examined the effect of predator pressure on parasitism using a global dataset of ungulate parasites. We found that while predator pressure was associated with changes in prevalence, at least for some types of parasites, there was no evidence for effects of predation on parasite species richness. The effects of predation on prevalence depended on parasite taxonomy, transmission mode, and sharing between host and predator. In particular, viruses and helminths were most responsive to predator pressure, typically increasing with increased predation, compared with all other parasite groups which showed no significant response. Likewise, effects of predation pressure on prevalence differed between parasites with and without intermediate hosts. For unshared parasites, i.e. parasites not shared between predators and prey, virus prevalence tended to increase with predator richness, whereas for shared parasites the interaction between parasite type and predator richness was driven by increases in helminths. Although it is impossible to conclusively infer mechanisms from macroecological patterns, we draw some broad conclusions from our results that can motivate and direct future experimental and observational study. First, both parasite transmission mode and other more taxon specific

parasite traits can influence the effect of predation pressure on parasitism in prey. Second, the importance of parasite taxon and transmission mode varies between shared and unshared parasites. Third, effects of predator pressure on parasite prevalence at the population level do not necessarily translate to effects on parasite species richness at the host species level. In combination, these findings suggest that predator pressure can substantially increase parasite prevalence under certain circumstances and that parasite traits are key in shaping this interaction.

We identified both intermediate host and non-close contact transmission as important in moderating the effects of predation pressure on parasitism in prey. This finding suggests that parasites with indirect transmission modes differ in their responses to predation pressure from those transmitted by close-contact or vectors. This difference is not surprising given the considerable variability in the ecology of parasites with indirect and direct transmission modes (Lafferty 1999, Packer et al. 2003, Rauch et al. 2005). For example, we found that predators tended to have a more positive effect on the prevalence of parasites without complex life cycles (intermediate hosts) than those with complex life cycles (Figure 3.3a). This effect may arise because complex life-cycle parasites are less responsive to predator-induced changes in group-size and contact rates of hosts/prey, which increase transmission (Stephenson et al. 2015, Duffy et al. 2019).

While certain parasite transmission modes (e.g. intermediate host transmission) were clearly important for determining the effect of predation pressure on parasite prevalence, there appear to be other traits associated with parasite taxonomy that modulate this effect. The interaction between parasite taxonomic group and predation pressure was significant in both the taxonomy full model and the transmission mode sub-

model, which also included taxonomy, though only in the full model did any of the taxon slopes differ significantly from zero. We suggest that the inclusion of transmission mode in the sub-model accounted for some but not all of the variation explained by parasite taxonomy. This residual difference between parasite taxa might be due to other biological taxonomic differences between parasites. For example, the responsiveness of viruses to local predation pressure in our analysis could be a result of their relatively short infectious periods (Swinton et al. 2002) combined with well documented high transmission rates independent of transmission mode (Hay et al. 2013). As a result of these traits virus transmission may be especially vulnerable to predator induced behavioral changes in host contact rates (Szuroczki and Richardson 2012, Stephenson et al. 2015). Targeted experimental work will be needed to identify the specific parasite traits that explain the effects detected in this study, but our findings support the idea that a variety of parasite traits contribute to variation in predator-prey-parasite interactions (Hatcher et al. 2006, Duffy et al. 2019).

Predator-prey-parasite interactions were also influenced by the degree to which parasites were shared between predators and prey. Viral prevalence increased with predator richness for parasites that were not shared between ungulates and carnivores, while helminth prevalence increased for shared parasites. Although the mechanism underlying this pattern is not identifiable with our approach, there are several plausible hypotheses. In particular, positive effects of predation on shared helminths may be due to transmission to prey from predators, while positive effects of predation for unshared viruses may be due to predator induced changes to prey physiology or behavior. The differential importance of transmission between predators and prey for helminths

compared to other parasite groups is supported by the literature (Murthy et al. 2013, Thieltges et al. 2013, Friesen and Roth 2016, Stephens et al. 2019), but much of this support comes from parasites that require trophic transmission. Interestingly, in our analysis, an intermediate host transmission mode significantly influenced the effect of predator richness on parasite prevalence for unshared (Figure 3.3c) but not shared (Figure 3.3b) parasites. Heterogeneity in the intermediate host transmission mode classification in our parasite database may explain this pattern. First, both parasites that use ungulates as intermediate hosts with carnivores as definitive hosts, and parasites that use invertebrates as intermediate hosts with ungulates as definitive hosts are identified as having an intermediate host transmission mode in the GMPD (Stephens et al. 2017). Therefore, unshared parasites assigned an intermediate host transmission mode are primarily environmentally transmitted without a role for carnivores in transmission, while shared parasites assigned an intermediate host transmission mode are likely a combination of the former parasite type and parasites for which ungulates are obligate intermediate hosts. As a result, intermediate host transmission is a heterogeneous and messy predictor of predator-prey-parasite interactions for shared parasites, while it more cleanly represents indirect transmission for unshared parasites. We found that parasite taxonomy and parasite sharing interact to influence the effects of predation on parasitism in prey. In particular, predation pressure increased prevalence in both groups for different parasites.

Despite clear effects of predation on parasite prevalence, there were no effects of predators on parasite richness. We predicted that predator pressure would decrease parasite richness as rare parasites are eliminated, but that the richness of parasites that are shared with carnivores would increase with increasing predator pressure. However, we

found no effect of predation on parasite richness even when shared and unshared parasites were analyzed separately. The lack of a protective effect of predators against parasite richness may be because decreases in parasite prevalence due to increased predation are small in magnitude and variable across parasites (Figure 3.3). Under these circumstances many rare parasites might escape extinction in prey. Alternatively, while differences in local predation pressure influenced the prevalence of parasites in our study, the average predation pressure experienced by a prey species across all locations, the metric we used for the parasite richness analyses, might be a less meaningful metric. Many host traits (e.g. body mass, home range size, social group size, population density, and migratory behavior) have been identified as important predictors of parasite species richness in ungulates (Ezenwa et al. 2006, Lindenfors et al. 2007, Teitelbaum et al. 2018), so average predation pressure may be inconsequential in comparison with many of these more proximate drivers of parasitism at the species level. Given these findings, more refined tests of the effect of predation on parasite richness are needed that compare parasite richness between populations of a single species experiencing different levels of predation pressure.

One of the main limitations of our study is the broad and imprecise way in which we measured predation pressure. First, we focused only on mammalian predators which ignores a variety of other organisms (e.g. birds of prey and reptiles) that prey on many ungulates (Poelen et al. 2014). This choice was made for two primary reasons: (i) mammalian predators of ungulates are better documented and their ranges are more commonly estimated ("The IUCN Red List of Threatened Species" n.d.) and (ii) mammalian predators are included in the GMPD making analyses of shared and unshared

parasites possible (Stephens et al. 2017). Second, our estimates of predation pressure rely on predator richness and average estimates of predator population density and body size. These measures, particularly predator population density, rely on the assumption that predators occur at a consistent average density across their entire range. This assumption is clearly false, but estimating or predicting population densities of wide ranging large carnivore species is remarkably difficult (Gros et al. 1996, Obbard et al. 2010, Ripple et al. 2014, Jędrzejewski et al. 2018), making more precise estimates beyond the scope of this work. Finally, since our study was observational, it is not possible to infer the causal direction of observed effects. For example, local predator pressure is correlated with the size of prey populations, and larger prey populations support more and higher prevalence parasites regardless of predation (Arneberg et al. 1998, Arneberg 2001). Likewise, predation pressure measures likely correlate with other proximate drivers of parasitism such as resource availability and host species diversity (VanderWaal and Ezenwa 2016, Rohr et al. 2020). Limitations such as these are common in macroecological studies (Dallas et al. 2018, Teitelbaum et al. 2018). Despite these limitations, our findings still suggest relationships between predation pressure and parasite richness worthy of additional study.

In conclusion, we found that the overall effects of predation pressure on parasite prevalence vary significantly according to the parasite taxonomy, parasite transmission mode, and whether parasites are shared between prey and predators. Interestingly, predator pressure had a negligible effect on parasite richness at the host species level, but targeted study of the effects of predator pressure on parasite richness within a single host species is needed. Our results support the idea that a variety of parasite traits moderate

predator-prey-parasite interactions (Hatcher et al. 2006, Duffy et al. 2019). Furthermore, our finding that the many sub-groups of parasites significantly and independently increased with predation pressure supports an emerging consensus (Duffy et al. 2019, Chapter 2) that protective effects of predators on parasites are uncommon and that predators frequently increase parasitism in their prey.

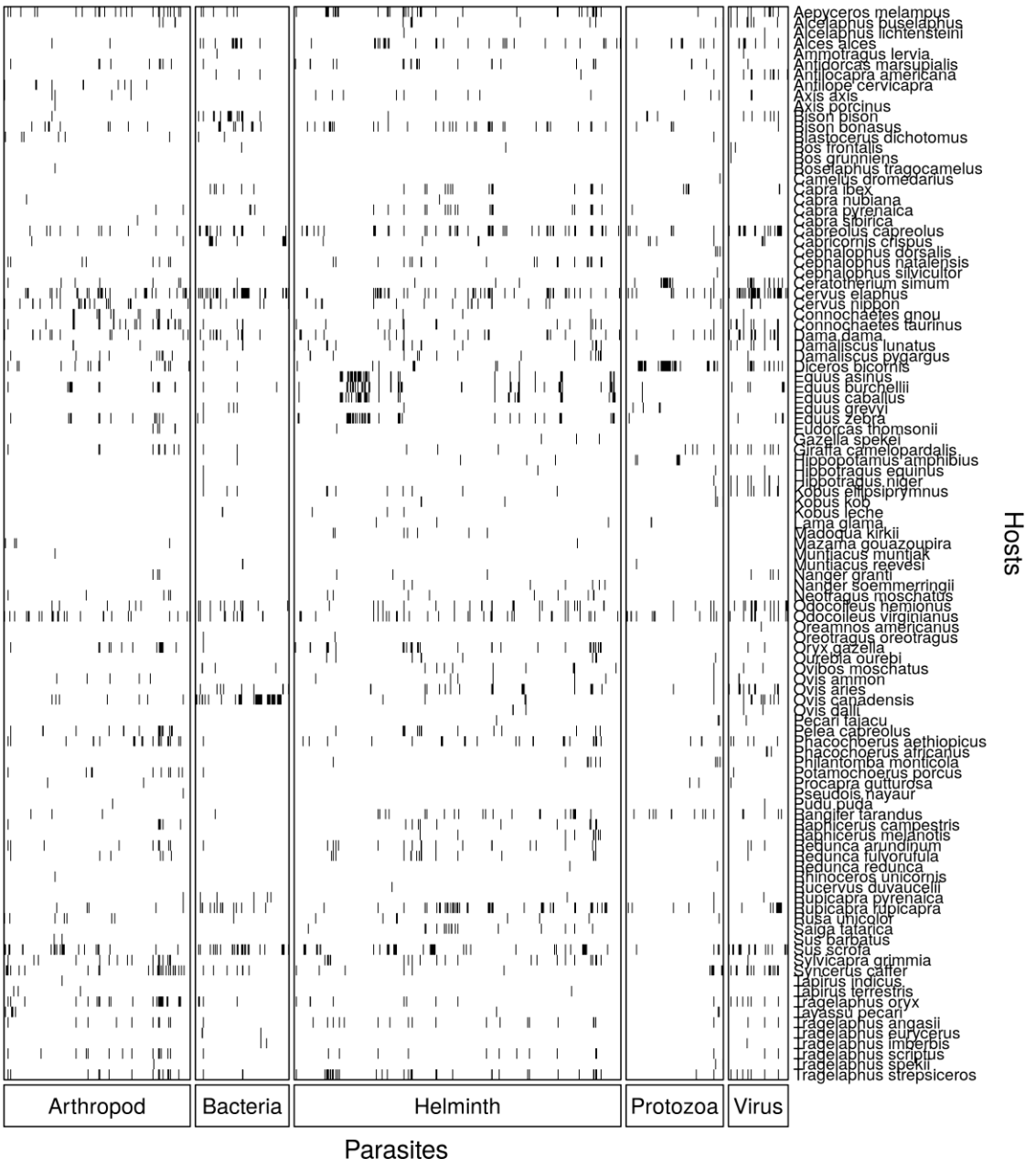

Figure 3.1 A matrix displays which parasite species were detected in each host species.

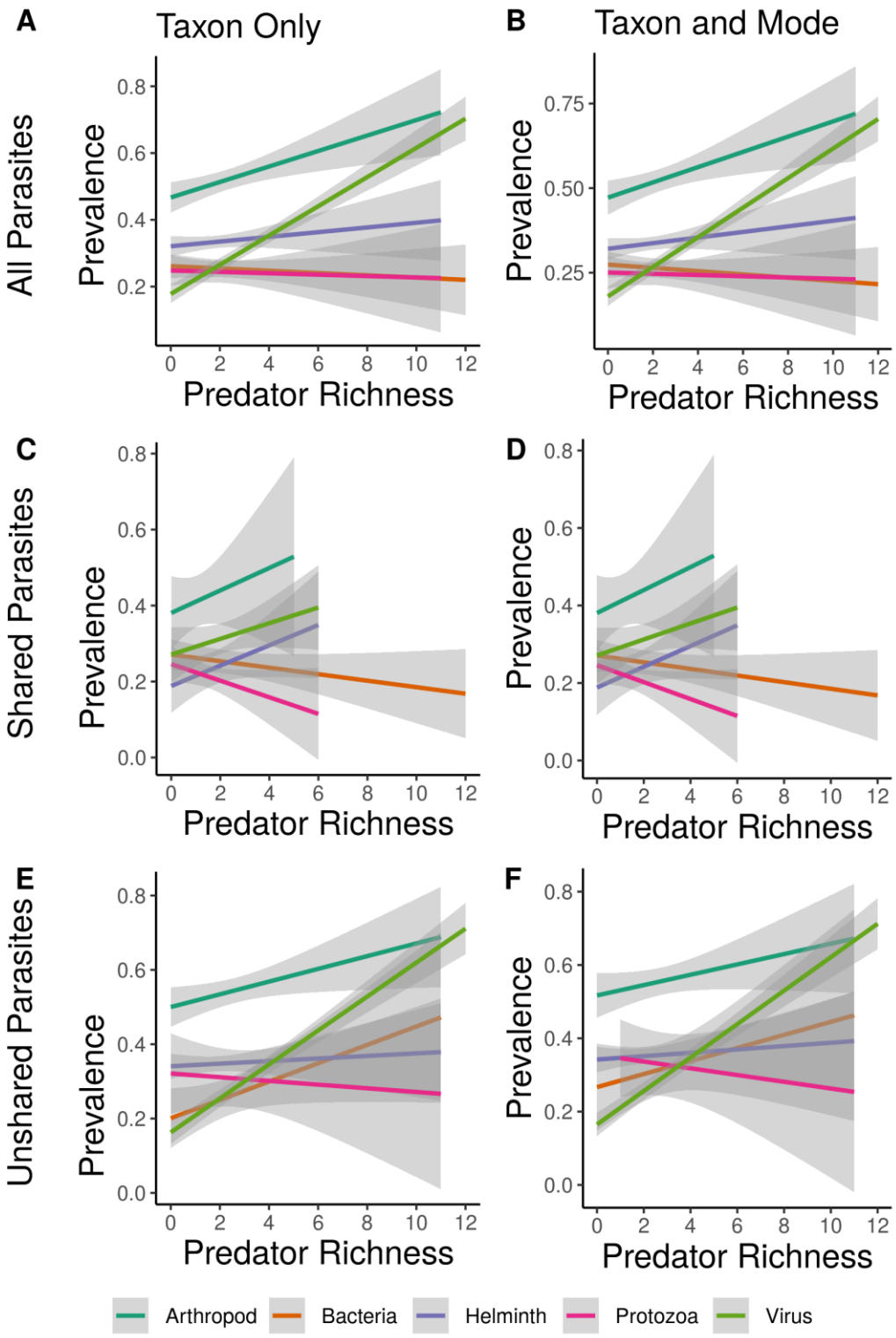

Figure 3.2: The effect of predator species richness on parasite prevalence, separated by parasite taxonomic group for (a, c, e) models including parasite taxon and (b, d, f) models including parasite taxon and transmission modes, including (a, b) all parasites, (c, d) parasites shared between ungulates and carnivores, and (e, f) unshared parasites.

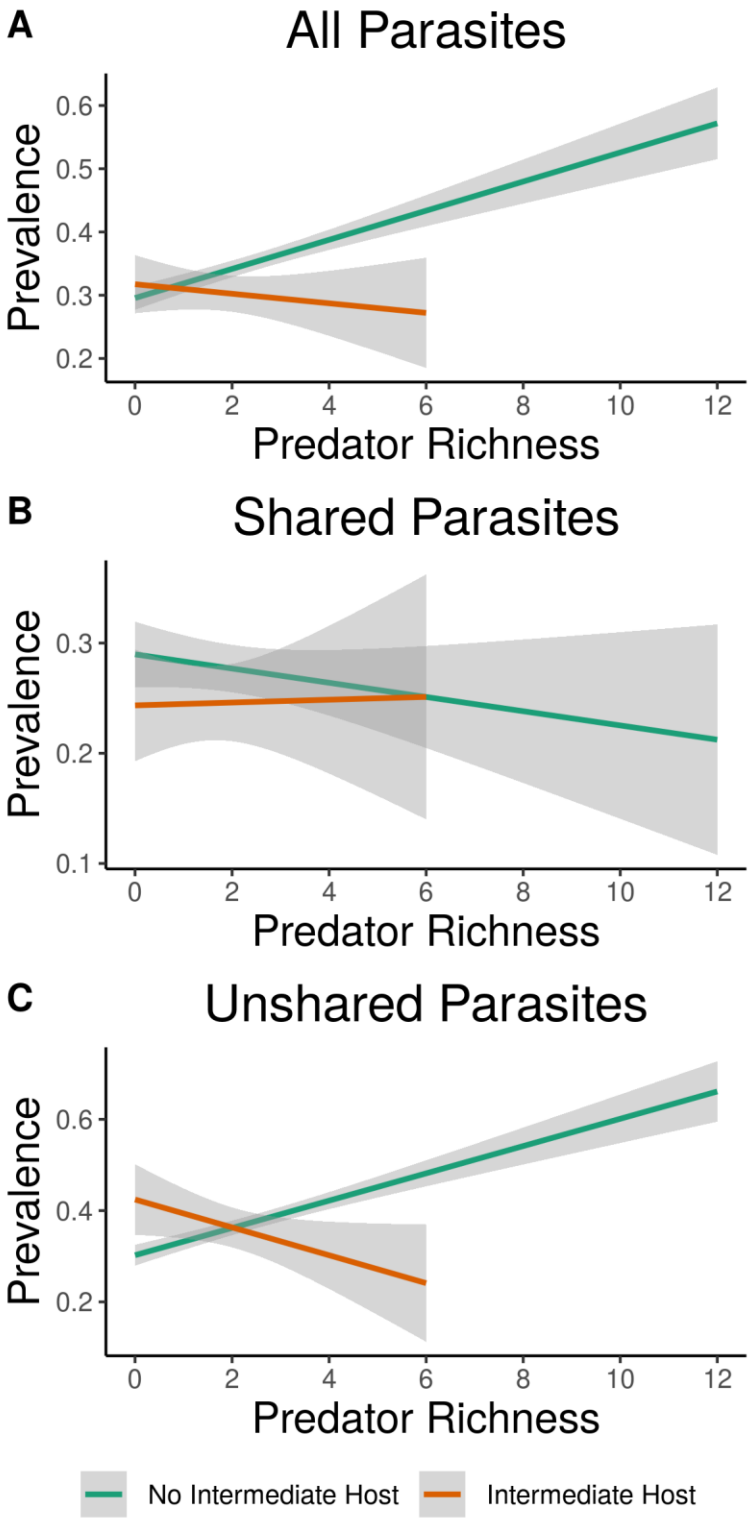

Figure 2: The effect of predator species richness on parasite prevalence separated by presence/absence of an intermediate host transmission mode for (a) all parasites, (b) parasites shared between ungulates and carnivores, and (c) unshared parasites.

Table 3.1 Model results predicting parasite prevalence across all parasites

	Chisq	Df	p
(Intercept)	79.2253	1	**<0.001**
Parasite Type	49.2478	4	**<0.001**
Mean predator body mass	1.0510	1	0.3053
Mean predator population density	1.3906	1	0.2383
Predator Richness	2.2928	1	0.13
Host adult body mass	0.4943	1	0.482
Number of Hosts Sampled	1.6411	1	0.2002
Parasite Type:Mean predator body mass	14.8935	4	**0.0049**
Parasite Type:Mean predator population density	5.4957	4	0.2401
Mean predator body mass:Mean predator population density	1.4046	1	0.2359
Parasite Type:Predator Richness	18.8486	4	**0.0008**
Mean predator body mass:Predator Richness	0.0500	1	0.8231
Mean predator population density:Predator Richness	0.0226	1	0.8806
Mean predator body mass:Host adult body mass	0.4463	1	0.5041
Mean predator population density:Host adult body mass	0.0085	1	0.9264
Predator Richness:Host adult body mass	0.4373	1	0.5084

Table 3.2 Model results predicting parasite prevalence across parasites with transmission mode classifications.

	Chisq	Df	p
(Intercept)	19.7273	1	**<0.001**
close	3.0095	1	0.0828
Mean predator body mass	0.0301	1	0.8624
Mean predator population density	0.0691	1	0.7927
Predator Richness	0.6964	1	0.404
nonclose	0.0298	1	0.863
intermediate	0.5995	1	0.4388
vector	0.0428	1	0.8362
Host adult body mass	0.3556	1	0.551
Number of Hosts Sampled	1.7456	1	0.1864
Parasite Type	24.8058	4	**0.0001**
close:Mean predator body mass	0.0026	1	0.9595
close:Mean predator population density	2.9018	1	0.0885
close:Predator Richness	0.0465	1	0.8292
Mean predator body mass:nonclose	1.6333	1	0.2013
Mean predator population density:nonclose	5.1236	1	**0.0236**
Predator Richness:nonclose	3.0535	1	0.0806
Mean predator body mass:intermediate	0.7510	1	0.3862
Mean predator population density:intermediate	0.0173	1	0.8953
Predator Richness:intermediate	3.9172	1	**0.0478**
Mean predator body mass:vector	0.0812	1	0.7757
Mean predator population density:vector	0.8034	1	0.3701
Predator Richness:vector	1.0320	1	0.3097
Mean predator body mass:Mean predator population density	0.2120	1	0.6452
Mean predator body mass:Predator Richness	0.3425	1	0.5584
Mean predator population density:Predator Richness	0.0101	1	0.92
Mean predator body mass:Host adult body mass	0.3165	1	0.5737
Mean predator population density:Host adult body mass	0.0513	1	0.8208
Predator Richness:Host adult body mass	0.6164	1	0.4324
Mean predator body mass:Parasite Type	8.0703	4	0.089
Mean predator population density:Parasite Type	6.4715	4	0.1666
Predator Richness:Parasite Type	10.9298	4	**0.0274**

Table 3.3 Model results predicting parasite prevalence in shared parasites.

	Chisq	Df	p
(Intercept)	40.1420	1	**<0.001**
Parasite Type	13.2309	4	**0.0102**
Mean predator body mass	0.6413	1	0.4232
Mean predator population density	0.0429	1	0.8359
Predator Richness	0.2235	1	0.6364
Host adult body mass	0.8794	1	0.3484
Number of Hosts Sampled	2.1061	1	0.1467
Parasite Type:Mean predator body mass	6.0178	4	0.1978
Parasite Type:Mean predator population density	6.1358	4	0.1892
Mean predator body mass:Mean predator population density	1.9992	1	0.1574
Parasite Type:Predator Richness	12.7966	4	**0.0123**
Mean predator body mass:Predator Richness	3.1572	1	0.0756
Mean predator population density:Predator Richness	0.8528	1	0.3557
Mean predator body mass:Host adult body mass	0.0669	1	0.7959
Mean predator population density:Host adult body mass	0.5275	1	0.4676
Predator Richness:Host adult body mass	0.2763	1	0.5992

Table 3.4 Model results predicting parasite prevalence in unshared parasites

	Chisq	Df	p
(Intercept)	64.8343	1	**<0.001**
Parasite Type	49.9528	4	**<0.001**
Mean predator body mass	6.9373	1	**0.0084**
Mean predator population density	1.6284	1	0.2019
Predator Richness	6.8784	1	**0.0087**
Host adult body mass	0.8185	1	0.3656
Number of Hosts Sampled	1.8973	1	0.1684
Parasite Type:Mean predator body mass	19.1312	4	**0.0007**
Parasite Type:Mean predator population density	4.3305	4	0.3631
Mean predator body mass:Mean predator population density	1.4894	1	0.2223
Parasite Type:Predator Richness	15.3427	4	**0.004**
Mean predator body mass:Predator Richness	0.4001	1	0.5271
Mean predator population density:Predator Richness	0.0627	1	0.8022
Mean predator body mass:Host adult body mass	0.4842	1	0.4865
Mean predator population density:Host adult body mass	0.0193	1	0.8894
Predator Richness:Host adult body mass	0.4423	1	0.506

Table 3.5 Model results predicting parasite prevalence in shared parasites with transmission mode classifications.

	Chisq	Df	p
(Intercept)	8.9842	1	**0.0027**
close	1.2651	1	0.2607
Mean predator body mass	0.5874	1	0.4434
Mean predator population density	0.2053	1	0.6505
Predator Richness	0.0672	1	0.7954
nonclose	0.4440	1	0.5052
intermediate	0.2526	1	0.6152
vector	1.4026	1	0.2363
Host adult body mass	0.0433	1	0.8352
Number of Hosts Sampled	2.0672	1	0.1505
Parasite Type	15.7175	4	**0.0034**
close:Mean predator body mass	0.2285	1	0.6327
close:Mean predator population density	0.8803	1	0.3481
close:Predator Richness	0.4793	1	0.4887
Mean predator body mass:nonclose	0.3971	1	0.5286
Mean predator population density:nonclose	0.9741	1	0.3237
Predator Richness:nonclose	0.2236	1	0.6363
Mean predator body mass:intermediate	0.2512	1	0.6162
Mean predator population density:intermediate	0.4538	1	0.5006
Predator Richness:intermediate	0.0486	1	0.8254
Mean predator body mass:vector	0.0023	1	0.9616
Mean predator population density:vector	1.9527	1	0.1623
Predator Richness:vector	1.8332	1	0.1757
Mean predator body mass:Mean predator population density	3.0089	1	0.0828
Mean predator body mass:Predator Richness	4.6761	1	**0.0306**
Mean predator population density:Predator Richness	0.4186	1	0.5176
Mean predator body mass:Host adult body mass	0.2977	1	0.5853
Mean predator population density:Host adult body mass	0.1960	1	0.658
Predator Richness:Host adult body mass	0.1301	1	0.7183
Mean predator body mass:Parasite Type	3.8046	4	0.4331
Mean predator population density:Parasite Type	5.6398	4	0.2277
Predator Richness:Parasite Type	10.1737	4	**0.0376**

Table 3.6 Model results predicting parasite prevalence in unshared parasites with transmission mode classifications.

	Chisq	Df	p
(Intercept)	5.8554	1	**0.0155**
close	0.4366	1	0.5088
Mean predator body mass	2.0532	1	0.1519
Mean predator population density	0.0291	1	0.8646
Predator Richness	0.6190	1	0.4314
nonclose	1.2237	1	0.2686
intermediate	3.4619	1	0.0628
vector	0.2943	1	0.5875
Host adult body mass	0.5844	1	0.4446
Number of Hosts Sampled	1.6796	1	0.195
Parasite Type	19.9959	4	**0.0005**
close:Mean predator body mass	0.0474	1	0.8276
close:Mean predator population density	0.8332	1	0.3613
close:Predator Richness	0.0096	1	0.9221
Mean predator body mass:nonclose	0.3463	1	0.5562
Mean predator population density:nonclose	2.4239	1	0.1195
Predator Richness:nonclose	2.4822	1	0.1151
Mean predator body mass:intermediate	0.2669	1	0.6054
Mean predator population density:intermediate	0.7824	1	0.3764
Predator Richness:intermediate	5.9997	1	**0.0143**
Mean predator body mass:vector	0.2357	1	0.6273
Mean predator population density:vector	0.5762	1	0.4478
Predator Richness:vector	1.2175	1	0.2699
Mean predator body mass:Mean predator population density	0.4858	1	0.4858
Mean predator body mass:Predator Richness	0.2291	1	0.6322
Mean predator population density:Predator Richness	0.2097	1	0.647
Mean predator body mass:Host adult body mass	0.1860	1	0.6662
Mean predator population density:Host adult body mass	0.0261	1	0.8716
Predator Richness:Host adult body mass	0.6727	1	0.4121
Mean predator body mass:Parasite Type	12.0853	4	**0.0167**
Mean predator population density:Parasite Type	3.3060	4	0.508
Predator Richness:Parasite Type	5.9251	4	0.2048

Table 3.7 Model results predicting estimated parasite species richness (Chao2) of hosts.

	Sum Sq	Df	t	p
(Intercept)	145.0729	1	62.6194	**<0.001**
Mean predator body mass	1.2196	1	0.5264	0.4699
Mean predator richness	1.2010	1	0.5184	0.4733
Host adult body mass	0.6496	1	0.2804	0.5977
Number of Studies	10.0055	1	4.3188	**0.0404**
Mean predator body mass:Mean predator richness	2.6223	1	1.1319	0.2901
Mean predator body mass:Host adult body mass	2.3857	1	1.0297	0.3129
Mean predator body mass:Number of Studies	0.5244	1	0.2263	0.6354
Mean predator richness:Host adult body mass	0.0256	1	0.0111	0.9164
Mean predator richness:Number of Studies	0.6192	1	0.2673	0.6064
Host adult body mass:Number of Studies	0.0159	1	0.0069	0.9342

Table 3.8 Model results predicting estimated parasite species richness (Chao2) of shared parasites across hosts.

	Sum Sq	Df	t	p
(Intercept)	27.8699	1	25.5423	**<0.001**
Mean predator body mass	0.8588	1	0.7871	0.3784
Mean predator richness	2.2340	1	2.0475	0.1575
Host adult body mass	0.0641	1	0.0587	0.8093
Number of Studies	2.4856	1	2.2780	0.1363
Mean predator body mass:Mean predator richness	0.4668	1	0.4279	0.5155
Mean predator body mass:Host adult body mass	0.0290	1	0.0266	0.871
Mean predator body mass:Number of Studies	0.7057	1	0.6468	0.4243
Mean predator richness:Host adult body mass	0.3634	1	0.3331	0.5659
Mean predator richness:Number of Studies	0.8471	1	0.7763	0.3817
Host adult body mass:Number of Studies	0.4011	1	0.3676	0.5465

Table 3.9 Model results predicting estimated parasite species richness (Chao2) of unshared parasites across hosts.

	Sum Sq	Df	t	p
(Intercept)	103.5440	1	47.6046	**<0.001**
Mean predator body mass	0.0254	1	0.0117	0.9142
Mean predator richness	2.3334	1	1.0728	0.3031
Host adult body mass	0.7700	1	0.3540	0.5534
Number of Studies	14.3157	1	6.5817	**0.012**
Mean predator body mass:Mean predator richness	2.2094	1	1.0158	0.3162
Mean predator body mass:Host adult body mass	0.1572	1	0.0723	0.7887
Mean predator body mass:Number of Studies	1.3724	1	0.6310	0.4291
Mean predator richness:Host adult body mass	0.1310	1	0.0602	0.8067
Mean predator richness:Number of Studies	2.0597	1	0.9470	0.3331
Host adult body mass:Number of Studies	0.7061	1	0.3246	0.5703

CHAPTER 4

SEASON AND PREY IDENTITY MEDIATE THE EFFECT OF PREDATORS ON PARASITES IN RODENTS: A TEST OF THE HEALTHY HERDS HYPOTHESIS[3]

[3] Richards R.L., Conner L.M., Morris G., Drake J.M., V.O. Ezenwa. Submitted to *Journal of Animal Ecology*.

ABSTRACT

1. The healthy herds hypothesis (HHH) suggests that predators decrease parasitism in their prey. Repeated tests of this hypothesis across a range of taxa and ecosystems have revealed significant variation in the effect of predators on parasites in prey.
2. Differences (i) between prey taxa, (ii) between seasons, and (iii) before and after catastrophic disturbance are common in natural systems, but typically ignored in empirical tests of the HHH. We used predator exclusion experiments to measure the effect of these heterogeneities on the tri-trophic interaction among predators, parasites and prey.
3. We experimentally excluded mammalian predators from the habitats of cotton rats (*Sigmodon hispidus*) and cotton mice (*Peromyscus gossypinus*) and measured the effect of exclusion on gastrointestinal parasites in these rodents. Our experiment spanned multiple seasons and before and after a prescribed burn.
4. We found that the exclusion of the same predators can have opposite effects on the parasites of similar prey species. Additionally, we found that the effect of mammal exclusion on parasitism can differ before versus after fire disturbance. Finally, we saw that the effect of predator exclusion is highly dependent on prey capture season. Significant effects of exclusion emerged primarily in the fall and winter months.
5. The presence of so many different effects in one relatively simple system suggests that predator effects on parasites in prey are highly context-dependent. We

recommend that future studies span multiple seasons and consider explicitly the differences and interactions among different predators.

INTRODUCTION

Ecological theory suggests that predators keep herds "healthy" by decreasing parasitism in their prey (Packer et al. 2003). Predators cull infected individuals from populations, often selectively (Hudson et al. 1992a, Stephenson et al. 2016, Gehman and Byers 2017), and decrease densities of both infected and susceptible hosts, limiting transmission (Dobson 1990, Arneberg et al. 1998). The *healthy herds hypothesis* (HHH) summarizes these predictions (Packer et al. 2003). However, empirical tests of the HHH have yielded conflicting results (Lafferty 2004, Groner and Relyea 2015, Koprivnikar and Urichuk 2017, Buss and Hua 2018). Heterogeneities in a variety of factors have been found to explain the presence, strength, and direction of responses in another, better studied, tri-trophic interaction, the trophic cascade (Norrdahl and Korpimäki 1995, Schmitz et al. 2004), suggesting that similar factors may be important to understanding predator-prey-parasite (PPP) interactions. As a result, it is unsurprising that empirical tests of the HHH consistently fail to support the underlying theory, because the theory assumes that systems are composed of a single generalist predator and a single homogenous prey population, and that non-predation components of systems remain constant through time. While additional theory has been developed to explore the outcome of violating a number of these assumptions of homogeneity (e.g. Choisy and Rohani 2006, Holt and Roy 2007), empirical work has largely focused on testing the central hypothesis. However, explicit empirical tests of the violation of homogeneity

assumptions are required to make reliable predictions about the potential effects of predator loss or reintroduction on parasites.

Variability among host/prey species is one major source of heterogeneity that can influence PPP interactions. Most theoretical and empirical tests of the HHH measure the effects of predator pressure on a single host/prey species (but see Hofmeester et al. 2017), obscuring important variation in how predators can influence parasitism in different prey species. Some predators may show strong preferences based on prey species identity or size (Pearre 1982, Dickman et al. 1991, Koivunen et al. 1996, Post et al. 2000). This type of variation can result in differences among prey species in the effect of predation on the population density and age, size, or sex structure of the population, each of which can in turn influence parasitism (Schalk and Forbes 1997, Wilson et al. 2002, Begon et al. 2002). For example, the exclusion of mammalian predators increases the mean body mass of cotton rat prey, suggesting that mammalian predators selectively remove larger, older, rats (Morris and Conner 2019). Since parasites are often highly aggregated in older and larger individuals (Pacala and Dobson 1988, Wilson et al. 2002) the loss of mammalian predation should have larger consumptive effects, effects due to direct killing of prey by the predator, on cotton rat parasites than on parasites of other host species whose size distributions are unaffected by predation. Differences in prey behavioral responses to predation risk and predator preferences may also result in variation in the non-consumptive effects of predators. Prey species vary in both their physiological and behavioral response to predation pressure (Preisser and Orrock 2012). While these non-consumptive effects of predators on prey can have both positive and negative effects on parasitism, on average, they tend to frequently increase parasitism in prey (Stephenson et

al. 2015, Koprivnikar and Urichuk 2017, Buss and Hua 2018). Thus, the parasites of prey species that experience effects of predation on population size, size structure, or sex ratio are expected to be most strongly influenced by consumptive effects of predation. In these species, we expect predators to decrease parasitism in prey. However, when predators do not directly influence a species' population density, size structure, or sex ratio we expect that non-consumptive effects of predators will increase parasitism in the prey.

Seasonal variation can also introduce heterogeneities that shape PPP interactions. For example, in systems where predators are seasonal migrants (e.g. migrating birds of prey; Smith et al. 2006, Farmer et al. 2007), the impact of predation on parasitism in prey is expected to occur primarily in the seasons when predators are present. Even resident predators show decreased activity in some seasons (e.g. some mammals and snakes; Gibbons and Dorcas 2005, Conner et al. 2011), suggesting that seasonal variation in predator activity should have strong effects on PPP interactions. Moreover, prey population size and behavior often varies seasonally, due to changes in food availability, reproduction, and torpor or hibernation (Merritt et al. 2001, Morris et al. 2011c, 2011a, 2011b), all of which can alter the relative availability of prey to different predator species and directly affect parasite distributions in prey populations. Consequently, we expect the effect of most predators on parasitism in prey to also vary seasonally.

Abundance and behavior of predators and prey also vary in response to disturbance events. In particular, fire frequently alters the distribution of animals on a landscape because some individuals vacate recently burned areas, while others colonize these areas, others do not survive the fire itself, and still others remain essentially unaffected (Hatchell 1964, Fox 1982, Kelly et al. 2018). Disturbances, like fire, which

drastically alter the vegetation structure of a landscape might directly affect interactions between predators and prey by reshaping the risk landscape. For example, the risk of predation for cotton rats in burned portions of longleaf pine savannas is significantly higher than that in unburned areas (Conner et al. 2011). As a result, the effect of predation on parasitism in prey populations is likely to vary according to the recent history of disturbances like fire. If predation tends to decrease abundance or shift size and sex structure towards smaller individuals and females in some seasons or burn regimes but not others, then we expect these periods to show the strongest negative effects of predation on parasitism.

Experimental exclusion of predators is a common method of quantifying the effect of predators on prey populations (Krebs et al. 1995, Morris et al. 2011b). In this study, we used this type of manipulative approach to quantify the effects of predator exclusion on parasitism in prey populations, and to evaluate whether these effects depended on (i) prey/host identity, (ii) seasonal variation, and (iii) fire disturbance. We focused on a small mammal prey community subject to avian, mammalian, and snake predation and dominated by two rodent species: hispid cotton rats (*Sigmodon hispidus*) and cotton mice (*Peromyscus gossypinus*) (Smith et al. 2006, Morris et al. 2011a, 2011b, 2011c). We performed a large-scale manipulative experiment to study the effect of predator removal on this small mammal community. The experiment consisted of a large-scale mammalian meso-predator exclusion, in which we monitored gastrointestinal parasites of both study species for two years, encompassing two full seasonal cycles and one prescribed burn event. We asked three questions about the context-dependency of PPP interactions: (i) Does the exclusion of mammalian predators differentially affect

parasites of different prey species? (ii) Does the effect of predator exclusion vary seasonally, and/or (iii) in response to prescribed burning?

We predicted that mammalian predator exclusion would elevate parasitism in cotton rats, due to elevated cotton rat body mass in exclosures (Morris and Conner 2019), and in cotton mice in the time following prescribed burns due to increases in their survival in exclosures after burns (Morris et al. 2011c). We also expected that seasonality in the effects of predation would result from differences between effects in winter, when mammal predation is rarest and spring/summer when mammal predation is most common (Conner et al. 2011).

METHODS

Terrestrial mammal predator exclusion experiment

Our terrestrial mammalian predator exclusion experiment was conducted at The Jones Center at Ichauway in Baker County, Georgia (31.22°, -84.48°). Longleaf pine (*Pinus palustris*) and wiregrass (*Aristida beyrichiana*) cover much of the 12,000-ha property, and the community of small mammals in this habitat is dominated by cotton rats and cotton mice. Longleaf pine ecosystems are fire-dependent and sites were burned in the winter of 2017 according to a 2-year prescribed burn regime (Atkinson et al. 1996). Previous research in our study sites has found that cotton rats and cotton mice were affected by fire in different ways. Cotton rat populations declined precipitously due to predation and emigration immediately following a burn (Conner et al. 2011, Morris et al. 2011b). Cotton mouse populations persisted through fire and exhibit increased survival after fire in the absence of mammalian predators (Morris et al. 2011c). Both species were

reliably trapped seasonally in the predator manipulation plots (Morris et al. 2011c, 2011b), and both were subject to predation from multiple predator guilds, including raptors, meso-mammalian carnivores, and snakes (Smith et al. 2006, Derrick et al. 2010, Conner et al. 2011). Cotton rats and mice also host a suite of gastrointestinal parasites (Kinsella 1974, 1991, Bergstrom et al. 2019, Thompson et al. 2019), making this system highly tractable for addressing questions about PPP interactions.

In 2002, the Jones Center constructed four ~40 ha terrestrial mesopredator exclosures and four control plots within similar habitat. The exclosures are surrounded by 1.2-m-tall woven wire fence with electrified lines running along it to discourage mammals from climbing over or digging under the fence. Excluded predator species included coyotes (*Canis latrans*), grey foxes (*Urocyon cinereoargenteus*), red foxes (*Vulpes vulpes*), raccoons (*Procyon lotor*), Virginia opossums (*Didelphis virginiana*), striped skunks (*Mephitis mephitis*), spotted skunks (*Spilogale putorius*), and nine-banded armadillos (*Dasypus novemcinctus*). Raptors and snakes were not excluded and have been shown to vary seasonally in their density and activity (Smith et al. 2006, Conner et al. 2011). Mammal predator presence in control and exclosure plots was monitored regularly with track counts and thermal camera surveys (Conner et al. 2010, Morris et al. 2011b). Although mammal predators occasionally penetrated the fence, the exclosures reduced mesopredator presence by approximately 90% in prior studies (Conner et al. 2011). We collected data on mammalian predator presence in both control and exclosure plots for 3 nights each season using track surveys in 1.5m by 0.5m raked sandy areas in each plot. The effect of mammalian predator exclusion on other rodent predators is well characterized in this system. There was no difference in the proportion of cotton rat

mortality due to snakes in the exclosures historically (Conner et al. 2011) and the abundance of snakes did not significantly differ between exclosure and control plots during the years of the study (Howze et al. Unpublished data). The proportion of cotton rat mortality due to avian predators was significantly greater in exclosure plots than control plots previously (Conner et al. 2011), but neither avian predator prevalence nor predation rates were directly measured during the period of this study.

Each control and exclosure plot contained a 12 trap × 12 trap small mammal trapping grid with 15-m spacing between trapping stations. Within exclosure plots these grids were placed in the center of exclosures so that the home ranges of all trapped rodents should fall entirely within the exclosure (Morris et al. 2011c, 2011a). For this study, grids were trapped four times per year (once each season) from October 2016 through August 2018 using baited Sherman live traps (H.B. Sherman Traps, Tallahassee, FL, USA). Granular insecticide (Bifen L/P Insecticide Granules, Control Solutions, Inc, Pasadena, TX, USA) was sprinkled around each trap to deter fire ants. New captures were marked individually with metal ear tags. Data recorded for all captures included site, trap location, species, sex, mass, reproductive condition (for males, testes descended or not, and for females, pregnant and/or lactating or not), and hind foot length. Fecal samples were collected from traps after animal release and stored on ice packs while in the field and then at ~4°C in the lab until processing.

Parasite Analysis

A modified double centrifugation method was used to quantify the number of helminth parasite eggs present in fecal samples (Foreyt 2013). Briefly, fecal samples

were weighed, homogenized in water and then concentrated by centrifugation. Pellets were resuspended in sugar solution (specific gravity = 1.27) and concentrated on a single microscope coverslip. Helminth eggs found on the coverslip were identified to taxonomic group and morphotype according to size and morphological characteristics and quantified under a compound microscope at 40× magnification. Fecal egg counts were used as a proxy for parasite abundance within the host (Cabaret et al. 1998, Pedersen and Antonovics 2013). Common parasites of cotton rats included two strongyle nematode morphotypes (strongyle 1 and strongyle 2, Figure 4.2a and b), and one spirurid nematode morphotype (Figure 4.2c). Common parasites of cotton mice included the same two strongyle nematode morphotypes described in cotton rats (Figure 4.2a and b). We were not able to identify parasites to the species-level, but strongyle nematodes of rodents are generally transmitted between host individuals via a fecal oral route (Morand et al. 2006), while spirurid nematodes of cotton rats typically have an insect intermediate host (Schell 1952, Kinsella 1974). Two additional parasite morphotypes present in less than 10% of cotton rats and mice were excluded from analyses due to issues with model fitting.

Statistical Analysis

We used a generalized linear mixed model (GLMM) to test for an effect of the exclosure treatment on mammalian predator track counts per observation night as an index of mammalian predator abundance. Models included exclosure treatment, season, burn status and all 2-way interactions as fixed effects and plot as a random effect. We also used identical GLMMs to test for an effect of the exclosure treatment on the estimated abundance of cotton rats and cotton mice in each plot. Abundances were estimated using Huggins closed population models for each plot in each season (Huggins

1989). Only grid-seasons with at least 4 individual capture histories were used for estimation due to constraints of the model. Additionally, we tested for an effect of exclosure treatment on cotton rat/cotton mouse body mass and sex ratio using GLMMs with fixed effects of treatment, season, burn status, and all 2-way interactions (sex model), and sex, treatment, season, burn status, and all 2-way interactions (mass model) with a random effect of plot included in both models.

We also used GLMMs to test for a relationship between exclosure treatment and parasite abundance. In this case, models were fit with both negative binomial and zero-inflated negative binomial distributions to account for aggregation in parasite count data. The best-fitting model was selected using the corrected Akaike Information Criterion (Anderson and Burnham 2002). All parasite models included the random effects of individual, nested within trapping block, to control for consistent variation in parasite abundances among individuals and blocks. Exclosure treatment, host mass, sex, season, burn status, and the interactions of all the latter terms with exclosure treatment were included as fixed effects. All models were assessed for violation of assumptions of distribution and uniformity of residuals using residuals plots. A type III analysis of variance was performed on all models to assess the significance of main effects and interaction terms (Fox 2015, Fox et al. 2019). When interactions with exclosure treatment were significant, we conducted a post-hoc analysis of differences between the marginal means of exclosure treatments across levels of the interacting factor while controlling for multiple comparisons using the Tukey method (Searle et al. 1980, Lenth et al. 2020).

All analyses were performed using the R programming software (R Core Team 2019). Small mammal abundances were estimated using the *F.huggins.estim* function of

the *mra* package (McDonald 2018). GLMMs were fit using the *glmmTMB* function from the *glmmTMB* package (Brooks et al. 2017), and model comparison was performed using the *model.sel* function from the *MuMIn* package (Barton and Barton 2019). Residual plots were produced using the *simulateResiduals* function of the *DHARMa* package (Hartig 2020), and ANOVAs were conducted using the *Anova* function of the *car* package (Fox et al. 2019). Posthoc analyses were performed using the *emmeans* and *pairs* functions of the *emmeans* package (Lenth et al. 2020) and the *glht* function of the *multcomp* package (Hothorn et al. 2008).

RESULTS

Mammalian predator abundance

There was a significant effect of exclosure treatment on predator track counts per per observation night ($\chi^2 = 15.37$, df = 1, $p < 0.001$, Figure 4.3, Table 4.3). On average, 88.8% fewer tracks were found in exclosures compared to controls.

Small mammal abundance, size, and sex ratio

We failed to detect any effect of the exclosure treatment or any interactions on estimated cotton rat abundance, though the effect of burn treatment was marginally significant ($\chi^2 = 3.750$, $p = 0.053$; Table 4.4). We did, however, find a significant interaction between season and exclosure treatment on cotton mouse abundance ($\chi^2 = 18.43$, df = 3, $p < 0.001$; Figure 4.4, Table 4.5), driven by a higher abundance in control plots than treatments plots in the spring ($t = 2.931$, $p = 0.005$), but no difference in fall ($t = -1.385$, $p = 0.175$), winter ($t = -1.690$, $p = 0.100$), or summer (0.957, $p = 0.345$). There

was not a significant effect of exclosure treatment or any of its interactions on cotton rat or cotton mouse sex ratio (Table 4.6, Table 4.7) or on cotton mouse body mass (Table 4.8). But we did find a significant effect of exclosure treatment ($\chi^2 = 22.10$, $p < 0.001$) and its interaction with season ($\chi^2 = 19.33$, $p < 0.001$) on cotton rat mass (Table 4.9, Figure 4.5). This pattern was driven by significantly higher body masses in exclosure plots than in controls in the fall ($t = -3.31$, $p < 0.001$), while there was no difference in winter ($t = -0.096$, $p = 0.924$), spring ($t = 1.60$, $p = 0.11$), or summer ($t = 0.998$, $p = 0.319$).

Cotton rat parasites

The effect of the exclosure treatment on the abundance of parasites in cotton rats depended on season. Three parasite taxa (Strongyle 1, Strongyle 2, and Spirurid) were observed infecting cotton rats in the terrestrial predator exclusion experiment (Figure 4.2, Table 4.1). For two of these parasite taxa (Strongyle 1 and 2), we failed to detect a significant effect of the exclosure treatment (Strongyle 1 (zero-inflated negative binomial model): $\chi^2 = 0.1286$, df = 1, $p = 0.720$; Strongyle 2 (zero-inflated negative binomial model): $\chi^2 = 0.0219$, df = 1, $p = 0.882$) or its interactions on parasite abundance (see Table 4.2). However, the abundance of both parasites varied significantly with capture season (Strongyle 1: $\chi^2 = 20.99$, df = 3, $p < 0.001$; Strongyle 2: $\chi^2 = 24.19$, df = 3, $p < 0.001$). Strongyle 1 abundance was generally highest in summer and winter, while Strongyle 2 abundance was generally highest in fall and spring. The interaction between exclosure treatment and season had a clear effect on the third parasite, the Spirurid. In this case, capture season (negative binomial model: $\chi^2 = 52.65$, df = 3, $p < 0.001$), and the

interaction between exclosure treatment and capture season (χ^2 = 22.10, df = 3, p < 0.001) were both significant predictors of abundance, while exclosure treatment alone was not (χ^2 = 1.880, df = 1, p = 0.170). Specifically, Spirurid abundance differed between treatment types in winter (t = -3.307, p = 0.001), but not in spring (t = 1.308, p = 0.191), summer (t = 1.521, p = 0.129), or fall (t = -1.808, p = 0.071; Figure 4.1A). Overall, the effects of predator exclusion occurred in the Spirurid exclusively during the winter, when predator exclusion increased parasite abundances.

Cotton mouse parasites

For cotton mice the effects of exclosure treatment on parasite abundance depended either on capture season or burn treatment. Two parasite morphotypes (Strongyle 1 and Strongyle 2) were observed to commonly infect cotton mice in the terrestrial meso-predator exclusion experiment (Figure 4.2, Table 4.1). For Strongyle 1, both capture season (zero inflated negative binomial model: χ^2 = 11.10, df = 3, p = 0.011) and the interaction between exclosure treatment and capture season (χ^2 = 9.349, df = 3, p = 0.025) were significant predictors of abundance, while exclosure treatment alone was not (χ^2 = 0.8595, df = 1, p = 0.354, Table 4.2). The effect of the interaction was such that Strongyle 1 abundance differed between treatment types in winter (t = 3.062, p = 0.002), when predator exclusion decreased parasite abundance, but not in fall (t = -1.026, p = 0.305), spring (t = 1.684, p = 0.093), or summer (t = 1.522, p = 0.129; Figure 4.1b). For Strongyle 2, the effect of predator exclusion on parasite abundance varied both seasonally and between burn treatments. The main effect of burn year (zero-inflated negative binomial model: χ^2 = 31.14, df = 1, p < 0.001), the interaction between exclosure

treatment and burn (χ^2 = 6.973, df = 1, p = 0.008), and the interaction between exclosure treatment and capture season (χ^2 = 15.84, df = 3, p = 0.001) were all significant predictors of Strongyle 2 abundance, while exclosure treatment alone was not (χ^2 = 1.428, df = 1, p = 0.232). With respect to the season by exclosure treatment interaction, Strongyle 2 abundance differed between exclosure treatment types in fall (t = -3.082, p = 0.002) and winter (t = 2.167, p = 0.031), but not in spring (t=-0.902, p = 0.367) or summer (t = -0.02, p = 0.984; Figure 4.1c). Interestingly, predator exclusion increased Strongyle 2 abundance in fall, while in winter predator exclusion decreased Strongyle 2 abundance. For the burn by treatment interaction, Strongyle 2 abundance differed between treatment types in the year without a burn (t = -2.071, p = 0.0387), increasing when predators are excluded. There was no effect in the burn year (t = 1.376, p = 0.169; Figure 4.1d).

DISCUSSION

We tested for effects of seasonality, disturbance, and variation in prey species identity on the predictions of the healthy herds hypothesis. The HHH posits that, because predators remove infected prey from a population and control prey population density, the loss of predators should increase parasitism in prey. However, we found, as predicted, that mammalian predator exclusion had variable effects on parasitism in prey. First, we found that both the strength and direction of the effect of predator exclusion on small mammal parasites varied seasonally, with effects concentrated in the fall and winter months. Second, we found that fire had a large influence on the size of the effect of predators on parasites in cotton mice. Finally, we found that the direction of the effect of predator exclusion varied with host species identity. These findings suggest that

taxonomic and seasonal variation in predator-prey interactions shape how predation affects parasitism in prey.

Seasonality influenced the effect of predators on parasite abundance in our experiment. Terrestrial mammal predator exclusion affected parasite abundances in cotton rats and cotton mice in the fall and winter. The importance of the fall-winter seasons in our study system may be explained by seasonality in the predator, the prey, or the predator-prey interaction itself. First, the focal predators that were excluded may vary seasonally in their abundance or behavior, making their exclusion most apparent during seasons when they would otherwise be present or active. However, based on track surveys, we saw no sign of seasonal variation in mammal predator presence, and prior studies found that winter months had the lowest proportional mammalian predation pressure (Conner et al. 2011). Second, the vegetation phenology and structure of our system varies seasonally. This variation almost certainly alters the interactions between predator and prey species. Notably, changes in vegetative cover and food availability alter rodent risk-taking behavior during foraging as well as predator foraging preferences (Schooley et al. 1996, Cherry et al. 2016) but the data collected in this study do not allow us to effectively test this mechanism.

Third, it is possible that seasonal changes in unmanipulated predator abundance magnified the effects of our predator manipulations during certain periods. For example, in addition to meso-mammals and raptors, snakes are well-known predators of rodents (Gibbons and Dorcas 2005, Conner et al. 2011) whose abundance was not influenced by our exclosure treatments (Howze et al. Unpublished Data, Conner et al. 2011). However, in much of the southeastern United States snakes enter a seasonal torpor during colder

months, eliminating the predation pressure on focal prey species during this period (Gibbons and Dorcas 2005, Conner et al. 2011). Thus, the loss of snake predation during the fall and winter may have magnified the importance of meso-mammal and avian predation for controlling both prey and parasite populations. We found limited evidence in support of this mechanism in the effect of predator exclusion on cotton rat body mass in the fall as snake predation begins to decline (Conner et al. 2011). When mammalian predators are excluded and snakes are becoming dormant, we found that average cotton rat body mass in exclosures increases without changes in density, possibly due to the increased importance of avian predators who prefer smaller prey items (Dickman et al. 1991, Koivunen et al. 1996). This pattern occurs just before the winter months when we see a strong, positive effect of exclusions on parasitism by Spirurids, a parasite that is aggregated in larger individuals in our system and therefore should be more abundant in a population with more larger rats. Because predator effects on cotton rat body mass align temporally with effects on parasitism, we suggest that the effects of predators on parasites in cotton rats are due primarily to these consumptive interactions. We also found seasonal variation in the consumptive effects of predators on cotton mice in the form of an effect of exclosure treatment on cotton mouse abundance. However, this effect on cotton mouse abundance was driven by differences in the spring while effects of predators on cotton mouse parasites were concentrated in fall and winter months. Because the consumptive effects that we detected on cotton mouse abundance do not align temporally with the effect of predator exclusion on parasites, the seasonal effects of predators on parasites in cotton mice are unlikely to be consumptive in nature. Therefore, we have evidence of the seasonality of PPP interactions being due to seasonality in the consumptive effects of

predators in cotton rats, but not in cotton mice. A key conclusion of our study is that seasonal variation is central to shaping PPP interactions and short-term experiments that fail to consider seasonality are inadequate for drawing inferences about the HHH.

Periodic disturbances, such as prescribed fire, alter predator-prey interactions. We found that predator exclusion only affected the abundance of a single cotton mouse parasite in the year without a burn. In this "non-burn" year we observed a classic healthy herds effect, with significantly higher parasite abundance in exclosure plots without predators. Burning drastically alters the ground cover and plant community (Glitzenstein et al. 1995, Brockway and Lewis 1997) in ways that influence both predator and prey behavior (Fordyce et al. 2015, Lyons et al. 2015). In our system, fire has strong short-term effects on small mammal survivorship and mortality due to predation (Conner et al. 2011, Morris et al. 2011c, 2011b). Of particular relevance to our finding, the effect of predator exclusion on cotton mouse survival varies with recent fire history, with increased survival in exclosures after the burn (Morris et al. 2011c). It therefore follows that recent fire history should also influence PPP interactions in cotton mice. Interestingly, while we would expect predator effects to be concentrated in burn years, as this is when rodents are typically exposed to additional predation, we instead found them in non-burn years. Over the time period of our study, we did not detect an effect of fire on the differences between exclosure treatments in cotton mouse abundance, average mass, or sex ratio. This lack of alignment between the consumptive effects of predators on cotton mice prey compared to effects on their parasites again supports the potential importance of non-consumptive effects of predators on parasites of cotton mice. Nonetheless, the effects of large-scale disturbances such as fire on predator-prey

interactions are well-documented (Torre and Díaz 2004, Lyons et al. 2015, Leahy et al. 2016). Our results highlight the important role fire also plays in the ecology of the parasites associated with predator-prey systems subject to this type of disturbance.

The effect of mammalian predator exclusion varied by prey species, but the drivers of this difference require further consideration. While exclusion of mesopredators increased parasite abundance in cotton rats during winter, in cotton mice the abundance of parasites decreased under the same conditions. The cotton mouse result is the opposite of what we expected based on the HHH (Packer et al. 2003). Moreover, the opposing pattern in cotton rats vs. mice suggests an important difference in the predation pressure experienced by cotton rats and cotton mice during the winter. One possible explanation is that the contrasting results in cotton rats and mice stem from differences in life-history timing of the two species, such that they respond to predators differently over the winter. Predators can have non-consumptive effects on the behavior and physiology of their prey (Schmitz et al. 2004, Preisser et al. 2007), which can in turn influence parasitism (Raffel et al. 2010, Bertram et al. 2013). For example, trinidadian guppies experiencing elevated predation shoal together more closely, increasing parasite transmission (Stephenson et al. 2015). Since seasonal cycles of reproduction and other behaviors can influence predator response behavior (Festa-Bianchet 1988), the difference between cotton rats and mice in their parasite responses to predator exclusion may stem from differential responses to the lack of terrestrial mesopredators during the winter. As discussed above, for cotton rats, we have strong evidence of seasonal variation in consumptive effects on prey that lines up with effects on parasites, while for cotton mice we found no such alignment. Discordance in the outcome of PPP interactions between the two prey species provides

additional support for a difference in the mechanisms of predator effects on parasites, i.e. predator effects on parasites are primarily consumptive in cotton rats and primarily non-consumptive in cotton mice

Overall, our exclusion experiment highlights the importance of species identity, seasonality, and disturbance in shaping the effect of predators on parasites in their prey. Given these findings, we propose that future investigations of the HHH consider the role of these factors explicitly to improve the accuracy of study predictions. While it may be daunting to consider the large number of possible drivers of tri-trophic PPP interactions, a path forward has already been paved by research on other three-species interactions, such as competition (Gurevitch et al. 1992, 2000) and trophic cascades (Shurin et al. 2002, Schmitz et al. 2004). To draw meaningful and generalizable conclusions about the role of different heterogeneities in shaping PPP interactions, future studies should move beyond the paradigm of measuring parasitism in a single host with or without a single predator over a short period of time.

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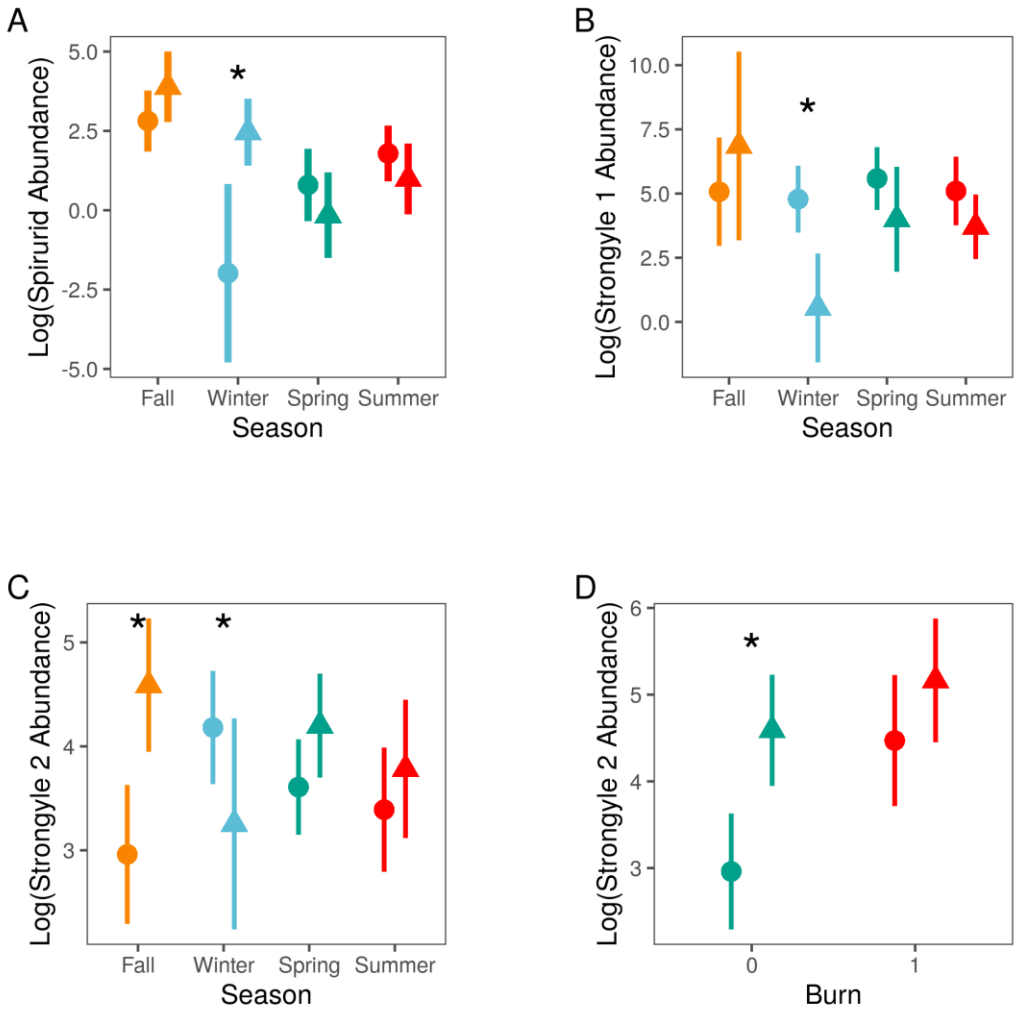

Figure 4.1 Mean parasite eggs per gram of feces in exclosure (triangle) and control (circle) plots of our terrestrial mesopredator exclusion experiment for (a) Spirurid in cotton rats, (b) Strongyle 1 in cotton mice and (c,d) Strongyle 2 in cotton mice, separated by season (a-c) or burn treatment history (d). Points are presented on a natural log scale. Error bars represent 95% confidence intervals.

Figure 4.2 Parasite morphotypes analyzed include two strongyle nematodes in cotton rats and cotton mice (a) 1 and (b) 2, and (c) one spirurid nematode in cotton rats.

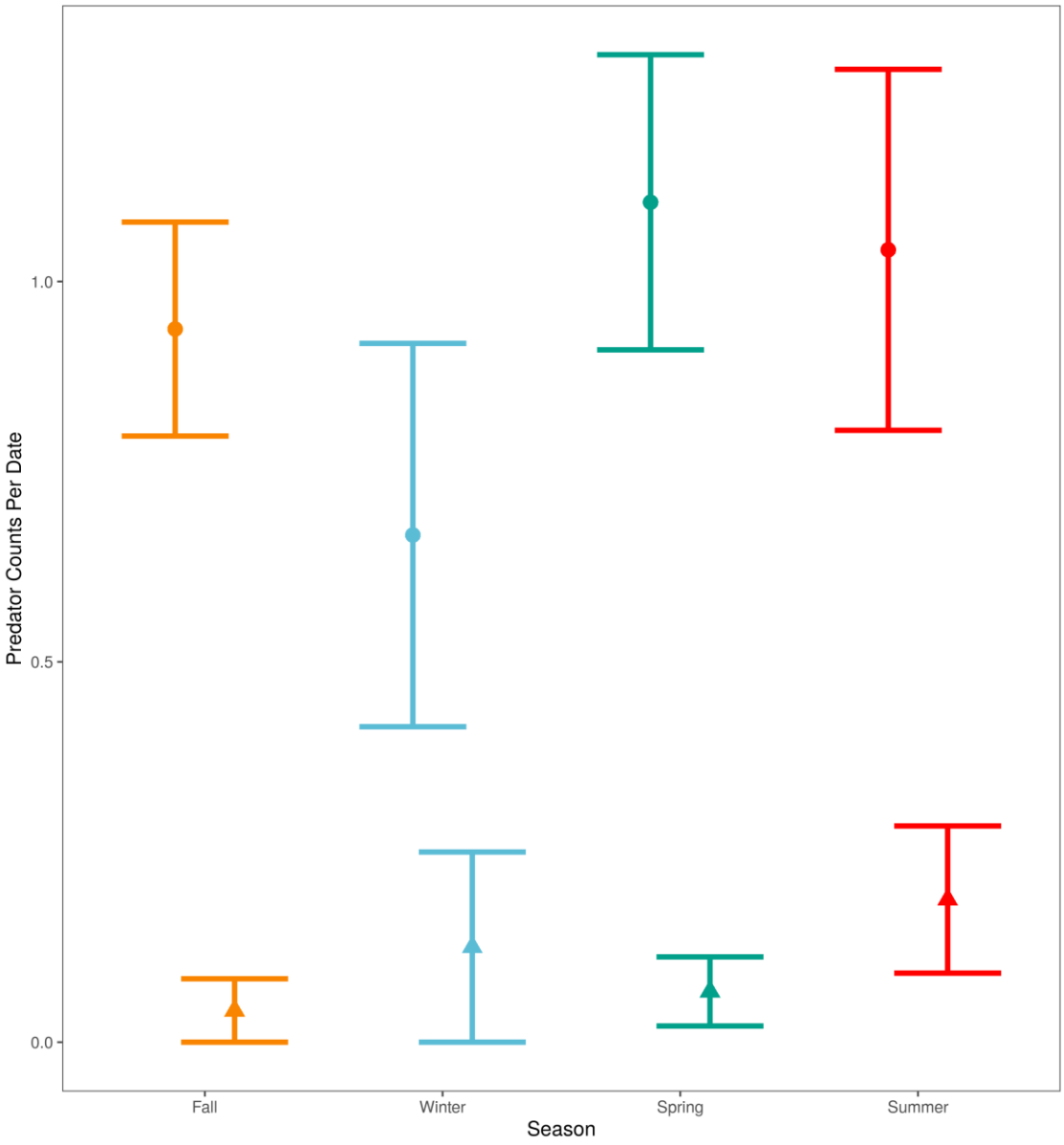

Figure 4.3 Mean number of mesopredator track counts in exclosure (triangle) and control (circle) plots of our terrestrial mesopredator exclusion experiment. Error bars represent 95% confidence intervals.

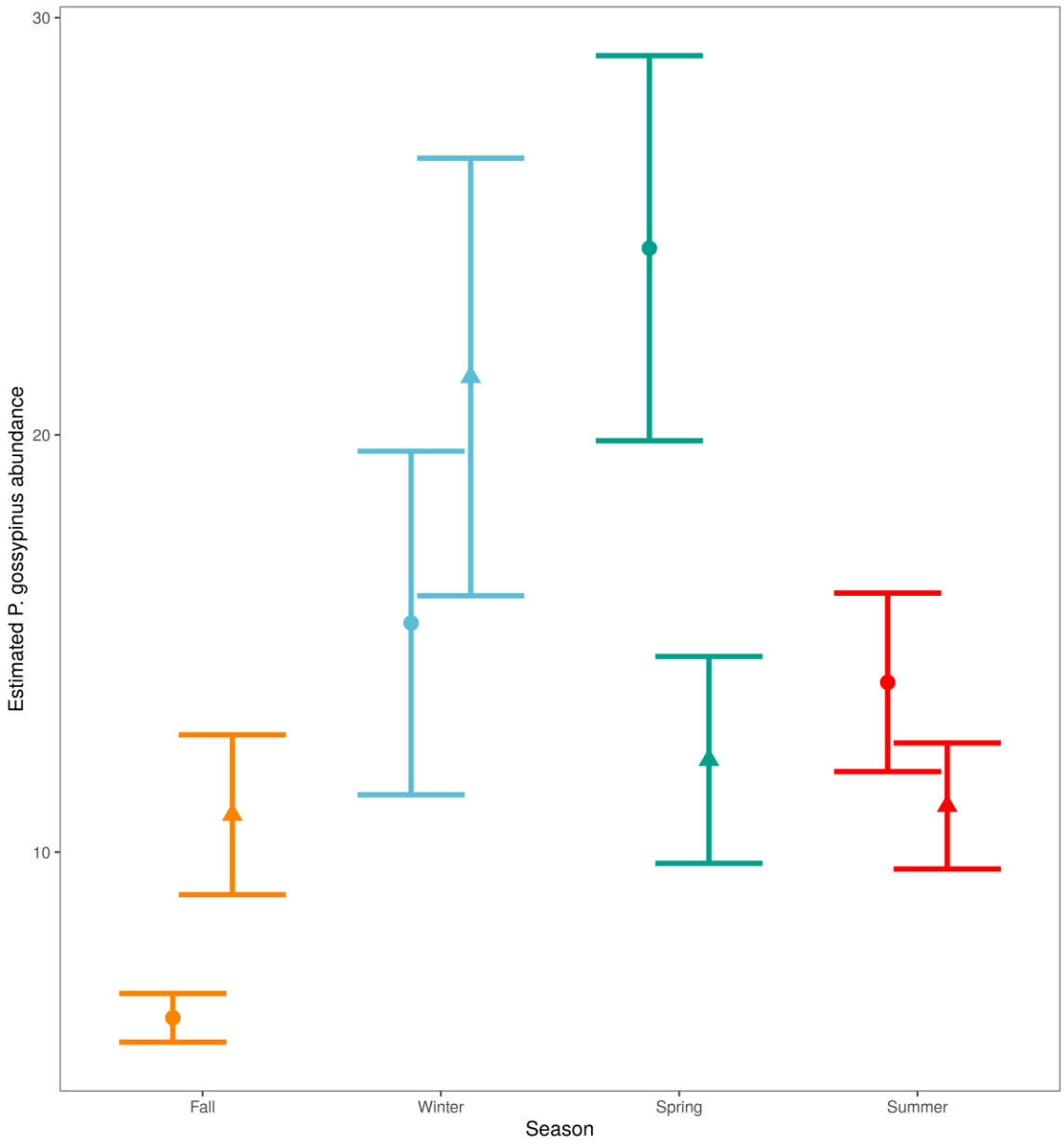

Figure 4.4 Mean estimated cotton mouse (*P. gossypinus*) abundance in exclosure (triangle) and control (circle) plots of our terrestrial mesopredator exclusion experiment. Error bars represent 95% confidence intervals.

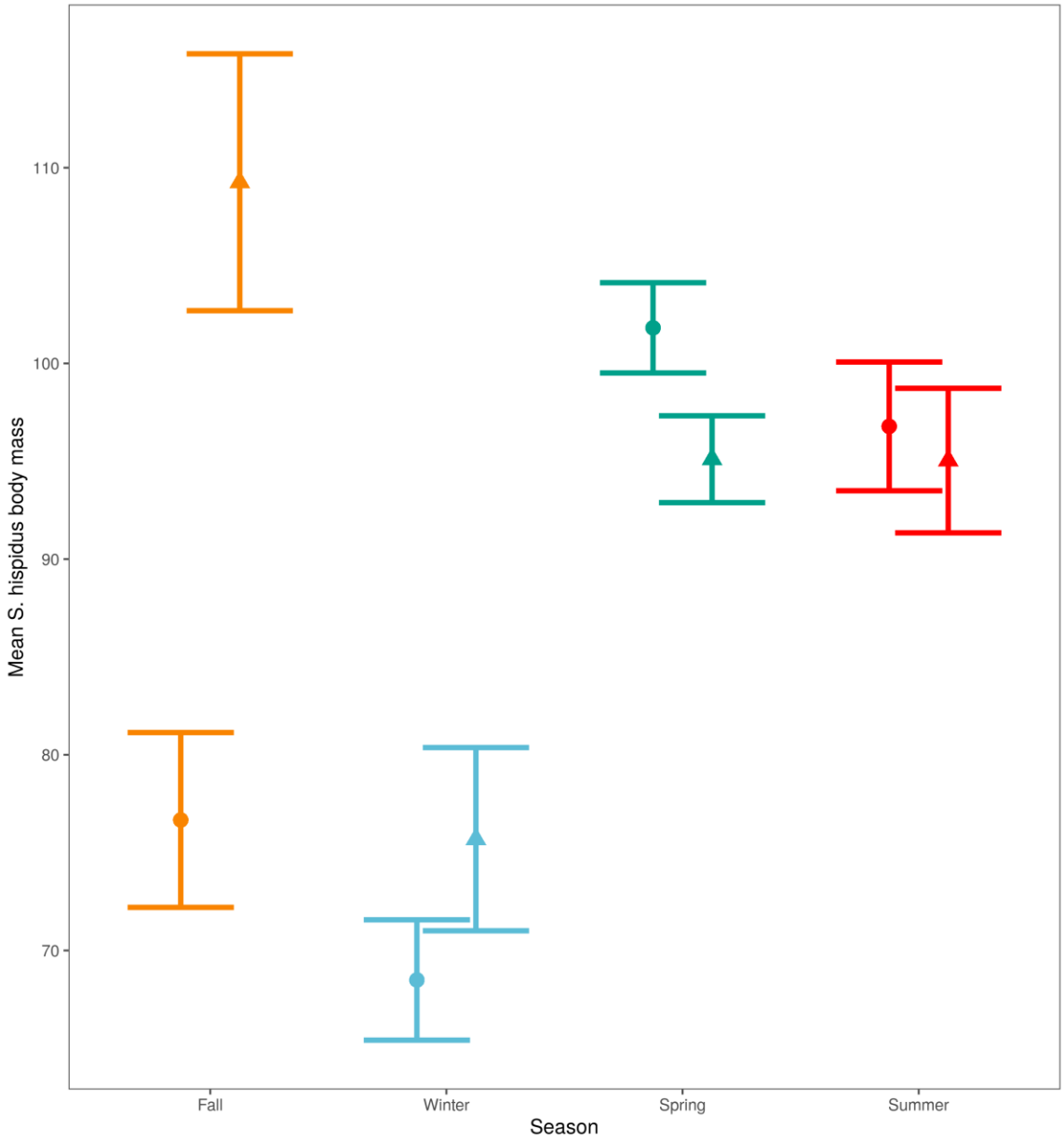

Figure 4.5 Mean estimated cotton rat (*S. hispidus*) abundance in exclosure (triangle) and control (circle) plots of our terrestrial mesopredator exclusion experiment. Error bars represent 95% confidence intervals.

Table 4.1 Prevalence and average abundance (in eggs per gram of feces) of parasites of cotton rats and cotton mice in our mammalian exclusion experiment. Standard error of prevalence is calculated by grid-season.

Parasite	Prevalence (SE)	Abundance [epg] (SE)
Cotton Rats (N = 1049)		
Strongyle 1	0.722 (0.070)	253.06 (28.62)
Strongyle 2	0.624 (0.38)	59.04 (15.18)
Spirurid	0.138 (0.029)	29.98 (10.20)
Cotton Mice (N = 646)		
Strongyle 1	0.148 (0.090)	27.64 (10.19)
Stronyle 2	0.566 (0.087)	111.34 (23.68)

Table 4.2 Full results of generalized linear models for parasite abundance in cotton rats and cotton mice, during our mammalian predator exclusion experiment. Statistics show the chi-squared value, degrees of freedom and (p-value). Significant effects are displayed in bold.

	Cotton Rats			Cotton Mice	
Variable	Strongyle 1	Strongyle 2	Spirurid	Stronygle 1	Strongyle 2
Sex	$\mathbf{14.03_1(0.0001)}$	$\mathbf{7.409_1(0.007)}$	$0.1688_1(0.681)$	$0.4125_1(0.521)$	$\mathbf{6.507_1(0.011)}$
Predator Treatment	$0.1286_1(.720)$	$0.0219_1(0.882)$	$1.880_1(0.170)$	$0.8595_1(0.354)$	$1.428_1(0.232)$
Body Mass	$\mathbf{11.68_1(0.0006)}$	$\mathbf{45.224_1(<0.0001)}$	$\mathbf{36.376_1(<0.0001)}$	$0.0778_1(0.780)$	$2.101_1(0.147)$
Season	$\mathbf{20.99_3(<0.0001)}$	$\mathbf{24.193_3(<0.0001)}$	$\mathbf{52.650_3(<0.0001)}$	$\mathbf{11.097_3(0.011)}$	$2.596_3(0.458)$
Burn Treatment	$0.6314_1(0.427)$	$\mathbf{3.878_1(0.049)}$	$1.382_1(0.240)$	$1.754_1(0.185)$	$\mathbf{31.139_1(<0.0001)}$
Predator Treatment*Sex	$2.401_1(0.121)$	$0.2007_1(0.654)$	$0.4771_1(0.490)$	$2.413_1(0.120)$	$0.2385_1(0.625)$
Predator Treatment*Body Mass	$0.8311_1(0.362)$	$0.8574_1(0.355)$	$0.1614_1(0.688)$	$1.321_1(0.251)$	$1.411_1(0.235)$
Predator Treatment*Season	$5.164_3(0.160)$	$6.894_3(0.075)$	$\mathbf{22.095_3(<0.0001)}$	$\mathbf{9.349_3(0.025)}$	$\mathbf{15.835_3(0.001)}$
Predator Treatment*Burn Treatment	$0.1843_1(0.668)$	$0.0851_1(0.771)$	$0.0537_1(0.817)$	$0.879_1(0.349)$	$\mathbf{6.973_1(0.008)}$

Table 4.3 Model results for predator track counts per observation night.

	Chisq	Df	p
(Intercept)	30.5146	1	**<0.001**
Season	6.3173	3	0.0972
Predator Treatment	14.9666	1	**0.0001**
Burn Treatment	0.3918	1	0.5314
Season:Predator Treatment	3.6977	3	0.296
Predator Treatment:Burn Treatment	0.6171	1	0.4321

Table 4.4 Model results for estimated cotton rat population size.

	Chisq	Df	p
(Intercept)	73.2819	1	**<0.001**
Season	1.1474	3	0.7656
Predator Treatment	0.0440	1	0.8339
Burn Treatment	3.7495	1	0.0528
Season:Predator Treatment	0.8225	3	0.8441
Predator Treatment:Burn Treatment	2.3396	1	0.1261

Table 4.5 Model results for estimated cotton mouse population size

	Chisq	Df	p
(Intercept)	59.3964	1	**<0.001**
Season	30.4627	3	**<0.001**
Predator Treatment	0.8034	1	0.3701
Burn Treatment	0.1061	1	0.7446
Season:Predator Treatment	18.4295	3	**0.0004**
Predator Treatment:Burn Treatment	1.0396	1	0.3079

Table 4.6 Model results for cotton rat sex ratio

	Chisq	Df	p
(Intercept)	2.8497	1	0.0914
Predator Treatment	0.2464	1	0.6196
Season	8.5800	3	**0.0354**
Burn Treatment	5.6430	1	**0.0175**
Predator Treatment:Season	6.4812	3	0.0904
Predator Treatment:Burn Treatment	0.0879	1	0.7669

Table 4.7 Model results for cotton mouse sex ratio

	Chisq	Df	p
(Intercept)	2.4642	1	0.1165
Predator Treatment	1.1366	1	0.2864
Season	3.1006	3	0.3764
Burn Treatment	2.0575	1	0.1515
Predator Treatment:Season	2.0447	3	0.5632
Predator Treatment:Burn Treatment	1.0126	1	0.3143

Table 4.8 Model results for cotton mouse body mass

	Chisq	Df	p
(Intercept)	823.7635	1	**<0.001**
Sex	2.5996	1	0.1069
Predator Treatment	0.1247	1	0.724
Season	47.3332	3	**<0.001**
Burn Treatment	20.5275	1	**<0.001**
Sex:Predator Treatment	0.0000	1	0.9973
Predator Treatment:Season	1.6118	3	0.6567
Predator Treatment:Burn Treatment	1.4977	1	0.221

Table 4.9 Model results for cotton rat body mass

	Chisq	Df	p
(Intercept)	229.5963	1	**<0.001**
Sex	4.8590	1	**0.0275**
Predator Treatment	22.0948	1	**<0.001**
Season	48.8227	3	**<0.001**
Burn Treatment	5.7225	1	**0.0167**
Sex:Predator Treatment	5.1071	1	**0.0238**
Predator Treatment:Season	19.3327	3	**0.0002**
Predator Treatment:Burn Treatment	0.9574	1	0.3279

CHAPTER 5

TRANSMISSION DOMINANCE AND THE SPATIAL DISTRIBUTION OF PARASITES IN MULTI-HOST COMMUNITIES[4]

[4] Richards R.L., Archie E.A., Drake J.M., Ezenwa V.O. To be submitted to *Journal of Animal Ecology*.

ABSTRACT

1. Many parasites are shared across a community of host species, but not all host species contribute equally to parasite transmission. These heterogeneities in transmission can have significant effects on disease dynamics. Particularly, it is not clear to what extent established relationships between community composition and parasite transmission may be complicated by heterogeneity in host and non-host species space use patterns.
2. Here we use a parasite sharing ungulate community to ask (i) whether considering host space use patterns produces spatial heterogeneity in transmission, and (ii) whether non-host predator space use intensity influences parasite community-level transmission potential by affecting host space use.
3. Species-level parasite transmission was measured using field collected fecal samples of wild ungulates containing gastrointestinal nematode eggs. We estimated host species space use patterns with species distribution models.
4. First, we found that parasites with multiple host species contributing substantially to transmission also have significantly more spatial variation in the identity of transmission dominant hosts due to space use. Second, those parasites whose transmission was dominated by the American bison (*Bison bison*) tended to increase with predation pressure while the remaining parasites tended to decrease with predation pressure.
5. Broadly we find that both host and non-host predator space use patterns introduce spatial heterogeneity in community-level transmission for some parasites but not others.

INTRODUCTION

Identifying heterogeneities in parasite transmission is key to understanding the dynamics of infectious diseases (Lloyd-Smith et al. 2005, Paull et al. 2012). Just like super-spreaders, defined as individuals that contribute disproportionately to transmission within populations of a single species, super-spreading, or transmission dominant, species often contribute disproportionately to transmission at the community-level when multiple host species share a common parasite, due to species competence and/or abundance (Logiudice 2003, Paull et al. 2012). While there has been much debate over the effects of host species diversity on disease dynamics (Ostfeld and Keesing 2000, Logiudice 2003, Dobson 2004, Lafferty and Wood 2013, Wood et al. 2014), there is broad agreement that differences in species composition can produce transmission hotspots or coldspots that are associated with the commonness of transmission dominant host species (Paull et al. 2012, Rohr et al. 2020).

Even species that are not viable hosts for a parasite can influence community-level parasite transmission via effects on host community composition. For example, the presence of competitors and predators can limit the abundance of otherwise transmission dominant hosts, depressing community-wide parasite transmission (Packer et al. 2003, Keesing et al. 2006). Predators can also alter the behavior of their prey (Schmitz et al. 2004, Preisser et al. 2007), which can in turn influence parasitism via changes in contact rates between individuals (Raffel et al. 2010, Bertram et al. 2013). For example, Trinidadian guppies experiencing elevated predation shoal together more closely, increasing parasite transmission (Stephenson et al. 2015). Areas with high levels of

predator use intensity are expected to have lower intensity use by prey species, because of behavioral avoidance(Laundré et al. 2001, Ripple and Beschta 2012, Weinstein et al. 2018). This is one key way in which predation might influence community-level parasite transmission, potentially creating hotspots and coldspots of transmission that are driven by heterogeneities in host species space use rather than by heterogeneities in host abundance alone.

Heterogeneity in space use introduces a new way in which host community composition may translate to variation in parasite transmission. While the abundance and competence of different host species are typically used to measure the importance of each species to community level-transmission (Keesing et al. 2006, Streicker et al. 2013, Johnson et al. 2013), when host species vary in their space use patterns then species local space use intensity, rather than abundance, may dictate patterns of transmission (Paull et al. 2012, Barasona et al. 2014, Woodroffe et al. 2016). The concept that host behavior can influence community-level parasite transmission is well established, including the consideration of how host diversity influences community-level contact networks, but rarely measured (Keesing et al. 2010). In the case of space use, much existing work suggests that transmission dominant hosts tend to be abundant even in species poor communities and therefore both regionally and locally important to transmission (LoGiudice et al. 2008, Johnson et al. 2013), but this pattern may be disrupted by variation among species in space use patterns. Therefore, we hypothesize that space use heterogeneity among host species creates locations across a landscape where the relative contributions to transmission by host species substantially differ from predictions based on abundance and competence alone. For example, if bison (*Bison bison*) are the primary

transmission dominant host for a parasite in a host community due to competence and abundance, our knowledge of host space use patterns instead suggests that in riparian areas, which bison avoid (McCullough 1981), bison may be fairly unimportant to community-level transmission while other ungulates which use these areas more intensely, such as white-tailed deer (McCullough 1981), may be more important. It is therefore necessary to examine whether the relative contributions of host species to transmission and the identities of transmission dominant host species vary spatially due to heterogeneity in space use across the host community.

Here we ask how consideration of space use heterogeneity improves our understanding of community-level parasite transmission in a community of six sympatric ungulate species at the National Bison Range, Montana, USA. Ungulates notoriously vary in infection and shedding rates of shared gastrointestinal nematode parasites (Hoberg et al. 2001, Archie and Ezenwa 2011), space use patterns (McCullough 1981, Schwartz and Ellis 1981, Johnson et al. 2000), and their response to predation pressure (Laundré et al. 2001, Hernández and Laundré 2005, Kittle et al. 2008). These three attributes and their ease of measurement in large, mobile, mammals, make this system particularly amenable to addressing the effects of space-use on community-level parasite transmission. Using this system as a model we examined (i) whether considering host space use intensity patterns produces spatial heterogeneity in transmission dominance, and (ii) whether predator space use intensity influences parasite community transmission potential by affecting host space use. To address these questions, we identified transmission dominance patterns for multiple parasite species shared across a community of ungulate hosts and then modeled host space use using host occurrences and a suite of

environmental predictors including predator presence. We predicted, first, that (i) the species most responsible for transmission would vary spatially due to differing patterns of host space use. We expected to see this variation particularly for parasites with multiple species that contribute substantially to transmission because less extreme differences in space use intensity patterns could still disrupt patterns of transmission dominance. Second, we predicted that (ii) areas with high predator space use intensity would have lower parasite community transmission potential as hosts are likely to avoid these areas.

METHODS

Study System

Ungulate species inhabiting the National Bison Range (NBR), Moiese, Montana, USA include a semi-managed population of approximately 400 American bison (*Bison bison*) and unmanaged populations of bighorn sheep (*Ovis canadensis*), elk (*Cervus canadensis*), mule deer (*Odocoileus hemionus*), white-tailed deer (*Odocoileus virginianus*), and pronghorn antelope (*Antilocapra americana*). The southern portions of the range are characterized by steep, rocky canyons, while the northern and eastern areas are more gently sloping palouse prairie with riparian areas along the northern and southern boundaries (McCullough 1981). The six species differ in their patterns of habitat use at the NBR (McCullough 1981). Pronghorn antelope and white-tailed deer tend to be found in the northern and eastern low elevation grasslands, with white-tailed deer showing a strong preference for the riparian woodlands at the very northern edge of the range. Mule deer are associated with the steeper, rocky southern reaches of the range. Elk also frequent these rocky southern areas but are more broadly distributed at high

elevations and near timber patches. Bison use the range widely but are rotated through eight fenced areas, while the other ungulates are able to cross the fences and move outside of the range. These six ungulate species are known to share a broad range of gastrointestinal nematode parasites, many of which are transmitted via a fecal-oral route, where an infected host sheds parasite eggs or larvae into the environment and a susceptible host becomes infected by ingesting the propagules during grazing (Hoberg et al. 2001, Archie and Ezenwa 2011). A wide range of predatory carnivores are active around and on the NBR including wolves (*Canis lupus*), coyotes (*Canis latrans*), Canada lynx (*Lynx canadensis*), bobcats, (*Lynx rufus*), mountain lions (*Puma concolor*), black bears (*Ursus americanus*), and brown bears (*Ursus arctos*) (Montana Natural Heritage Program. 2020).

Estimating Parasite Shedding Rate Across Hosts

To estimate the mean nematode shedding rate of each host species, we collected fecal samples from all ungulate host species between July and December 2007. When possible, samples were collected from animals directly observed defecating. Otherwise, when no defecation was observed we searched areas vacated by animals for fresh fecal piles, with piles separated by >2m considered independent samples. After collection, all samples were stored at 4°C until processing. To estimate the average parasite shedding, 1 gram of feces from each sample was processed using sugar flotation with a 1.27 specific gravity sugar solution and a Fecal Egg Count (FEC) was performed (Foreyt 2013), and then we estimated the mean FEC for each host species (n = 15-35 fecal samples per species). The FEC provides a measure of parasite shedding rate per gram of feces

defecated, but since all species do not defecate at the same rate we rescaled the average shedding rate using the well-established ¾ scaling of Basal Metabolic Rate (BMR) with body mass (defecation rate $\propto$ body mass$^{3/4}$) (Genoud et al. 2018). Mean body mass values for each species were taken from the Pantheria database of mammalian species traits (Jones et al. 2009). This combination of FECs with BMR transformation provides a relative measure of an average host individual's shedding rate of parasite eggs.

Estimating Nematode Species Prevalence

We used DNA barcoding to quantify the prevalence of eighteen nematode species in each host species and then estimate the average proportion of eggs shed that belong to each nematode species. First, we used larval culture to isolate nematode larvae. We placed 10-20g of feces in sealed 30ml culture jars at room temperature. After 10 days we filled the jars with deionized water, inverted them on a Petri dish, and let them sit for ~24 hours. Larvae migrated into the clean water of the Petri dish and were collected in a 200µl sample of water. Larval suspensions were stored at 4°C and processed for DNA extraction within 4 days. DNA was extracted from individual larvae as described in (Archie and Ezenwa 2011). We then amplified and Sanger sequenced a ~250bp of the ITS2 rRNA region to identify larvae to the species-level (Archie and Ezenwa 2011). Up to 33 larvae from 22-32 individuals of each host species were used. We estimated the average proportion of parasite eggs shed by a host species that belonged to each parasite species as the mean proportion of sequenced worms that belonged to each species in host fecal samples.

Estimating Host Transmission Dominance

We define transmission dominance as the contribution to transmission of a population of a species relative to the populations of other host species. It is common to conceptually identify key hosts as those that are responsible for the largest portion of the basic reproductive number of a parasite, R_0 (e.g. Rudge et al. 2013, Fenton et al. 2015). It is often difficult, however, to collect sufficient data to quantify inter- and intra-species transmission (Dobson 2004), making many of the specific parameters of R_0 unestimable. Therefore, by considering transmission dominance we avoid the need to specifically calculate R_0 but assume that transmission dominance is roughly proportional to a species' proportional contribution to the transmission pool (Marm Kilpatrick et al. 2006, Streicker et al. 2013). For generalist, environmentally transmitted, parasites, such as the GI nematodes we study here, transmission dominance can be measured as proportional to the number of parasite eggs that the population of species *i* (of *N* total host species) sheds over an arbitrary unit of time (Streicker et al. 2013):

$$\pi_i = \frac{p_i * \lambda_i * H_i}{\sum_j^N p_j * \lambda_j * H_j},$$

where p_i is the average proportion of eggs shed that belong to the focal parasite in host species *i* and λ_i the mean shedding rate of an individual of species *i*, both estimated above. H_i is the abundance of host species *i* which is estimated annually by the NBR and we use a single annual estimate.

Estimating Spatial Heterogeneity

The pattern of host species' space use across the NBR was based on a set of observations conducted in June and July of 2006 and 2007. Driving transects were

conducted along roads that span the NBR. When a group of ungulates was sighted, the species and GPS coordinates of the group were recorded. We used these presence data to construct species distribution models for five of our six host species with the Maximum Entropy (MaxEnt) method (Phillips and Dudík 2008, Elith et al. 2011). There were not sufficient presence records for bighorn sheep to construct a reliable species distribution model so this species was excluded from spatial analyses. Briefly, MaxEnt uses environmental covariate data from occurrence records and "background" records, which are representative of the sampled geographic space, to estimate the ratio of $f_1(z)/f(z)$. $f_1(z)$ is the probability density of environments where the species is present and f(z) the probability density of environments on the landscape, both evaluated at the given set of environmental variable values (z). MaxEnt, in particular, estimates $f_1(z)$ in such a way that it minimizes the difference between it and f(z) using Kullback-Liebler divergence (Kullback and Leibler 1951). To limit spatial sampling bias from road-based transects, we used the collection of sightings of all species as background points for all MaxEnt models (Ranc et al. 2017). Since bison are limited in their movement by fences, we limited background points in the bison model to those within enclosures in which bison were also sighted. Prior to model training, presence and background data were randomly partitioned into training (80%) and evaluation (20%) sets. MaxEnt models were tuned through 10-fold cross validation on training data of each species for inclusion or exclusion of feature transformations, including linear (i.e., untransformed), quadratic, product, and hinge features and the value of the regularization parameter, which prevents overfitting by smoothing the model. The regularization parameter was tuned between 1 and 10 with larger values representing stronger smoothing of the model. All tuned models

included all feature transformations with a range of regularization parameters from 1 to 4. MaxEnt models were then trained with tuned parameters on all training data and evaluated with held-out evaluation data using area under the receiver operator curve (AUC), a measure of a model's ability to discriminate between presence and background points (Bradley 1997).

Covariates for species distribution models were collected based on prior study of the NBR and its resident ungulates. In particular, previous studies of space use on the NBR noted the importance of topography and vegetation in predicting ungulate space use (McCullough 1981) and climate and predation pressure have been identified more broadly as important for limiting the ranges of North American ungulates (Krausman and Bleich 2013, Dawe and Boutin 2016). The covariate data we used consisted of 19 Bioclimatic variables describing temperature, precipitation and their variation from the WorldClim 2.0 dataset (Hijmans et al. 2005, Fick and Hijmans 2017), four topographical variables based on the SRTM 90m DEM Digital Elevation Database, canopy cover (Coulston et al. 2012), a suite of soil variables (e.g. organic carbon content, nitrogen conten, bulk density; Ramcharan et al. 2018), and the predicted presence of known predator species of the focal ungulates (Montana Natural Heritage Program. 2020, Figure 5.6, Table 5.2). Although we did not include contemporary measurements of local vegetation type, a combination of soil, climate, and topographic covariates typically describe local vegetative variation in the Western US (McCarley et al. 2020). For analysis, continuous variables were box-cox or log transformed to improve model training on highly skewed distributions (Box and Cox 1964). Models such as MaxEnt are designed to use many potentially relevant covariates, but a large number of collinear

variables can hamper the effectiveness of these models and lead to overfitting (Júnior and Nóbrega 2018). Therefore we use a Ward-clustering approach to diagnose significant collinearity in predictors based on the spearman correlation matrix (Kaufman and Rousseeuw 2009, Dormann et al. 2013). Collinear clusters were reduced to the single most central variable in the cluster (the variable with highest mean correlation to all others) according to Dormann et al. (2013).

To quantify spatial heterogeneity in the transmission dominance for each parasite-host pair, we first generated relative suitability maps for each host species by applying each species model across the range and rescaling so that the values across all grid cells on the NBR summed to 1. Next, we multiplied the transmission potential of a parasite-host pair ($\lambda_i * p_i * H_i$) by the rescaled space use intensity of the respective host for each grid cell on the range. This procedure resulted in a map of grid-cell community-level transmission potential for each parasite by summing the transmission potential maps from each host-parasite pair across a single parasite species.

All data manipulation, modelling and analysis was conducted in the R programming language (R version 3.6.3 (R Core Team 2020)). MaxEnt models were fit using the *maxent* function which interfaces with the MaxEnt java applet and model performance was calculated with the *performance* function, both from the *dismo* package (Hijmans et al. 2017).

Statistical Analyses

To assess the degree to which heterogeneity in host space use produces spatial heterogeneity in transmission dominance for each parasite, we identified the most

transmission dominant host in each grid-cell across the NBR. Next, we measured the spatial variation in which host species is identified as transmission dominant using Simpson's Diversity Index ($1 - \frac{\sum_i^R n_i(n_i-1)}{N(N-1)}$, where there are *R* host species, and n_i is the number of grid cells dominated by host species *i* and *N* represents the total number of grid cells) (Oksanen et al. 2019). In this context, index values range from zero to one and represent the probability that two grid cells chosen at random will have the same transmission dominant host species. Finally, to test the effect of predator pressure on spatial patterns in community transmission, we performed a type 3 ANOVA with community transmission potential in a grid cell as a response variable and parasite identity, mean predator presence probability and their interaction as predictor variables.

RESULTS

Eighteen parasite species were identified across the community of 6 host species. However, only 7 parasite species (*Cooperia oncophora*, *Haemonchus contortus*, *Oesophagostomum venulosum*, *Ostertagia leptospicularis*, *Ostertagia ostertagia*, *Spiculopteragia sp.*, and *Trichostrongylus axei*) were identified in >50% of host species and we confined our further analyses to these parasites. The species composition of the GI parasite community varied among host species as did the mean egg shedding rate across all parasite species (Table 5.3). Mule deer and pronghorn antelope were both hosts to only 6 of the 7 parasites (excluding *Spiculopteragia sp.*) while all other host species shed all 7 shared parasite species. Mule deer shed the most nematode eggs per gram of feces (mean=49.3 epg, sd=18.8) on average, while pronghorn antelope shed the least (mean=31.6 epg, sd=19.6). Bison were the most common transmission dominant host

accounting for the majority of transmission for 4 out of 7 parasites (Figure 5.1). Moreover, the degree of evenness in transmission dominance across the host community varied by parasite, with *H. contortus* showing the most evenness in transmission dominance and *C. oncophora* and *O. ostertagia*, both dominated by bison, showing the least (Figure 5.1).

Heterogeneity in host space use

We detected substantial variation between host species in space use patterns (Figure 5.4). Pronghorn antelope and white-tailed deer (Figure 5.4d-e) tended to preferentially occupy the wooded, riparian, northern and eastern edges of the range while Bison were more often found in the more open, higher elevation, central parts of the range (Figure 5.4a). Elk are predicted to sample the range fairly evenly (Figure 5.4b). Our trained species distribution models varied in their performance on held-out evaluation data (Table 5.1). Elk models performed less well than other models (AUC = 0.705), perhaps due to more indiscriminate use of the range by this species or due to an inappropriate choice of model covariates.

Spatial heterogeneity in host importance

The identity of the transmission dominant host species varied spatially for most parasites (Figure 5.3), so that in large areas of the NBR the host identified as transmission dominant on the basis of abundance and competence was not locally transmission dominant due to low space use intensity. However, parasites (i.e. *Cooperia oncophora*, *Ostertagia ostertagia*) in which transmission is almost entirely dominated by a single host species based on abundance had the least spatial diversity in transmission dominant

host (Simpson's D=0.0006 (*C. oncophora*), 0.0006 (*Ostertagia ostertagia*)). In fact, spatial diversity in the identity of the transmission dominant host was highly correlated with the evenness of abundance-based transmission dominance for that parasite (Pearson's $\rho = 0.843$, $p = 0.017$). Therefore, host space use heterogeneity is most likely to disrupt patterns of transmission dominance in parasites with multiple species that contribute substantially to transmission.

The effect of predator space use intensity on transmission

The association between parasite community transmission potential and predator space use intensity varied by parasite species. We found a significant interaction between parasite species and mean predator presence probability in a grid cell ($F_6 = 37.3$, $p < 0.001$, Figure 5.2b). There were 3 groups of parasites with different responses to mean predation pressure. *C. oncophora, O. Ostertagia,* and *S. sp.* community transmission potential all substantially increased with predator pressure while *T. axei* and *O. leptospicularis* increased more gradually. *O. venulosum* and *H. contortus* both decreased with increasing predator pressure. Heterogeneity in predator space use not only influences community-level transmission potential of parasites, but the effect also varies in direction based on the parasite in question.

DISCUSSION

Differences among host species in their contributions to community-level parasite transmission can have profound effects on disease dynamics (Paull et al. 2012). We asked whether the consideration of space use patterns of species within the community provides

additional insight into patterns of transmission dominance. We considered a single, well studied, host community and asked (i) whether considering host space use intensity patterns produces additional spatial heterogeneity in transmission dominance, and (ii) whether non-host predator space use intensity influences parasite community transmission potential by affecting host space use. First, we identified that some parasites had substantial spatial variation in the species responsible for most transmission while others had so little as to be negligibly different from zero. Second, we found that the effect of non-host predator space use on spatial variation in community-level transmission potential varied by parasite species. These findings suggest that the explicit consideration of species' space use patterns provide important nuance to our understanding of transmission dominance patterns for some parasite species but not others.

We found that direct consideration of host space use caused species' relative contributions to transmission to vary spatially for parasite species without a single clearly dominant host. In general, parasites with more even patterns of transmission across the host community without incorporating space use also had more spatial heterogeneity in transmission dominance due to space use patterns. Prior studies that measured species importance to transmission in multiple locations find both highly conserved transmission dominance patterns (Barbour et al. 2015) and stark differences in transmission dominant host species (Rudge et al. 2013, Gervasi et al. 2015) across space. However, these studies generally rely on differences in population-level abundances of different host species. Our findings show that heterogeneity in space use patterns across the host community can produce additional spatial heterogeneity in transmission dominance and that this pattern

is variable, but predictable, across parasite species. By extension, we would suggest that parasites with multiple host species that contribute substantially to transmission are most in need of explicit study of the effects of host space-use variation.

The relationship between mean predator space use intensity and parasite transmission potential varied by parasite species. In particular we detected 3 clear groups of parasites: (i) strongly increasing with predation, (ii) gradually increasing with predation, (iii) gradually decreasing with predation. Despite predictions that predators would generally decrease parasite transmission in communities of their prey (Packer et al. 2003, Keesing et al. 2006), empirical tests have found substantial variation in both the strength and direction of the effect (Chapter 2). While there are likely multiple mechanisms operating in the complex host-parasite community studied here, we note that the pattern relates to the importance of bison to parasite transmission. Parasites for which bison contributed little to transmission (*O. venulosum*, *H. contortus*) had a negative relationship with predation while the difference between a gradual positive and steep positive relationship was associated with a difference in evenness across the community, particularly how many other host species, in addition to bison, contributed substantially to transmission (Figure 5.1). Bison are not likely targets of predation by any but the largest of the predators in the region (Poelen et al. 2014), and while they do respond to increased abundance of predators such as gray wolves (Laundré et al. 2001), this response does not extend to changes in space use (Hernández and Laundré 2005). So, for parasites not dominated by bison, host avoidance of predators likely decreases parasite transmission in high predator pressure areas. However, for parasites for which bison is important to transmission, their use of high predation areas vacated by other ungulates

can increase transmission. Therefore, the space use of even non-host predator species had substantial effects on spatial patterns of transmission across the host community, though the direction of these effects may vary among parasites based on the behavioral response of transmission dominant host species to predator presence.

One of the main limitations of this study is its small temporal range. Just as we demonstrate that transmission dominance can vary spatially, we would expect similar temporal and spatio-temporal variations as host habitat use and metabolic rates vary from season to season (Moen 1978, Festa-Bianchet 1988, Kissell 1996, Ager et al. 2003). For example, elk have been shown to spend more time in areas with substantial canopy cover in the summer than in the spring (Ager et al. 2003). Therefore, we predict that the spatial patterns of transmission dominance produced by host space use heterogeneity in this study, which were based on summer observations, would differ from patterns based on space use measurements during other parts of the year. As such, we likely underestimate the amount heterogeneity in transmission dominance due to host space use by ignoring the seasonality of host space use patterns.

We also note that our method for estimating the effect of predation pressure on transmission dominance is based not on localized measures of predation or predator density, but rather predicted predator presence from species distribution models. We use this method because of the difficulty of effectively measuring local, spatially explicit, large carnivore presence (Prugh et al. 2019) but it poses two key challenges. First, areas of high predicted predator presence may not reflect the true local predation risk. Second, predicted predator distributions are themselves based on covariates which were included in the prior predator species distribution models (Montana Natural Heritage Program.

2020). As a result, any effects of predators on ungulate space use detected might actually be due to an underlying effect of one of these predator-model covariates. However, despite the fact that we use many of the same covariates in our model as were used to build the predator-models we found very little evidence of correlation between predator presence prediction scores and any of our other covariates. One exception to this rule was that *C. latrans* presence was somewhat correlated with elevation and roughness (Table 5.2). We excluded *C. latrans* presence from this large covariate cluster due to its lack of correlation with most other variables in the cluster and our specific interest in predator pressure. In this case, we actually underestimate the effect of *C. latrans* on spatial transmission dominance. More generally, this problem is not unique to our method of measuring predation pressure. Even if predation pressure were measured directly, predator activity would be driven biologically by underlying factors that could also directly influence host space use.

In conclusion, this study has documented for the first time substantial variation in community transmission due to heterogeneity in both host and non-host space use. This effect of heterogeneity in space-use patterns across the community varied by parasite species. We found that parasites with multiple host species that contribute substantially to transmission were particularly sensitive to host space use patterns and more negatively affected by predator space use. We suggest that future studies of community-level parasite transmission, especially those of parasites with many host species contributing substantially to parasite transmission, consider differences in species' space use patterns, rather than just host competence and abundance, when assessing transmission dominance in host communities.

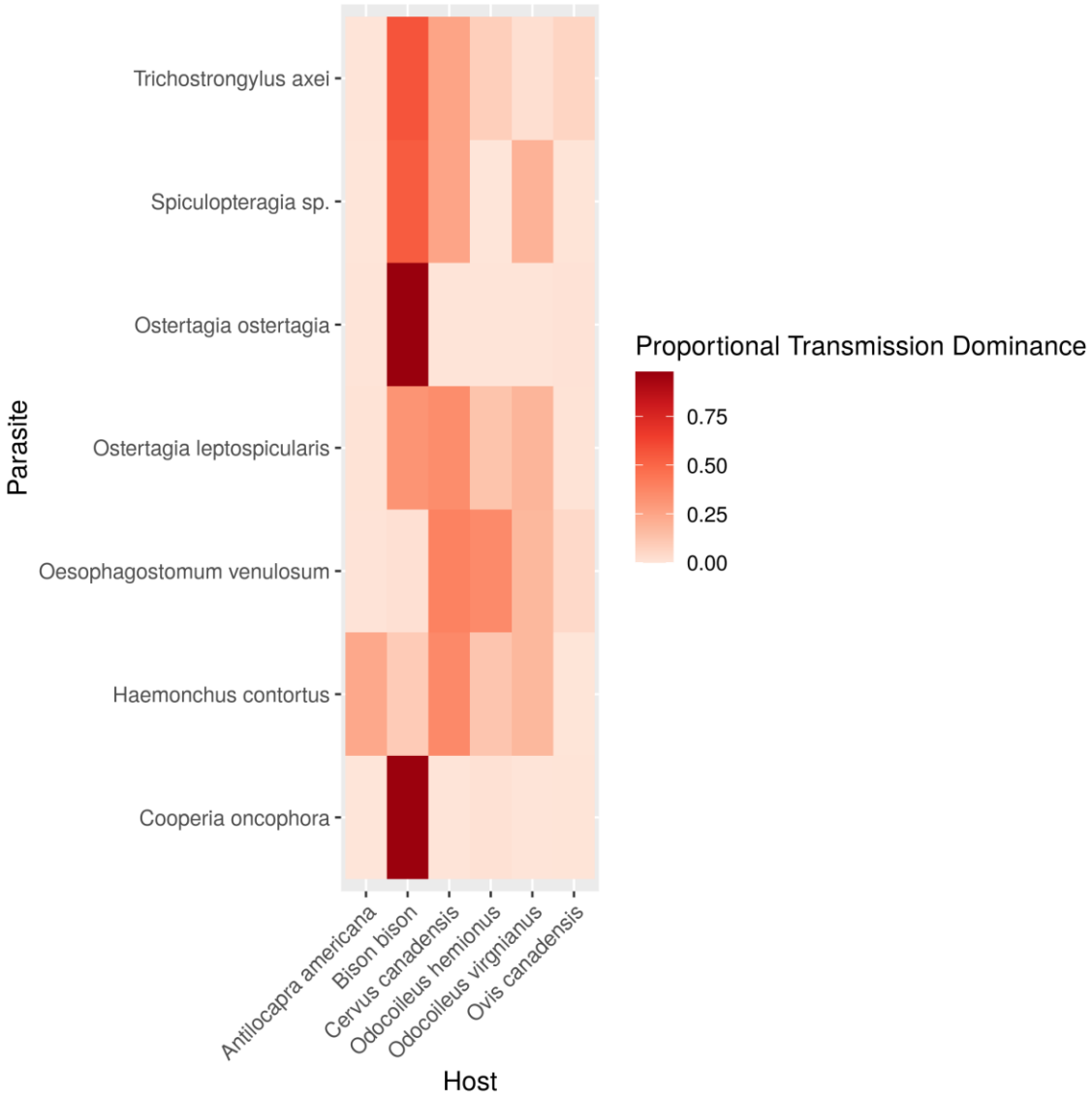

Figure 5.1 The relative transmission dominance of each host species on the NBR for seven shared parasite species. Transmission rates are rescaled as proportion of the transmission of the transmission dominance host species.

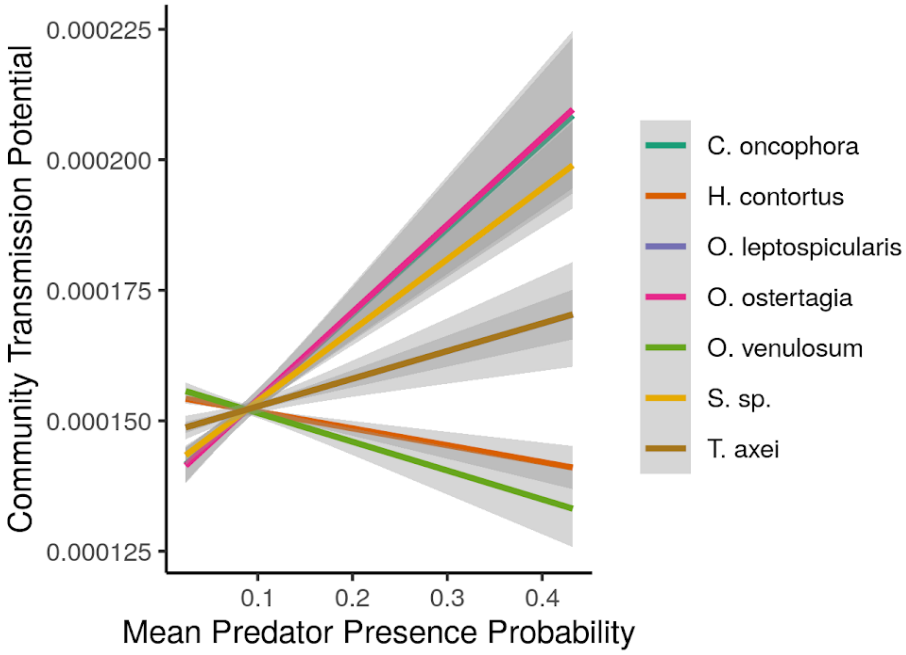

Figure 5.2 The effect of mean predator presence probability in a grid cell on community transmission potential varies by parasite species.

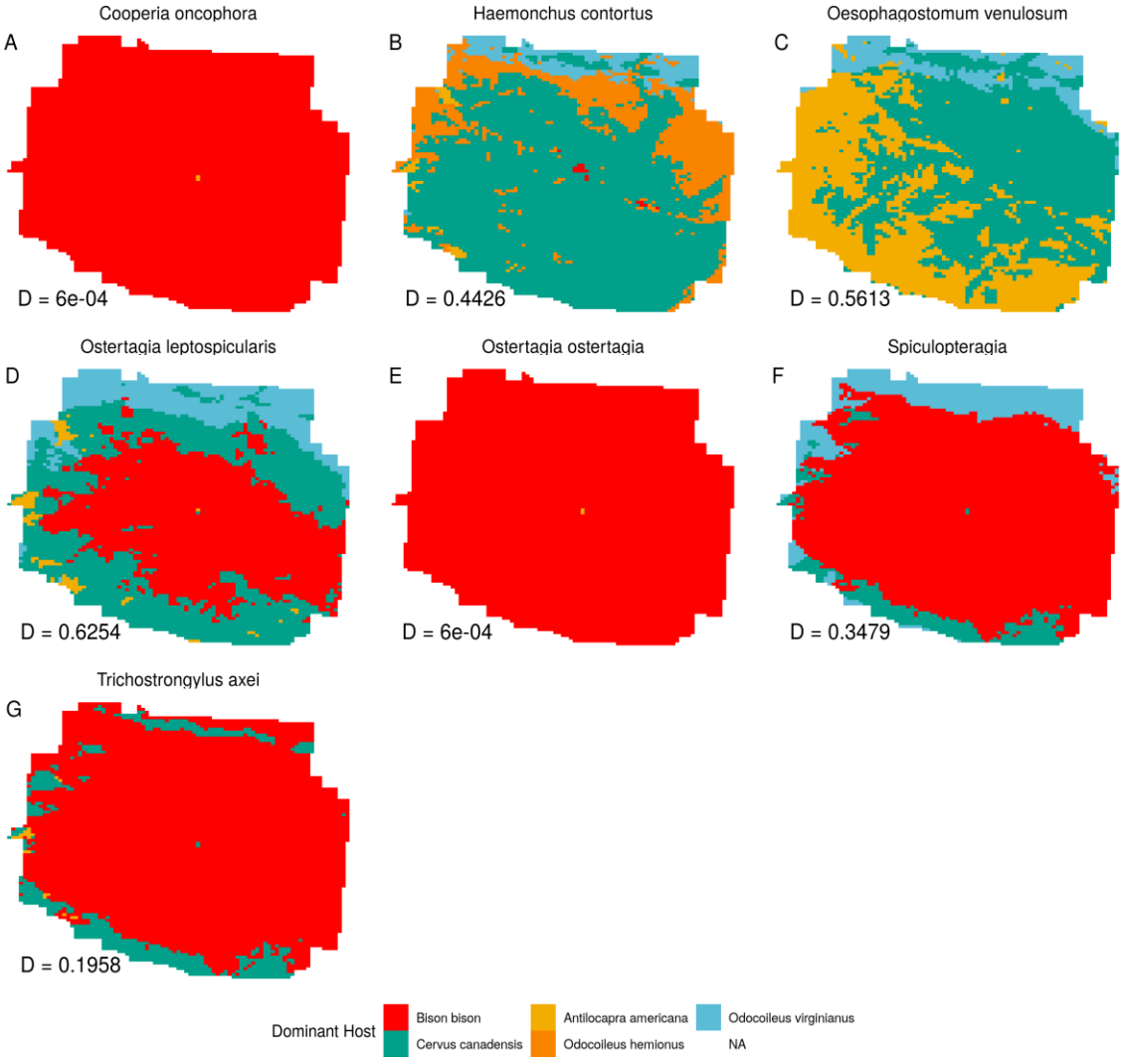

Figure 5.3 Maps show the host species responsible for the largest proportion of the total FOI for each 30x30m grid cell. Simpson's D metrics of spatial diversity of transmission dominance is displayed to the bottom left of each map.

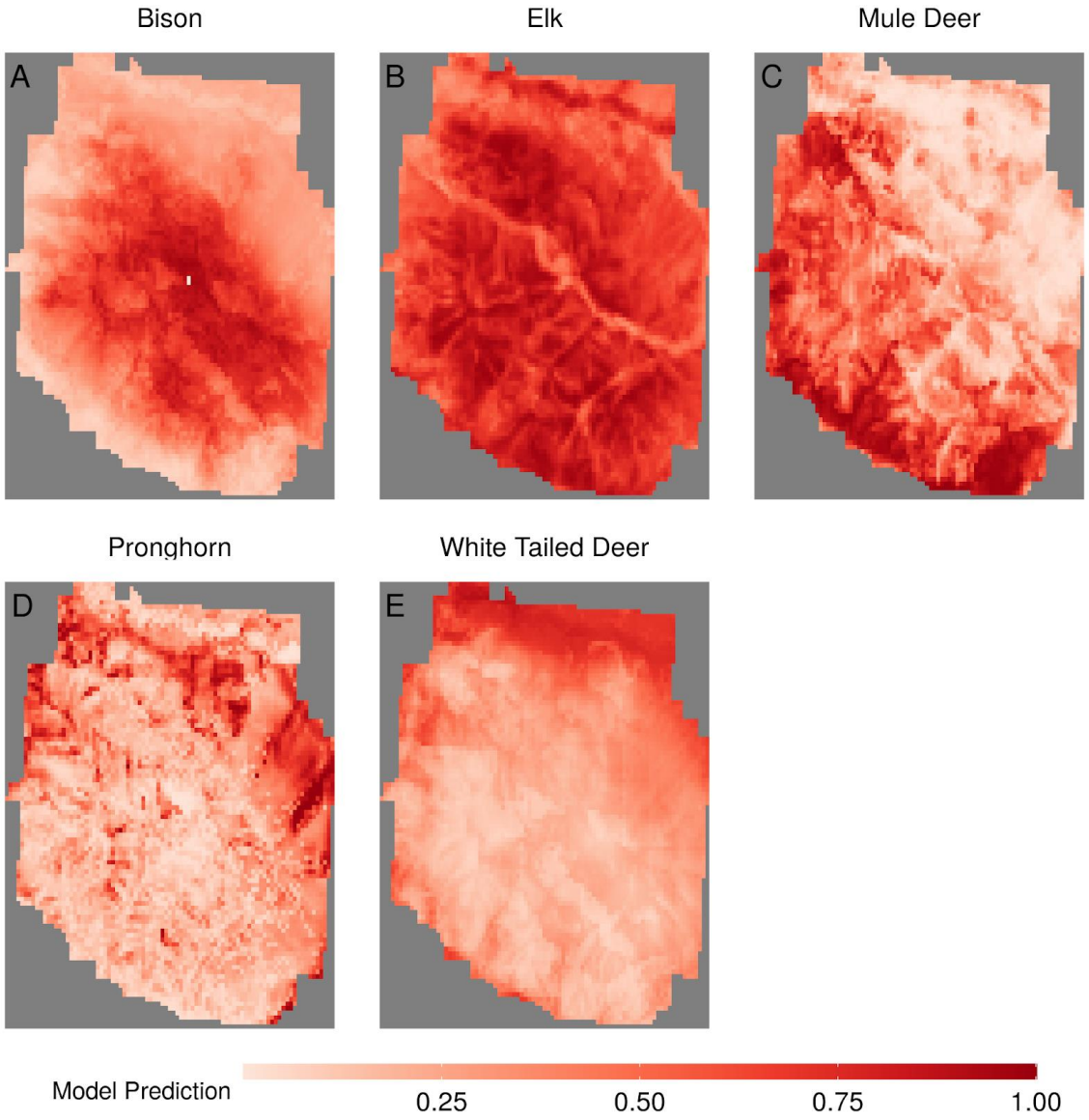

Figure 5.4 The maps above (a-f) present the relative suitability for each host species across the National Bison Range at a 30m scale.

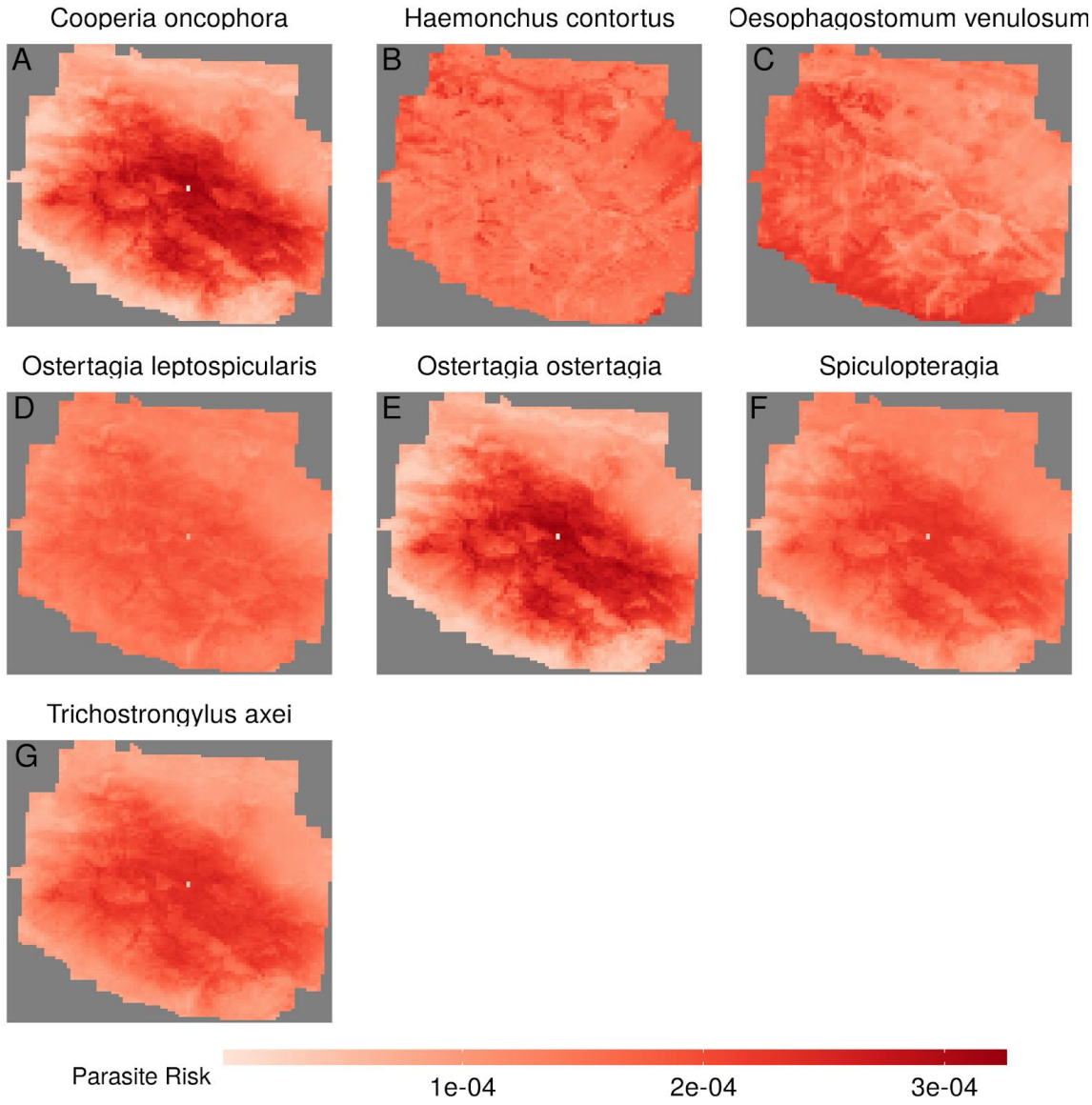

Figure 5.5 Relative transmission potential for each parasite species across the National Bison Range at a 30m scale.

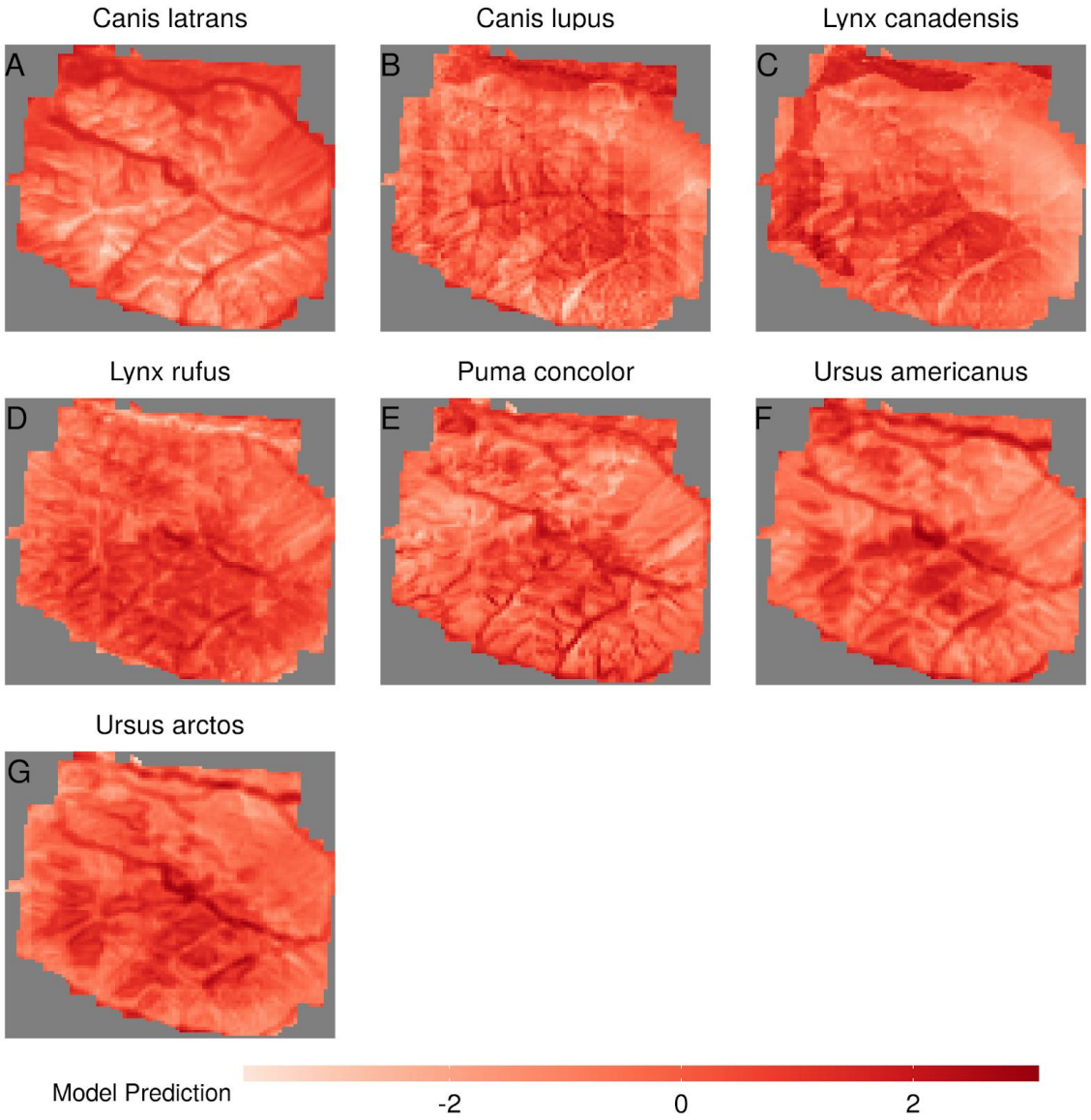

Figure 5.6. Rescaled relative presence probability prediction for each predator species occurring across the National Bison Range at a 90 m scale (Montana Natural Heritage Program. 2020).

Table 5.1: Performance of MaxEnt species distribution models of ungulate host species on the NBR. Performance measured by AUC in 10-fold cross validation and AUC on heldout evaluation data.

Modeled Species	**CV AUC**	**Evaluation AUC**
Antilocapra americana	0.689	0.9
Bison bison	0.734	0.827
Cervus canadensis	0.607	0.705
Odocoileus hemionus	0.812	0.854
Odocoileus virginianus	0.678	0.864

Table 5.2: Environmental covariates included in ungulate species distribution models. Covariates included in the final model are in bold. The cluster column denotes the representative variable from the correlated variable cluster which was included in the final model.

Variable	Resolution	Cluster	Source	Citation
Anual Mean Temperature (BIO1)	30 arc second	BIO19	WorldClim 2.0	Fick and Hijmans 2017
Mean Diurnal Range (BIO2)	30 arc second	BIO7	WorldClim 2.0	Fick and Hijmans 2017
Isothermality (BIO3)	30 arc second	BIO19	WorldClim 2.0	Fick and Hijmans 2017
Temperature Seasonality (BIO4)	30 arc second	BIO19	WorldClim 2.0	Fick and Hijmans 2017
Max Temperature of Warmest Month (BIO5)	30 arc second	BIO19	WorldClim 2.0	Fick and Hijmans 2017
Min Temperature of Coldest Month (BIO6)	30 arc second	BIO19	WorldClim 2.0	Fick and Hijmans 2017
Temperature Annual Range (BIO7)	30 arc second	-	WorldClim 2.0	Fick and Hijmans 2017
Mean Temperature of Wettest Quarter (BIO8)	30 arc second	BIO19	WorldClim 2.0	Fick and Hijmans 2017
Mean Temperature of Driest Quarter (BIO9)	30 arc second	BIO19	WorldClim 2.0	Fick and Hijmans 2017
Mean Temperature of Warmest Quarter (BIO10)	30 arc second	BIO19	WorldClim 2.0	Fick and Hijmans 2017
Mean Temperature of Coldest Quarter (BIO11)	30 arc second	BIO19	WorldClim 2.0	Fick and Hijmans 2017
Annual Precipitation (BIO12)	30 arc second	BIO19	WorldClim 2.0	Fick and Hijmans 2017
Precipitation of Wettest Month (BIO13)	30 arc second	BIO19	WorldClim 2.0	Fick and Hijmans 2017
Precipitation of Driest Month (BIO14)	30 arc second	BIO19	WorldClim 2.0	Fick and Hijmans 2017
Precipitation Seasonality (BIO15)	30 arc second	BIO19	WorldClim 2.0	Fick and Hijmans 2017
Precipitation of Wettest Quarter (BIO16)	30 arc second	BIO19	WorldClim 2.0	Fick and Hijmans 2017
Precipitation of Driest Quarter (BIO17)	30 arc second	BIO19	WorldClim 2.0	Fick and Hijmans 2017
Precipitation of Warmest Quarter (BIO18)	30 arc second	BIO19	WorldClim 2.0	Fick and Hijmans 2017
Precipitation of Coldest Quarter (BIO19)	30 arc second	-	WorldClim 2.0	Fick and Hijmans 2017
Elevation	90 m	BIO19	SRTM 90m DEM Digital Elevation Database	
Slope	90 m	BIO19	SRTM 90m DEM Digital Elevation Database	
Aspect	90 m	-	SRTM 90m DEM Digital Elevation Database	

Roughness	90 m	BIO19	SRTM 90m DEM Digital Elevation Database	
Canopy Cover	30m	-	National Land Cover Database	Coulston et al. 2012
Soil Organic Carbon	100m	-	Soil Properties and Class 100m Grids United States	Ramcharan et al. 2018
Soil sand content	100m	-	Soil Properties and Class 100m Grids United States	Ramcharan et al. 2018
Soil electroconductivity	100m	BIO19	Soil Properties and Class 100m Grids United States	Ramcharan et al. 2018
Soil total nitrogen	100m	Soil Organic Carbon	Soil Properties and Class 100m Grids United States	Ramcharan et al. 2018
Soil pH	100m	BIO19	Soil Properties and Class 100m Grids United States	Ramcharan et al. 2018
Soil bulk density	100m	-	Soil Properties and Class 100m Grids United States	Ramcharan et al. 2018
Soil clay content	100m	-	Soil Properties and Class 100m Grids United States	Ramcharan et al. 2018
***Canis latrans* predicted presence**	90m	- *	Montana Natural Heritage Program	
***Lynx canadensis* predicted presence**	90m	-	Montana Natural Heritage Program	
***Lynx rufus* predicted presence**	90m	-	Montana Natural Heritage Program	
***Puma concolor* predicted presence**	90m	-	Montana Natural Heritage Program	
***Ursus americanus* predicted presence**	90m	-	Montana Natural Heritage Program	
***Ursus arctos* predicted presence**	90m	-	Montana Natural Heritage Program	

*Canis latrans correlates with elevation and roughness but not with the rest of the BIO19 cluster. It was included separately to allow measuring of the effect of predator pressure on distributions. But note that the effect of Canis latrans may be underestimated due to the diagnosed correlation.

Table 5.3: Parameter values used in estimating proportional contribution of each host species to parasite transmission.

		λ = EPG*BMR		Proportions (p)						
	Abundance	EPG	BMR	C. oncophora	H. contortus	O. venulosum	O. leptospicularis	O. ostertagia	S. sp.	T. axei
Antilocapra americana	90	31.58	3214.97	0.167	0.86	0.111	0.096	0.099	0	0.178
Bison bison	400	35.72	22217.21	0.436	0.052	0.053	0.135	0.422	0.267	0.303
Cervus canadensis	248	33	10872.59	0.068	0.396	0.361	0.255	0.047	0.323	0.579
Odocoileus hemionus	161	49.29	4958.79	0.365	0.305	0.521	0.254	0.154	0	0.398
Odocoileus virginiana	190	34.2	4572.85	0.104	0.457	0.418	0.322	0.1	0.25	0.188
Ovis canadensis	88	40.11	4515.96	0.123	0.039	0.182	0.059	0.117	0.077	0.574

CHAPTER 6

CONCLUSIONS

The goals of this dissertation were to rigorously assess the generality of predictions of the healthy herds hypothesis for the effect of predators on parasites in prey and evaluate the importance of heterogeneities in natural systems to the prediction of these effects. Predator-prey-parasite interactions are both ubiquitous and consequential in natural systems but the effects of predators on parasites in prey are so varied that consensus is elusive. I used a wide range of methods, from computational macroecology to experimental field studies, from meta-analysis to species distribution modeling, to identify predictable patterns in the variety of predator-prey-parasite interactions. Here I highlight the contributions that this dissertation makes to our understanding of the effects that predators have on parasites in their prey.

In chapter 2, I used a meta-analysis of published empirical literature to estimate the average effect of predation on parasites in their prey and to test the influence of predator interaction type and parasite group on the strength and direction of this effect. I found that there was no overall directional effect of predators on parasites, instead the effect varied significantly by parasite type and predator interaction. In particular, parasitoids were the only parasite group that significantly decreased with increased predator pressure while non-consumptive predator interactions had more positive effects on parasites than consumptive interactions. The substantial variation we detected even

within these sub-groups suggests that a variety of unmeasured variables also contribute to the strength and direction of predator effects on parasites in their prey.

In Chapter 3, I used a global-scale macroecological analysis of parasites of ungulates (orders: Artiodactyla and Perissodactyla) to measure the importance of parasite traits to the effect of predation pressure on local parasite prevalence, and host-species level parasite diversity. I found that the effect of predation pressure on parasite prevalence varied significantly by parasite transmission mode, parasite taxa, and whether parasites are shared with carnivores, while predation pressure had no effect on parasite species richness. I was not able to elucidate specific mechanisms for the effects of predation on parasite prevalence, but my findings support a role for a variety of different parasite traits in moderating the effect of predators on parasites in prey. In comparison to my meta-analysis (Chapter 2) which detected a difference in predator effects between traditional parasites and parasitoids, this macroecological approach provided the opportunity to explore the role of specific parasite traits. However, the remaining significance of parasite taxa to the predator-parasite interaction in our data supports the need to search further for additional parasite traits, beyond transmission mode and parasite sharing, which moderate predator effects on parasites in prey.

In Chapter 4, I used a large-scale predator exclusion experiment to test whether the effect of mammalian mesopredators on the gastro-intestinal parasites of rodents was influenced by seasonality, fire disturbance, and prey species identity. These factors are rarely included in experimental studies of predator-prey-parasite interactions. We found clear seasonal patterns in the effects of predators on parasites in prey and those patterns varied by prey species and disturbance regime. These findings raise concerns when

compared to my meta-analysis (Chapter 2) in which most studies were conducted either over short time periods or in highly controlled settings, and my macroecological study (Chapter 3) which averages effects across seasons and host species. I suggest that much of the residual variation in both our meta-analysis and macroecological study is due to seasonality and host-species differences. While future experimental and observational studies should explicitly measure these types of variation, it will also be important to assess whether seasonal and disturbance-based variation in predator-prey-parasite interactions produce different long-term effects.

Finally, in Chapter 5, I used a study of transmission and space use heterogeneity in a community of six sympatric ungulate species at the National Bison Range to measure the effect of carnivore space-use on community-level spatial transmission potential. I found that the relationship between predator space use and parasite transmission potential varied by parasite species. Parasites for which *Bison bison* contributed substantially to transmission tended to increase with predation while those for which *B. bison* played little role in transmission tended to decrease. Therefore, our findings of the importance of parasite (Chapter 2, Chapter 3) and host species (Chapter 4) identity to the effect of predators on parasites in prey both extend to the spatial distribution of parasite transmission within host ranges and have important implications for community-level disease transmission. As I've shown that predator effects on parasitism vary from prey species to prey species, the effects of predation on community-level transmission will be unpredictable unless the parasite-host and predator-prey relationships across the community are well characterized.

Together, the chapters of this dissertation identify a number of factors that moderate the effect of predators on parasites in their prey. There are, however, a few important themes among all this variation. Across all chapters, I found strong positive effects of predators on parasites in their prey. While predators also decreased parasitism under certain circumstances, I suggest that the core predictions of the healthy herds hypothesis, that predators protect prey populations from parasitism, may be rarer than previously believed. This finding is further supported by the diversity of mechanisms by which predators can influence parasites in their prey beyond the simple protective consumptive interaction (Duffy et al. 2019). In particular in my work I find strong evidence of the importance of both non-consumptive interactions (Chapter 2, Chapter 4, Chapter 5), and the sharing of parasites between predators and prey (Chapter 3) contributing to these positive effects. Future work ought to focus on further dissection of the drivers of variation that we identified in this dissertation. I found evidence of heterogeneity in predator-prey-parasite interactions not just between but within predator interaction types, parasite groups, and host species. Though I found that consumptive interactions are generally more negative than non-consumptive interactions (Chapter 2, Chapter 4), this pattern was reversed when the consumer was a predator spreader (Chapter 2). Though I found that parasite transmission modes explained some of the differences in parasite responses to predator pressure, parasite taxa also significantly explained additional variation (Chapter 3) making it important to identify the actual parasite traits that produce these taxonomic differences. Though I found that host species differed in the effect of predators on their parasites, these differences were variable across both time (Chapter 4) and space (Chapter 5). Careful study of the drivers of residual

variation across a broad range of host, parasite, and predator taxa may hold the key to both a better understanding of the mechanisms behind predator-prey-parasite interactions and more accurate predictions of how parasite transmission should respond to changes in predation pressure.

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