Intraguild Predation: Interactions between Predators, Pathogens, and Their Shared Resources in Crop Pest Communities

Andrew Jason Flick
Louisiana State University and Agricultural and Mechanical College

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INTRAGUILD PREDATION: INTERACTIONS BETWEEN PREDATORS, PATHOGENS, AND THEIR SHARED RESOURCE

A Dissertation

Submitted to the Graduate Faculty of the Louisiana State University and Agricultural and Mechanical College in partial fulfillment of the requirements for the degree of Doctor of Philosophy

in

The Department of Biological Sciences

by

Andrew Jason Flick
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ABSTRACT

Crop pest management requires an understanding of the complex interactions among pest species that potentially damage crop yield and species that may be crucial for controlling pest species outbreaks. For example, predators, parasitoids, and pathogens are constantly interacting via their shared prey or hosts. Predators may prefer infected prey, which can be easier to catch; however, infected prey may be less nutritious or even lethal for predators. These interactions then dictate the short-term dynamics of host and pathogen as well as between prey and predator. "How these dynamics change as the species in the system change either empirically or theoretically?" is the underlying question in this dissertation.

I conducted a meta-analysis to determine the effects of virus- and fungus-infected prey on predators. Examining experiments with one predator/parasitoid and one pathogen, I quantified life-history responses of predators consuming infected prey across published studies. Predators and parasitoids responded separately to infected prey. True predators had no preference, while parasitoids preferred healthy prey. Both predators and parasitoids had reduced fitness when reared in infected hosts. For example, if the host died from infection before the parasitoid completed development, the parasitoid also died. Predators also had a reduction in fitness when consuming infected prey (i.e., shorter lifespans, fewer offspring produced). I then used lab and field studies to expand on these results.

In the lab, I reared a common agricultural predator, the spined-soldier bug (*Podisus maculiventris*) on a diet of either healthy or infected prey, which responded similarly to those in my meta-analysis. They suffered increased developmental time and decreased longevity. I also found that predators exhibited preference for infected prey while the prey were alive. When prey were frozen, the predators exhibited no preference for healthy or infected prey. This indicates
that prey are likely easier to consume when they are infected. Field studies investigated how predators change disease transmission in their prey. I found that predators increased transmission by decreasing the prey's heterogeneity in susceptibility to the disease. That is, the spread between the least susceptible and the most susceptible host decreased, which increase overall disease transmission. This research extended the results of the meta-analysis from the individual effects to population dynamics.

Finally, I created two mathematical models to show how predators and pathogens interact across multiple generations as compared to the single generation in the field. These models compare and contrast the differences between predator response to prey and the effects the predator response has on disease dynamics. Predators that show a Holling type I response can lead to stable states, while predators exhibiting a type III response lead to cycles exemplified by boom and bust dynamics. Through a meta-analysis, field and lab studies, and a mathematical model, I explored the interactions between predators and pathogens when they attack the same prey or host species. By combining a variety of quantitative techniques to investigate a single question, my work adds important insight into how ecological interactions can help improve agricultural practices.
CHAPTER 1.
INTRODUCTION

Pest control is a multi-billion dollar industry in the United States of America (Paoletti and Pimentel 2000). Chemical pesticides are being developed that are efficient against many pests (Headley 1968). However, the evolution of defenses against pesticides (Georghiou 1972, Maino et al. 2018), along with some deleterious side effects (Paoletti and Pimentel 2000), are pushing the call for safer biological pest control alternatives.

Many biocontrol agents have been introduced throughout history with varying degrees of success. Understanding non-target interactions of biocontrol agents can inform potential ramifications of bio-pesticide misuse. Some consequences of a lack of due diligence before new species are introduced are exemplified below. First, Cactoblastus cactorum was introduced to control prickly pear cactus plants (Opuntia spp) and was successful in Australia (Freeman 1992). However, this was a rare example of successful introduction of biocontrol agents with little preliminary testing as biocontrol efforts are often thwarted by negative spillover or rapid pest evolution. For example, cane toads (Bufo marinus) were introduced to control crop pest scarab beetles (Adoryphorus couloni), but ended up being a bigger pest than the beetles with negative non-target consequences of lowering amphibian diversity and poisoning predator species (Phillips and Shine 2004, Crossland et al. 2008). Additionally, myxoma virus was introduced to control invasive European rabbits (Oryctolagus cuniculus); which were introduced as both pets and game animals (Fenner 2010). While the virus has not had deleterious effects on non-host organisms, the rabbits are quickly evolving anti-viral defenses (Kerr et al. 2003). Predators and pathogens are common choices for biocontrol agents so understanding how they interact with their target species is critical to predicting non-target consequences.
Recent studies to understand the effects of biocontrol agents before they are released have been conducted (Fravel 2005) leading to an increased understanding of some interactions between biocontrol agents and non-target species and basic biotic interactions (Bathon 1996, Kuhlmann et al. 2006). For example, theoretical studies investigating interactions between predators and prey (Berryman 1992, Liu et al. 2005), pathogens and hosts (Anderson 1982, Reilly and Elderd 2014), and parasitoids and their hosts (Briggs and Hoopes 2004); empirical studies test these theories (Morris et al. 2002, Moon and Stiling 2005), and reviews cover the more well-studied questions, such as the effectiveness of generalist predators as biocontrol agents (Symondson et al. 2002).

Theoretical studies often begin by exploring basic interactions between species. For example, one of the first disease models compartmentalized individuals into three different classes and, from a mathematical perspective, looked at how individuals moved from the healthy/susceptible to the infected class and then to the recovered class (Kermack and McKendrick 1927). This so-called SIR model has had important and lasting effects in the fields of epidemiology and disease ecology. Later models building on a similar structure focused on how individuals within each of these classes may differ, which influence the rates of movement between the susceptible and infectious classes (cite Anderson and May's HIV work, Dwyer et al., 1997 as well). While the above focused on one of the simplifying assumptions of the SIR models such that all individuals within a compartment are the same, other model modifications focused on transmission dynamics. In Kermack and McKendrick's model, hosts may become infected following a mass-action function, where hosts are infected at a fixed rate, unrelated to density. However, they may also be infected through a frequency-dependent interaction, where the number of new infections is based on the total population of healthy and infected individuals.
(Antonovics et al. 1995, Begon et al. 1998), or other, more complicated interactions (McCallum et al. 2001). The way in which the model changes depends upon the biology of the disease as dictated by both the host and the pathogen.

This development of theoretical models is common; for instance, predator-prey models started with basic consumption of prey causing increases in predator population size (Lotka 1920, Volterra 1936), and have been expanded to include predator response to low levels of prey and satiation (Holling 1987). Empirical studies test specific conditions of these theoretical models (Harrison 1995, David et al. 2006, Banerji et al. 2015) and reviews and meta-analyses summarize these empirical studies (Preisser et al. 2005). For example, a meta-analysis of the effects of predation risk on prey behavior showed that prey often mediate their behavior based on factors associated with predators, like predator speed and size (Stankowich and Blumstein 2005).

One well-studied interaction is intraguild predation (IGP), which describes how two competitors interact when one competitor consumes the other (unidirectional IGP), or when both competitors can consume one another, usually at different life stages (mutual IGP) (Polis et al. 1989, Polis and Holt 1992). In unidirectional IGP, the competitor that consumes the other is referred to as the top predator and the competitor that is consumed by the other is the bottom predator. Theory predicts that these communities are unstable unless the bottom predator is a more efficient consumer of the shared resource than the top predator (Polis et al. 1989). Many empirical studies have tested these interactions in natural communities and lab studies. A meta-analysis shows that theory is often correct in predicting that bottom predators are more efficient than top predators (Vance-Chalcraft et al. 2007). These results can be applied across community types.
Intraguild predation theory is well suited for biocontrol efforts, especially in crop communities. A suite of natural enemies often exist with the potential to control particular pests. For example, birds, mice, insects, and pathogens consume caterpillar pests in soybeans (Lautenschlager and Podgwaite 1979, Lautenschlager et al. 1979, Abot et al. 1996, Bell et al. 2004). In these natural communities, IGP predicts that if there is already a more efficient consumer of the pest present, then adding a top predator will actually lead to increased pest populations (Vance-Chalcraft et al. 2007). Less well-studied among intraguild predation communities, are the interactions between predators and pathogens used in these biocontrol programs (Fig. 1.1). Predators and pathogens can interact via IGP when they attack the same host and the predator stops reproduction of the pathogen in the prey (Thomas et al. 2006).

![Diagram](image)

Figure 1.1. Direct (black and red) and indirect (blue) interactions between predators and pathogens sharing a prey/host. Black lines indicate conversion of biomass, red lines indicate movement of pathogen particles. Blue lines indicate indirect feedbacks; for instance, predators change prey behaviors that can influence disease dynamics. Lines can represent positive or negative feedbacks, depending on the community.

Many different indirect and direct interactions can exist in these predator, pathogen, and prey communities. For example, predators can reduce the movement of healthy and infected prey, which can lead to lower disease transmission when healthy and infected individuals are separated spatially. Pathogens can also change behavior of their hosts which can lead to
increased predation (Hudson et al. 1992, Lafferty and Morris 1996). Susceptible hosts may also avoid infected conspecifics (Kiesecker et al. 1999), which could lead to increased or decreased predation (Fig. 1.1, blue lines). Directly, predators that consume infected prey may or may not be capable of spreading the pathogen, or even becoming infected themselves (Fig. 1.1, black lines). Predators in agricultural communities tend to spread bio-pesticides by messy eating or defecating infective particles (Abbas and Boucias 1984, Reilly and Hajek 2012). However, birds that consume infected fish near the top of the water column can become infected and die (González et al. 1998), dually suffering mortality (direct negative effect on predator) and removing the pathogen from the host’s community (direct negative effect on pathogen population and indirect positive effect on the susceptible population). There are a myriad of ways in which these interactions dictate the effectiveness of the biocontrol effort and the stability of the community.

A number of interactions between predators and pathogens have received attention in the theoretical and empirical literature. For example, theoretical studies suggest that predators can stabilize chaotic host pathogen dynamics and pathogens could do the same for unstable predator-prey interactions (Hethcote et al. 2004, Ong and Vandermeer 2015). There are two competing theoretical hypotheses for the effects of predators on disease spread. First, predators reduce disease incidence by removing easier to capture, infected prey resulting in a greater proportion of the population being composed of healthy prey (i.e., healthy herds hypothesis) (Packer et al. 2003). Second, predators may spread disease by releasing it in novel areas with healthy hosts (i.e., predator-spreader hypothesis) (Caceres et al. 2009). Some empirical studies exist testing this theory, though more are needed to infer general trends within and among communities.

Many empirical studies in predator, pathogen, and prey communities focus on the potential negative impacts of predators that consume infected prey. These studies are ripe for
meta-analysis to generalize patterns across agricultural communities which I present in chapter 2. I use classic meta-analytical techniques applied to data collected from the ISI Web of Science in 2014 to show that predators have fitness parameters negatively influenced by infected prey. I also show that true predators (e.g., wolf spiders) show no preference for healthy or infected prey, while parasitoids prefer healthy prey. These results suggest that parasitoids can judge a host as fit or unfit for development of their offspring before laying eggs, while true predators will consume any prey that crosses their path. These differences between parasitoids and true predators can have consequences for community dynamics as a whole.

In chapter 3, I use lab and field studies to explore in-depth dynamics between a generalist predator and a baculovirus within a crop pest community. I test interactions between the spined soldier bug (Podisus maculiventris), a naturally occurring predator in crop communities; its prey, the soybean looper (Pseudoplusia includens) a native crop pest to many species of plants; and a generalist lepidoptera virus (Autographa californica Multi-capsid Nuclear polyhedroirus - AcMNPV). I reared soldier bugs using healthy or infected prey and measured fitness parameters (e.g., longevity, fecundity) in response to diet. I also tested soldier bug preference for healthy or infected prey. Predators can influence their prey not just by removing them (possibly in unequal abundances of healthy and infected prey) but also by changing prey behavior. This behavior alteration may lead to changes in disease transmission rates compared to a community without predators. I found that soldier bugs have reduced fitness when consuming infected larvae and prefer infected larvae. In the field, I tested how the presence of soldier bugs influences disease spread by using plots with predators, plots with predators with no mouth-parts, and plots without predators. I found that plots with predators had the highest transmission rates; however, the transmission rate in plots with predators with no mouth parts
responded similarly to plots without predators. Finally, I fit a mechanistic model to data to show that predators decrease the heterogeneity in disease risk of their prey. These predators preferred infected prey that negatively influenced their life-history proxies while increasing disease transmission in the field.

While the experiments quantified how IGP affects disease outbreaks over the course of a single round of transmission, they are unable to provide any insight with regards to the IGP community's long term dynamics. Thus, in chapter 4., I present two mathematical models exploring long-term outcomes of these communities. I use a Holling type I response for specialists (predators can always find prey at low densities and do not become satiated at high densities) and a Holling type III response for generalists (predators switch prey at low densities and become satiated at high densities) to test dynamics of the interactions among healthy prey, infected prey, cadavers, and predators. I found that under a suite of parameter values the type of predation (i.e., the Holling response) is important for predicting if communities will be stable or unstable. I also found predators that can spread pathogen when consuming infected prey can rescue the pathogen from extinction. This suggests that generalist predators and pathogens cannot coexist within the range of parameters I tested.

In chapter 5., I discuss the results from the meta-analysis, empirical studies, and modeling. I also present opportunities to expand current research. In sum, the dissertation work uses a variety of techniques to understand how predators and pathogens interact within agricultural communities. I found important differences between generalist and specialist predators which can have important consequences for understanding natural communities and for developing effective biocontrol strategies.
CHAPTER 2.
THE NEGATIVE EFFECTS OF PATHOGEN-INFECTED PREY ON PREDATORS: A META-ANALYSIS*

INTRODUCTION

Top–down interactions play a vital role controlling population dynamics at lower trophic levels (Paine 1980, Power et al. 1985, Kohler and Hoiland 2001, Schmitz and Suttle 2001). Typically, these interactions consist of a predator consuming its prey or a pathogen consuming its host. Historically, studies that allowed for multiple predators in a community assumed they acted as competitors (Griffiths and Holling 1969, Bazykin et al. 1981, Creel and Creel 1996); however, predators and pathogens may affect community dynamics through intraguild predation (IGP). IGP interactions between predators have been well-studied empirically (Rosenheim et al. 1993, Browne and Rasmussen 2013) and synthesized through meta-analyses (Vance-Chalcraft et al. 2007, Mooney et al. 2010). These studies suggest that the effects of the predator on the resource vary across ecosystems and the species of the predator. While Thomas et al. (2006) suggested that parasitoids and pathogens interact through IGP, few studies have set out to directly test this hypothesis. The importance of parasites for understanding community ecology and structure has recently been recognized; however, it is still a major gap in the literature (Lafferty et al. 2008, Johnson et al. 2010, Thieltges et al. 2013). Macro-parasites have received some attention (Rohr et al. 2015), though micro-parasites or pathogens have not been well considered.

Previous theoretical work on within generation dynamics, shows that predators under most circumstances should consume parasitized prey (Lafferty 1992). However, Lafferty (1992) only considered parasites that are trophically-transmitted (i.e. when the infected prey represent a secondary host of the parasite). Yet, a large number of parasites, both macro- and micro-, are consumed concomitantly with the prey and the parasites are not transmitted to the predator (Johnson et al. 2010). Additionally, Lafferty (1992) assumed that parasites do not alter the energetic value of the prey. This may not always hold true since parasites may affect the energy gained from consuming infected prey compared to non-infected prey (Thieltges et al. 2013). Changes in the energetic value of the prey, in turn, may affect the predator’s foraging behavior as well as important life-history metrics (e.g. fecundity and survival).

Theory predicts that predator choice and behavior, along with prey quality (e.g. pathogen-infected or healthy), are important in determining IGP community structure (Holt and Polis 1997, Borer et al. 2007, Mooney et al. 2010, Sieber and Hilker 2011). Empirical evidence examining the impacts of predator choice and host quality on the interactions between predators and pathogens is currently lacking. However, there are a considerable number of studies showing the short-term impacts of predator choice and host quality on the intraguild predator fitness and associated life-history traits (e.g. predator life-span) in agricultural systems. By examining how predator behavior and life-history traits may change due to interactions with pathogens specific to the prey, I can gain greater insight into IGP community dynamics.

In general, IGP communities consist of three main players: an intraguild predator (IG$_{\text{Pred}}$), an intraguild prey (IG$_{\text{Prey}}$), and a basal resource (Fig. 2.1). In agricultural systems specifically, many biocontrol programs use a combination of predators and pathogens, creating predator–pathogen–resource IGP communities (Poland 2007). The experiments investigating these
biocontrol programs provide information on how a single pathogen influences life-history traits of a single, non-target predator. These experiments isolate the IGP interaction without having to separate the various components of a community into simplified community modules (Holt and Polis 1997). Thus, a meta-analysis of these single IGP systems will allow us to make generalizations about how resource quality influences IGP interactions.

Figure 2.1. A diagram of interacting enemies described by intraguild predation (IGP) including a predator (IGPred), a pathogen (IGPrey), and a prey resource. Arrows represent the conversion of biomass. Here, the IGPRey is represented by an infected prey. The lower black curved arrow represents infected prey that clear the pathogen and become healthy prey. The IGPred can consume either infected prey or healthy prey. The pathogen can only infect healthy prey. Adapted from Borer et al. 2007.

In this study, I used a meta-analytical approach to synthesize empirical work on how prey quality influences predator behavioral and life-history traits in pathogen-driven IGP communities consisting of predators, pathogens and prey. Since most field experiments do not run long enough to investigate long-term population dynamics, I were limited to life-history responses within a generation. I further focused the efforts on crop pest insects as resources, infected pest insects as IGRey, and their predators as IGPred (Fig. 2.1), given the plethora of studies available
and the degree to which these studies are able to isolate IGP interactions (Supplementary material Table A.4). In agricultural ecosystems, a great deal of research is focused on economically costly pests and controlling them (King and Saunders 1984, Moscardi 1999, Williams et al. 2013). While the study contains four orders of insect pests, many of these pests are contained within Lepidoptera whose members are frequently preyed upon by predators and pathogens alike (Clark et al. 1994, Moscardi 1999, Liu et al. 2014). I predict that infected resources would reduce longevity, fecundity, and survival of the IG_{Pred}. I also predicted that predators would prefer healthy prey to infected prey across predator and pathogen types; as infected prey represent low quality resources. The results show that lowered resource quality reduces life-history metrics such as lifespan and fecundity of the IG_{Pred}, which can have important consequences for disease dynamics and IGP interactions.

MATERIALS AND METHODS

Literature Search

I searched the ISI Web of Knowledge database (ending November 2015) for the following Keywords: “virus insect predator”, “fungus insect predator”, “virus insect parasitoid” and “fungus insect parasitoid” (see Table A.4 for a list of studies used). Bacterial studies were not included for two reasons, 1) the majority focus on crops containing Bacillus thuringiensis (Bt) in their genome and thus do not represent IGP communities and 2) the majority of non-Bt studies focus on bacterial pathogens that are in some way symbiotic with their hosts (e.g. studies on the effects of Wolbachia) (Xie et al. 2014, Furihata et al. 2015). I restricted the analysis to studies that included a predator or parasitoid in the presence of both an inherently lethal pathogen-infected (treatment) and healthy prey (control). I excluded pathogens that do not
regularly kill their hosts. Using these data I were able to compare the effects of consuming healthy prey versus infected prey on predator and parasitoid life-history parameters. I also included cross-citations from the studies chosen that included a consumer exposed to pathogen-infected prey.

To conduct the meta-analysis, I included studies (N = 50) that investigated arthropod predation of crop pests that reported mean, standard errors and sample sizes. I combined all consumer and pathogen types within each life-history trait. Then I categorized the studies by pathogen type (virus or fungus) and finally consumer type (strict predator or parasitoid). Using each of the above categories (Supplementary material Table A.3), I analyzed whether there were differences in each of the traits considered.

In the systematic literature review, I searched for studies that compared the influence of infected and healthy insect crop pests on non-target consumer life-history parameters (i.e. longevity, development, fecundity and survival) that influence realized fitness (Roitberg et al. 2001). I also examined predator preference when presented with healthy and infected prey. For each study, I compared control to treatment groups. Control groups of IG_{Pred} were exposed to healthy prey while the treatment groups were exposed to pathogen-infected prey. Each life-history parameter of the IG_{Pred} was defined a priori as follows. I defined the development time as the mean time from egg to adult or mean time from the nymph to the adult stage. Longevity was quantified as mean life-span from egg or nymphal stage to death. Fecundity was the mean number of eggs produced. To quantify survival, I extracted the mean number of consumers surviving after two weeks. Finally, I defined the IG_{Pred} choice as the mean number of infected prey chosen compared to the mean number of healthy prey chosen. In addition to these means, I also collected standard deviations and sample sizes. Many studies focused on one of the above
traits, however, a few studies focused on two or more. If each life-history trait was tested independently, than they were included in the meta-analysis; if they were not independent I randomly chose only one life-history trait from a single experiment.

**Data Analysis**

To standardize data reported in different scales or magnitudes, I calculated Hedges’ $d$ weighted average metrics using means, standard deviations, and samples sizes from each study (Rosenberg et al. 2000). Hedges’ $d$ incorporates overestimate-bias, working well for small sample sizes in meta-analyses ($N = 5$). Mean effect sizes were considered small in the range from 0.2–0.4; moderate effects ranged from 0.4–0.7; strong effects ranged from 0.7–1.0 (Cohen 1988, Gaskin and Happell 2013). Any results with a mean effect size greater than 1.0 were considered very strong (Cohen 1988, Gaskin and Happell 2013).

I calculated Hedges’ $d$ for each study, $i$, as:

$$d_i = \frac{\bar{X}_i^E - \bar{X}_i^C}{S} J_i$$  \hspace{1cm} (1)

where $\bar{X}_i^E$ is the mean of the treatment and $\bar{X}_i^C$ is the mean of the control group. $S$ is the pooled standard deviation of the control and experimental groups for each study within a treatment. $J_i$ incorporates overestimate-bias by standardizing for small sample sizes such that:

$$J_i = 1 - \frac{3}{4(N_i^C + N_i^E - 2)} - 1$$  \hspace{1cm} (2)

where $N_i^C$ is the number of replicates in the control and $N_i^E$ is the number of replicates in the experimental treatment for study $i$ (Gurevitch 1993). The variance in Hedges’ $d_i$ is defined as:

$$\nu_{d_i} = \frac{N_i^C + N_i^E}{N_i^C N_i^E} + \frac{d_i^2}{2(N_i^C + N_i^E)}$$  \hspace{1cm} (3)
I used Hedges’ $d_i$ values and variances to calculate the overall mean effect size, $E$:

$$
\bar{E} = \frac{\sum_{i=1}^{M} w_i d_i}{\sum_{i=1}^{M} w_i}
$$

(4)

where $w_i$ is $1/v_{di}$ and $M$ is the total number of studies. This value describes the direction (i.e. positive or negative) and the strength of the effect. The mean effect size is expressed as the number of standard deviations from the experimental treatment to the control. I considered treatments significant when their 95% confidence intervals did not overlap zero and the absolute value of the mean effect size was greater than 0.2 (Rosenberg et al. 2000).

All analyses were conducted using MetaWin 2.1 (Rosenberg et al. 2000). If the confidence interval did not overlap zero, I used Rosenthal’s value ($N_R$) to determine if results were robust. This measure calculates the number of insignificant studies with mean effect size of zero needed to render the results insignificant at the 0.05 level (Rosenberg et al. 2000). I calculated Rosenthal’s value as:

$$
N_R = \left( \sum_{i=1}^{N} Z(p_i) \right)^2 \frac{Z_{\alpha}^2}{N} - N
$$

(5)

where $Z(p_i)$ is defined as the individual $Z$ score for each Hedges’ $d_i$ and $Z_{\alpha}$ is the associated one-tail $Z$ score with $\alpha = 0.05$ (Rosenberg et al. 2000). I consider Rosenthal’s number to be robust if $N_R > 5M + 10$ (Stiling and Cornelissen 2005). That is, I would still have significant results if more than five times the number of published studies were unpublished due to insignificant results.
While Hedges’ $d$ incorporates a standard overestimate-bias for small sample sizes, I also used trim and fill analyses which can effectively evaluate publication bias in meta-analyses (Duval and Tweedie 2000). Using this method allowed us to assess the number of missing studies due to publication bias against null results (Supplementary material Table A.1).

**Data Deposition**

Data available from the Dryad Digital Repository: <http://dx.doi.org/10.5061/dryad.9ht4g> (Flick et al. 2016).

**RESULTS**

![Figure 2.2](image)

Figure 2.2. The effect of pathogen type on life history of the IG$_{Pred}$. Mean effect sizes and 95% confidence bars for the influence of virus-infected prey (black points) and fungus-infected prey (open points) compared to healthy prey on consumer life history traits. Combined fungus and virus results are shown in gray triangles. Cross symbols above the individual points represent robust results based on Rosenthal’s fail safe number ($N_R$). $N_R$ is the hypothetical number of unpublished studies with null results necessary to create a non-significant result. Specific Rosenthal’s values can be found in Supplementary material Table A.3.

Resource quality had a significant effect on the fitness of the consumer. Pathogen infection reduced consumer longevity by 26%, fecundity by 31%, and survival by 13% (Fig. 2.2, gray triangles). When given a choice between healthy and infected prey, consumers chose pathogen-infected prey 28% less often (Fig. 2.2, gray triangles). Development time was not significantly longer in consumers exposed to infected prey with the 95% confidence intervals
overlapping zero (Fig. 2.2, gray triangles). Overall, the IG_{Pred} decreased in survival, longevity, and produced fewer offspring when they consumed pathogen-infected prey (Fig. 2.2, gray triangles).

![Figure 2.3](image)

Figure 2.3. The effect of the IG_{Prey} on life history traits of predators and parasitoids. Predators and parasitoids responded differently when comparing survival and choice of infected prey to healthy prey. Mean effect sizes and 95% CIs of life history parameters of predators (open points) or parasitoids (black points) consuming infected prey compared to healthy prey. Combined predator type results are shown in gray triangles. Note, the gray triangles are the same as those in Fig. 2.2 and are show for comparison. Cross symbols above the individual points represent robust results based on Rosenthal’s fail safe number ($N_R$). $N_R$ is the hypothetical number of unpublished studies with null results necessary to create a non-significant result. Specific Rosenthal’s values can be found in Supplementary material Table A.3.

When analyzing the data by pathogen type, fungus-infected prey caused a 5% increase in developmental time and a 22% reduction in longevity of the IG_{Pred} (Fig. 2.2, open points). Fungus-infected prey did not influence fecundity, survival, or choice. Prey infected with viruses caused a 29% decrease in longevity, a 32% decrease in fecundity, and a 30% reduction in survival of the IG_{Pred} (Fig. 2.2, black points). The IG_{Pred} chose healthy prey 29% more often than virus-infected prey (Fig. 2.2, black points). When examining the effects on different types of intraguild predators, virus-infected prey did not affect development of predators or parasitoids (Supplementary material Table A.2). Clearly, virus-infected prey were driving the combined
effect seen in Fig. 2.2 with respect to development and fecundity. However, virus- and fungus-infected prey both lowered consumer fitness, albeit in different ways.

Differences in various life-history metrics also depended upon whether the IG$_{\text{Pred}}$ was a strict predator or a parasitoid. Parasitoids had a 22% decrease in longevity and a 32% decrease in fecundity when parasitizing pathogen-infected prey compared to healthy prey (Fig. 2.3, black points). Parasitoids also chose healthy prey 31% more often compared to prey infected with pathogens. Pathogen-infected prey did not influence parasitoid development or survival. For strict predators, pathogen-infected prey caused a 33% reduction in longevity, a 38% reduction in fecundity, and a 45% decrease in survival (Fig. 2.3, open points). However, there was no influence of pathogen-infected prey on predator development or choice. The largest overall effects were on strict predator fitness in general and parasitoid choice.

DISCUSSION

Infected prey clearly represent a poor resource regardless of infection type (Fig. 2.2), and predators respond to those infected prey in different ways (Fig. 2.3). For instance, parasitoids preferred healthy prey, while non-parasitoid, or strict, predators did not exhibit a preference for or against healthy prey. Thus, the type of predator affected whether or not IGP occurs in a community. The type of IGP dynamics, in turn, will have important consequences for whether the predators increase or decrease pathogen spread in the community (Rohr et al. 2015) and may have both short and long-term consequences for community dynamics.

Strict predators passively interact with pathogens through IGP (Fig. 2.1). In fact, pathogens can cause prey to be more readily captured, thus increasing the likelihood of a community exhibiting IGP dynamics (Thomas et al. 2006). This may often be the case if the energy gain from easier to capture prey outweighs the cost of nutrient loss due to suboptimal
prey (Holmes and Bethel 1972). Predators may also consume pathogen-infected prey if they are unable to identify a prey item as infected. In terms of community dynamics, predators may remove pathogens from the environment (Roy et al. 1998); however, the predator may defecate viable pathogen (Beekman 1980, Biever et al. 1982, Bruck and Lewis 2002), thus increasing the number of infected resources (Caceres et al. 2009). The importance of the nutritional value of infected prey, the energetic consequences of consuming infected prey, and increasing or decreasing pathogen availability in the community are important topics that require further investigation (Johnson et al. 2010) and are likely to vary among predators and pathogens.

Parasitoids may be either the IG$_{\text{Pred}}$ or the IG$_{\text{Prey}}$ (Hochberg et al. 1990, Thomas et al. 2006). They are the IG$_{\text{Prey}}$ when pathogens kill a parasitized host before the parasitoid can complete development (Furlong and Pell 1996, Thomas et al. 2006), and are the IG$_{\text{Pred}}$ if they finish development in the host, thus reducing the amount of host available or even killing the pathogen (Pell et al. 1997, Packer et al. 2003). However, as parasitoids avoid infected prey (Fig. 2.3), IGP interactions are likely rare in a parasitoid–pathogen-resource community. Instead, the community will simply consist of a predator and a pathogen competing for a shared resource and would not constitute an IGP community, though this may increase pathogen spread (Rohr et al. 2015). Predator behavior is also important for shaping the interactions in a predator–pathogen IGP community. Rosenheim et al. (1995) showed that predators had varying levels of preference for parasitized larvae (i.e., higher preference: Ruberson and et al. 1991, lower preference: Brodeur and McNeil 1992, no preference: Hoelmer et al. 1994). As suggested by Hochberg et al. (1990), consumers of pathogen-infected prey respond in a like manner (i.e., lower preference: Pell and Vandenberg 2002, higher preference: Thomas et al. 2006, no preference: Roy and Holt 2008). In general, I showed that parasitoids prefer healthy prey while strict predators, on average,
do not prefer healthy or infected prey (Fig. 2.3). This result has important consequences for whether an IGP community can be maintained or if one or more members will be excluded (Vance-Chalcraft et al. 2007). Using IGP theory and experimental evidence to understand when a pathogen acting as a biocontrol agent is excluded through prey release rather than suppression will reduce wasted effort as those communities would collapse into simple predator–prey systems (Holt and Polis 1997, Vance-Chalcraft et al. 2007).

IG$_{\text{Pred}}$ preference as well as IG$_{\text{Prey}}$ behavior can also affect both short-term and long-term dynamics of a community. Rohr et al. (2015), using a trematode–amphibian system, showed that the IG$_{\text{Pred}}$ of the host and the free-living parasite decrease infection rates in the host to a lesser extent than a predator that only consumes the parasite. The system’s response to the IG$_{\text{Pred}}$ is driven by changes in host density via density-mediated indirect effects and host behavior via trait-mediated indirect effects. This can have important consequences for IGP communities and the introduction of potential biocontrol agents in agricultural systems. For instance, using a predator that does not discriminate against infected prey would drive the pathogen locally extinct.

IGP theory also predicts that increased habitat complexity increases long-term stability (Janssen et al. 2007), and empirical studies support this prediction (Finke and Denno 2002, Okuyama 2008). Resources that become infected often change their movement behavior (Vasconcelos et al. 1996a). As the pathogen spreads through a population, differential movement of infected and healthy individuals may set up a spatial mosaic such that certain parts of the landscape are dominated by either low or high quality prey items. This shifting mosaic may allow for long-term IGP stability on a larger spatial scale. Long-term studies investigating IGP stability in these communities will elucidate important consequences for disease dynamics.
Previous theoretical work on short-term dynamics showed that predators should readily consume parasite-infected prey if the cost of a potential infection for the predator is low and catchability of the prey is high (Lafferty 1992). However, the model assumed that infected prey were trophically-transmitted and did not differ in quality. For the study, the parasites were concomitantly consumed and are lower quality as evidenced by changes in various life-history metrics, especially for strict predators. These metrics represent proxies for what may happen under field conditions; however, they are not direct measurements of a predator’s response to the environment when presented with a landscape of non-infected and infected prey. For instance, I do not have enough information on differences in overall attack rate and handling time between infected and non-infected prey (but see Jiang et al. 2011). The results point to the need to better understand how changes in foraging strategies in the field will affect both short-term and long-term dynamics from an empirical and theoretical perspective.

I focused the attention on communities made up of crop pests and their natural enemies. Given that these communities are simplified and potentially novel systems (Altieri and Letourneau 1982, Swift and Anderson 1994), they may not reflect the complexities of other ecological systems. However, to understand how intraguild predation influences more complex communities, it is necessary to start with communities where specific interactions can be directly observed and tested. These tractable systems also represent a sub-set of natural communities or community modules (Holt and Polis 1997), which are often the focus of research in nonagricultural systems. These communities isolate predators and pathogens and may yet hold more insights for future work.

Theory and empirical evidence suggest that resource quality affects long-term stability of an intraguild predation community. Given that resource quality affects both behavioral and life-
history traits of consumers, resource quality can clearly decrease the fecundity and survival of the $I_{G_{pred}}$ over a short time scale, such as that of an experiment. While the long-term effects are unknown, I can speculate that the short-term impacts arising from changes in resource quality will have important consequences for system stability. Long-term experiments are still needed to better understand the impacts of resource quality on IGP dynamics.
CHAPTER 3.
INTRAGUILD PREDATION INCREASES PATHOGEN TRANSMISSION IN AN HERBIVORE HOST AND DECREASES PREDATOR FITNESS

INTRODUCTION

In natural and agricultural communities, predators and pathogens exert strong forces on the population dynamics of their prey and hosts, respectively (e.g., McMurtry et al. 1992, Massana et al. 2007). The extent of their effects are exemplified by the number of systems in which predators or pathogens drive the long-term dynamics of their prey or hosts (Elton and Nicholson 1942, Hudson et al. 1992, Dwyer et al. 2004). In most natural systems, predators and pathogens are both present. Predators and their prey or pathogens and their hosts do not live in isolation and can interact via intraguild predation (IGP), where species consume potential competitors for a resource (Polis et al. 1989). A community made up of pathogens, predators, and prey is very similar to classic examples of unidirectional IGP, where one predator (i.e., the intraguild predator) consumes another (i.e., the intraguild prey), but not vice versa (Borer et al. 2007). Here, the predators consume both the pathogen and the prey, while the pathogen consumes only the prey (Thomas et al. 2006, Fig. 3.1). The combined effects of predators and pathogens may have important consequences on the short-term and long-term dynamics of these IGP systems (Ong and Vandermeer 2015).

From the pathogen’s perspective, the predator can decrease transmission by consuming healthy or infected prey or by changing prey behavior due to non-consumptive effects. For instance, predators may reduce disease transmission via the "healthy herds" hypothesis (Packer et al. 2003) such that infected individuals are easier to capture for predators and the predators in turn are incompetent hosts for the parasites (Thomas et al. 2006, Holt and Roy 2007, Johnson et al. 2010). Predators can also kill hosts before the parasite reaches an infectious stage (Thomas et
al. 2006). Thus predators can cull the host population below the epizootic threshold population size (Packer et al. 2003), a density-mediated indirect effect (DMIE). Additionally, predators can reduce prey activity (Preston et al. 2014), which may make prey less likely to become infected by an environmental pathogen, constituting a non-consumptive trait-mediated indirect effect (TMIE). Each of the above examples, whether consumptive or non-consumptive, results in a decrease in disease prevalence.

Figure 3.1. A predator, pathogen, and prey/host diagram of intraguild predation in this study. Arrows represent the conversion of biomass between organisms. *Podisus maculiventris* consumes both healthy and infected *Pseudoplusia includens*. The baculovirus only infects healthy *P. includens*. *P. maculiventris* spreads the virus without becoming infected, thus potentially increasing viral distribution. The dashed line indicates virus transferred from the infected group to the virus group via the predator. Adapted from Borer et al. (2007).

There are a number of other possible ways in which a predator can alter disease transmission and disease outbreak dynamics. For instance, others have also posited that predators may produce "unhealthy herds" arising from TMIEs where adding predators to the system results in prey becoming more susceptible to their potential pathogen (Duffy et al. 2011). In contrast, under the "predator spreader" hypothesis, transmission increases due to predators physically
spreading parasites to susceptible hosts (Pell et al. 1997, Caceres et al. 2009) or by changing the behavior of prey to increase prey susceptibility to parasites; another example of a potential TMIE (Raffel et al. 2010, Duffy et al. 2011, Rohr et al. 2015). Additionally, predators may increase the proportion of infected individuals by feeding exclusively on healthy prey, an example of a DMIE driven by consumptive mechanisms (Lozano 1991, Holt and Roy 2007). In general, the importance of predator consumptive and non-consumptive effects are equivocal in terms of whether pathogen transmission should increase or decrease.

The above hypotheses regarding consumptive and non-consumptive effects guide the understanding of how predators change disease dynamics. However, predators are also affected by the parasites of their prey. Parasites influence the prey quality for predators (Sanchez et al. 2009, Thieltges et al. 2013, Flick et al. 2016) as well as catchability (Hudson et al. 1992, Arthurs and Thomas 2001, Thomas et al. 2006). In some cases, the reduction in fitness caused by consuming infected prey is compensated for by the increase in ease of capture for the predator (Dobson 1988, Lafferty 1992, Johnson et al. 2010). The likelihood for negative fitness consequences likely varies based on the severity of indirect effects of the pathogen on predator fitness, increased catchability of the prey item, and whether the predator is a generalist or a specialist. For example, specialist parasitoids are more likely to avoid infected prey compared to true predators (Flick et al. 2016).

In the study, however, I investigate both parts of the IGP interaction using the same system by testing predator preference, predator fitness when consuming infected hosts, and also how predators change the disease dynamics of their prey. I investigated how a predator, the spined soldier bug, interacts with a lethal baculovirus and its host, the soybean looper, which is also the prey of spined soldier bugs (Fig. 3.1). Following a recent meta-analysis, I predicted that
spined soldier bugs would not show preference (Flick et al. 2016). Next, I predicted infected soybean loopers would negatively impact fitness parameters of the spined soldier bugs as spined soldier bugs cannot digest virus occlusion bodies. Finally, I predicted that the spined soldier bugs would reduce disease transmission in soybean loopers via both consumptive and non-consumptive effects following the healthy herds hypothesis. Additionally, I tested if predator preference was driven by prey behavior. Many empirical studies exist testing preference of predators for infected or healthy prey and fitness consequences of predators consuming infected prey (e.g., Abbas and Boucias 1984, Down et al. 2004, Jiang et al. 2011). Theoretical and empirical studies investigate the influence of predators on pathogens of their prey (e.g., Packer et al. 2003, Caceres et al. 2009, Sieber and Hilker 2011, Rohr et al. 2015).

MATERIALS AND METHODS

Study system

To understand the potential consumptive and non-consumptive effects associated with intraguild predation on predator fitness and pathogen transmission, I conducted a series of laboratory and field experiments using an easily manipulated system of naturally occurring agricultural species. The system consisted of a single host/prey species, the soybean looper (Pseudoplusia includens Walker, Lepidoptera: Noctuidae) that can be consumed by a generalist predator, the spined soldier bug (Podisus maculiventris Say, Heteroptera: Pentatomidae), and infected by a lethal baculovirus, Autographa californica multicapsid nuclear polyhedrovirus (AcMNPV). The soybean looper is a widespread polyphagous multivoltine pest in crop systems throughout North and South America (Herzog 1980, Smith et al. 1994, Bernardi et al. 2012). The spined soldier bug is a common predatory stink bug, with a distribution from Mexico to Canada that feeds on many crop pests (O’Neil 1995, Yang 2000). The AcMNPV baculovirus contains
multiple copies of a double-stranded DNA virus within a protein coat or occlusion body that can infect a relatively large number of lepidopteran species during their larval stage (Goodman et al. 2001).

For individual loopers to become infected and horizontal disease transmission to occur, the larval host must consume a lethal dose of virus, which often resides on the leaf tissue that larvae consume. Once infected, the virus halts the host’s growth and begins replication within the host. In the final stages of the infection, the host liquefies and the occlusion bodies spill out of the cadaver, which contaminate the leaf tissue on which the now deceased host was feeding. The infection cycle repeats when uninfected larvae consume the newly contaminated leaf tissue (Elderd 2013).

For the experiments, spined soldier bug eggs were obtained from a lab colony maintained on *Trichoplusia ni* Hübner, (Lepidoptera: Noctuidae) (Wittmeyer et al. 2001). Spined soldier bugs that were not part of a fitness experiment were maintained in the lab on a diet of frozen *T. ni* and *Spodoptera frugiperda* Smith, (Lepidoptera: Noctuidae). For all experiments, I used healthy or AcMNPV infected soybean loopers. Virus was amplified using larval hosts and extracted in the lab. Healthy larvae were reared on artificial diet until the fourth instar. Since horizontal transmission requires a vegetative substrate on which to conduct the experiments, I carried out the experiment on soybeans (*Glycine max*) grown from seeds obtained from the USDA-GRIN seed bank using the Gsoy 17 variety. Larvae used in the experiments were ordered as eggs from Benzon Research Inc. (Carlisle, PA).

**Laboratory studies**

The laboratory experiments examined predator preference and the fitness consequences of consuming an infected prey item via IGP. Individual soybean looper larvae were starved for
24 hours and then fed a small cube of artificial diet (Southland Products, Lake Village, AR) with a 3 μl droplet of water containing $10^5$ viral particles. This dose ensures larvae become infected as it is roughly 500 times greater than the lethal dose at which 50% of larvae succumb to the virus, the LD50 (Kunimi et al. 1997). Larvae were used in experiments only if they consumed the entire diet cube to ensure infection.

**Predator Preference.** To test whether spined soldier bugs avoid infected prey, preference experiments were carried out in the laboratory using petri dishes (63 cm$^2$) and moistened filter paper to maintain humidity levels (Appendix Fig B.1). I conducted two types of preference experiments. The first used live, UV powder-dusted fourth-instar soybean loopers while the second used dead, frozen fourth instar soybean loopers. I used 200 adult spined soldier bugs in total (60 for live tests, 140 for dead tests), and 143 of those spined soldier bugs consumed a larva within 24 hours (39 for live tests, 104 for dead tests). Spined soldier bugs that did not consume a larva within 24 hours were not used in this study.

For the first preference experiment, I infected larvae, waited 96 hours, and then dusted one healthy larva and one infected larva with alternating colors of UV fluorescent powder. 96 hours is the longest amount of time where infected caterpillars still active, afterwards they begin to change in appearance and die. I conducted a control test and found that spined soldier bugs do not exhibit preference for or against dusted larvae of any color (Flick, unpublished data). Once larvae were dusted, I placed one healthy and one infected larva in a petri dish. After allowing the larvae to acclimatize for one hour, I placed one soldier bug in each petri dish. I then waited until the soldier bug continuously fed for ten minutes on one larva, and recorded that larva as the preference of the soldier bug. As UV powder dust could be toxic to predators, these spined soldier bugs were not used in fitness experiments.
To test if any differences in preference seen in the first experiment were driven by behavioral traits in the prey, I used frozen healthy and frozen infected larvae in a second experimental trial. Viral load increases exponentially over the course of an infection (Kennedy et al. 2014); therefore, I infected fourth instar larvae and then waited 24, 48, 72, 96, 120, 144, or 168 hours before freezing the infected larvae for a total of seven different treatments. The experiment consisted of placing one healthy frozen larva and one infected frozen larva in each petri dish, waiting two hours for the caterpillars to thaw, and adding an adult spined soldier bug (Fig. B.1). I then waited until the soldier bug continuously fed for ten minutes on one larva, and recorded that larva, healthy or infected, as the soldier bug’s preference. As there were no differences among treatments due to the time since infection ($F_{6,97} = 0.64, P = 0.70$), I pooled those data.

**Predator Fitness.** I used spined soldier bugs to investigate how predator fitness is affected by prey quality. Spined soldier bugs were reared on either frozen healthy prey or frozen infected prey and were reared individually and followed from birth to death. Longevity was calculated as time from adulthood to death and development was calculated as time from first instar to adulthood. Spined soldier bugs were given one cadaver every other day, which is more than adequate to ensure that spined soldier bugs did not die from starvation (Flick unpublished data). For females, I also recorded the number of eggs laid as a measure of fecundity. Additionally, I tested how a mixed diet affects developmental time by feeding spined soldier bugs one infected cadaver followed by one healthy cadaver every other day.

**Disease transmission in the field**

To quantify the consumptive and non-consumptive effects of intraguild predation on pathogen transmission, I manipulated virus density and the presence of adult spined soldier bugs
within experimental and control plots. By manipulating the predator and virus density in the field, I directly test how IGP affects pathogen transmission dynamics. For the experiment, I used a fully factorial, randomized block study design with one bagged soybean plant as a plot. Each plot contained one soybean plant of similar size (approximately five trifoliate leaves), one of four virus (i.e., cadaver) densities (0, 15, 60, and 75 infected, first-instar larvae), one of three spined soldier bug treatments (predator, non-consumptive predator, and no predator), and 30 healthy larvae. To test the non-consumptive effects of intraguild predation on disease transmission, I snipped the proboscis of spined soldier bugs before releasing them in the non-consumptive predator plots. Surgically altering spined soldier bugs so that they will hunt but not eat has been shown to be an effective means for inducing prey behavioral responses without significantly altering predator behavior (Thaler et al. 2012, Hermann and Thaler 2014). The treatments were replicated five times for a total of 60 plots. The field study was carried out at LSU’s South Campus, Baton Rouge, LA.

Newly hatched first instar soybean loopers were infected in the lab with a lethal dose of virus ($10^5$ virus particles) two days prior to being placed on plants. Infected soybean loopers were added to each plot as appropriate (i.e., 0, 15, 60, or 75 infected larvae). Three days later (after infected larvae had died from infection), healthy fourth-instar soybean loopers and the appropriate spined soldier bug treatment (i.e., no soldier bug, soldier bug, or soldier bug with snipped proboscis) were added to each plot. The use of first and fourth instars replicates natural epizootics; whereby recently hatched individuals become infected and stop molting while uninfected larvae grow to third or fourth instars (Elderd 2013). Soybean loopers then fed for three days. At the conclusion of the experiment, I collected surviving soybean loopers and spined soldier bugs. When collected, each soybean looper was placed in an individual one ounce cup
with artificial diet and monitored in the lab for signs of infection. Soybean loopers were reared until death or pupation. Since infected individuals liquefy upon death, baculovirus infections were easily diagnosed. If any doubt as to the cause of death, baculovirus infection was confirmed under a light microscope since the occlusion bodies are quite large (Elderd et al. 2013). To ensure an adequate sample size for the transmission analysis, plots were included only if more than five soybean loopers survived the duration of the experiment.

Data analysis

I analyzed preference tests using a chi-square goodness of fit test for binomial distributions. Differences in development between healthy, infected, and mixed groups were analyzed using the Waller-Duncan Bayesian K ratio procedure using the agricolae package in R (Waller and Duncan 1969, Ruberson and et al. 1991, De Mendiburu 2014). In light of the findings from a meta-analysis that predators generally suffer reduced fitness when feeding on disease-infected prey (Flick et al. 2016), we predicted that soldier bug fitness would be lower when fed infected as compared to uninfected prey. Data were analyzed using Welch’s two sample, one-tailed t-tests.

Given the experimental design of the field study, I can quantify the effects of intraguild predation on transmission dynamics using a series of solved differential equations. If I assume that all larvae are equally susceptible to the pathogen, the change in susceptible individuals over time, \(dS/dt\) is governed by:

\[
\frac{dS}{dt} = -\beta SV.
\]

(1)

where \(dS/dt\) is determined by the transmission rate \(\beta\), the number of susceptible individuals \(S\), and the amount of virus \(V\) or, in this experiment, the number of cadavers in the system. The above equation, which is derived from classic Susceptible-Infected-Recovered (SIR) models
(Elderd and Reilly 2014), can be integrated from the start of the experiment (time 0) to the end of the experiment \( T \). The integrated equation takes the form: 

\[-\ln \left( \frac{S(T)}{S(0)} \right) = \beta V(0) T,\]

which is linear with a slope dictated by the transmission rate, \( \beta \) (Elderd 2018). Here \( S(0) \) and \( V(0) \) are the number of susceptible individuals and the number of cadavers at the beginning of the experiment, respectively. \( S(T) \) is the number of susceptible individuals at the end of the experiment.

However, individuals may be heterogeneous with regards to their susceptibility to the virus such that some individual larvae are more or less susceptible. This variability among individuals causes the relationship between \( \ln[ S(T)/S(0) ] \) to become non-linear. The equation associated with the non-linear dynamics is similar in form to eqn. 1 and takes the following form:

\[
\frac{dS}{dt} = -\bar{\beta} SV \left( \frac{S(t)}{S(0)} \right)^C.
\]  

Here, \( C \) is the coefficient of variation associated with the mean transmission rate, \( \bar{\beta} \). For the non-linear model, the transmission rate is assumed to have a mean \( \bar{\beta} \) and an associated variation (Elderd and Reilly 2014). When integrated from 0 to \( T \), the equation becomes: 

\[-\ln \left( \frac{S(T)}{S(0)} \right) = \frac{1}{C^2} \ln(1 + \beta C^2 V(0) T).\]

As the coefficient of variation goes to zero, the non-linear model’s dynamics behave in a similar manner to the linear model.

In total, I fit 18 candidate models to the data using the integrated forms of eqns. 1 and 2 (Table 1). Models varied based on pathogen transmission dynamics (i.e., classic linear transmission (eqn. 1) vs non-linear heterogeneous (eqn. 2) transmission) and whether or not the plot contained a consumptive predator, a non-consumptive predator, or no predator. To test whether or not there were differences between treatments that contained predators as compared
to no predator treatments, I pooled the data from the consumptive and non-consumptive predator treatments. I also tested viable predators against non-viable predators by pooling the non-consumptive predator and no predator treatments. Given that the field data consisted of binary categories of infected or healthy, I used a binomial error distribution (McCullagh and Nelder 1989) to calculate the log likelihood of the data. I calculated Akaike Information Criteria corrected for small sample sizes (AICc) and used AICc scores to compare ΔAICc scores, AIC weights, and evidence ratios (Table 1) between models (Burnham and Anderson 2003). Given that count data may be prone to over-dispersion, I also calculated the variance inflation factor for the global model. Since the factor was less than 1, I did not need to correct AIC scores (Burnham and Anderson 2003).

I also created bootstrapped mean disease transmission rates for the best model (Fig. 3.4 c) by sampling with replacement from the data. I ran the bootstrap for 10,000 iterations. For the linear model with a single parameter to estimate, I used a golden mean search in R’s optimize function. For the non-linear models, the estimates of the two associated parameters, and C, were derived using the Nelder-Mead method in optim function in R. For the non-linear model, I only included samples that converged following the Nelder-Mead method. The failure rate for convergence was about 20% driven by large differences in the infection of high density treatments such that estimates associated with the coefficient of variation, C, would not converge. All data were analyzed using R software version 3.4.3 (R Core Team 2013).

RESULTS

Laboratory studies

Predator Preference. Predator feeding preference differed depending upon whether or not the prey were alive. When the prey were alive, soldier bugs chose live infected soybean
loopers twice as often compared to live healthy soybean loopers (13 chose healthy, 26 chose infected, \( \chi^2 = 4.33, P = 0.037, n = 39 \)), whereas they had no preference when soybean loopers were dead (49 chose healthy, 55 chose infected, \( \chi^2 = 0.35, P = 0.56, n = 104 \)).

Figure 3.2. The effects of consuming AcMNPV infected soybean loopers compared to healthy soybean loopers on longevity and fecundity of spined soldier bugs. The difference between soldier bugs reared on healthy prey and those reared on infected prey is significant for a) longevity, and not significant for b) fecundity. Error bars represent one standard error; asterisk indicates significant difference.

**Predator Fitness.** While fecundity did not change due to prey infection state/resource quality, other metrics associated with predator fitness increased when the predator fed on healthy prey compared to infected prey. In general, the metrics associated with predator fitness increased when feeding only on healthy prey items as compared to only infected prey. Developmental times of spined soldier bugs reared on infected, healthy, or mixed prey were significantly different (\( P < 0.01 \), Fig. 3.3). Developmental times were 20% longer for predators reared on infected prey; however, there was no difference between predators reared on mixed or healthy diets (Fig. 3.3). Spined soldier bug longevity decreased by 45% when consuming infected soybean loopers compared to healthy soybean loopers (\( t_{10.5} = -3.06, P = 0.006 \), Fig. 3.2).
Table 3.1. The eighteen models considered to assess whether intraguild predation via consumptive and non-consumptive interactions affects pathogen transmission. The data collected were tested by fitting the linear (eqn. 1) or the non-linear heterogeneous (eqn. 2) model to individual treatments or groups of treatments. The number of parameters in the model (k), the Akaike Information Criterion corrected for small sample sizes (AICc), the difference between each model and the AICc best model ($\Delta$AICc) and the weight of evidence for that model (AICc wt) are reported. The final column is the evidence ratio or relative likelihood of each model as compared to the best fit model (Burnham and Anderson 2003). The best model is in bold. Predator is the viable consumptive predator treatment. Non-viable is the non-viable non-consumptive predator and No is the no predator treatment. Treatments analyzed together are denoted by a back slash ('/').

<table>
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<tr>
<th>Model</th>
<th>k</th>
<th>AICc</th>
<th>$\Delta$AICc</th>
<th>AICc wt</th>
<th>Evidence Ratio</th>
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Fecundity was not affected by differences in diet. In terms of fecundity, soldier bugs did not lay significantly more eggs when consuming healthy soybean loopers compared to infected soybean loopers ($t_{5.4} = -1.43, P = 0.10$, Fig. 3.2).

Disease transmission in the field

The addition of a predator clearly had an effect on disease transmission in this community. Given the lack of support for the null models (i.e., models that assume no differences between predator, no predator, and non-consumptive predators), whether assuming the classic linear transmission or heterogeneous non-linear transmission (Table. 3.1). The model with the lowest AICc score and, subsequently, highest AICc weight with 26% of the support, separates the predator treatment from the non-consumptive and no predator treatments, which are grouped together (Fig. 3.4). When non-consumptive predators or no predators are present, the best model accounts for heterogeneity in transmission dynamics but no difference between the two treatments. Thus, there appears to be no effect of the non-consumptive predator on transmission. When consumptive predators are added, transmission dynamics change from the non-linear model, which assumes heterogeneity in transmission, to the linear model. The bootstrapped confidence intervals of the transmission rates overlapped between the predator treatment and the combined no predator and non-consumptive predator treatments; however, the mean disease transmission rate was nearly twice as large for the predator only treatment (Fig. 3.4). Linear transmission with consumptive predators results in a larger number of infected hosts at higher pathogen loads as compared to non-linear transmission (Fig. 3.4).
Figure 3.3. The effects of consuming AcMNPV infected soybean loopers compared to healthy soybean loopers on mean developmental time of juvenile spined soldier bugs. Development units are mean days from 1st instar to adulthood. There is a significant difference among diet types on soldier bug developmental times (P < 0.001). Soldier bugs reared alternatively on healthy soybean loopers and infected soybean loopers responded similarly to those reared on healthy soybean loopers, and both developed faster than soldier bugs reared on infected prey. Error bars represent standard errors. Letters indicate significant differences between groups.

Some models had AICc scores close to that of the best model while other models fared much worse. All models including and below model 11 had ΔAICc values greater than four and would be considered to have “considerably less” support than the remaining models (Burnham and Anderson 2003). The remaining competing models, models 2 through 10, group the treatments in a variety of different ways. Part of the similarity in AICc scores among these models stems from fitting the predator treatment data. When fitting the predator treatment data to the non-linear model, the maximum likelihood estimate of the Coefficient of Variation is quite small (CV = 0.134). As the CV approaches zero, the non-linear model collapses into the linear model and, thus, both the non-linear and linear models predict the same dynamics for the
predator treatment. Still, the model with the lowest AICc is more than 2.5 times as likely as any of the other models considered and, overall the null models are extremely unlikely.

Figure 3.4. The best fit model (choice of linear or non-linear defined by AIC and model selection) of disease transmission of soybean loopers in field experiments. a) Consumptive predators are represented by the classic model (Eq 1) while b) non-consumptive predators and no predators were grouped and fit best by the heterogeneity model (Eq 2). At high virus densities, consumptive predators increase disease transmission. In a) and b) lines represent model estimates and the open circles indicate means from the data, and i is the fraction infected during the experiment. c) is the bootstrapped mean disease transmission rate of the predator only treatment compared to the non-consumptive plus no predator treatments.

DISCUSSION

Predators influence pathogens of their prey and pathogens influence the predators of their hosts. The predator, spined soldier bug, prefers to prey on infected individuals, which can have negative fitness consequences (Figs. 3.2 and 3.3). For the pathogen, consumptive effects associated with depredation increase pathogen transmission due to a decrease in heterogeneity associated with susceptibility between individuals. Non-consumptive effects appear less important (Fig. 3.4 and Table 3.1). Given the above, consumptive effects outweigh non-consumptive effects and help drive pathogen transmission. While the research here has focused on a single round of transmission and depredation, the long-term dynamics may also differ when
comparing a system in which IGP is or is not taken into account (e.g., predator preference; Sieber and Hilker 2011).

From the transmission perspective, predators could help increase pathogen transmission via consumptive effects either by prey processing or defecating pathogen after consuming infected individuals (Down et al. 2004, Caceres et al. 2009). Prey processing by predators feeding on infected prey can increase pathogen transmission by assuring that a greater area of leaf tissue contains a lethal dose of virus or that the virus is more evenly spread across the leaf tissue. Evenly distributed virus as compared to clumped distributions associated with baculovirus-killed cadavers decreases heterogeneity in transmission for gypsy moth larvae (D’Amico et al. 2005). This increases the likelihood that a feeding larva will encounter and consume the virus. For example, avian predators consume infected gypsy moth larvae by throwing the larvae against branches to knock off distasteful hairs, at the same time releasing virus onto otherwise uninoculated leaves (Reilly and Hajek 2012). Pathogen particles can also spread by passing through guts of many predators (Beekman 1980, Down et al. 2004, Reilly and Hajek 2012). In fact, spined soldier bugs pass upwards of $10^8$ baculovirus particles when feeding on infected velvetbean caterpillars (Abbas and Boucias 1984). Given that $7.7 \times 10^4$ particles is the lethal dose at which 50% of fourth instar velvetbean caterpillars become infected (Castro et al. 1997), by defecating virus the predator can be instrumental in spreading viral particles across leaf tissue. Thus, defecation of viable viral particles may play an important role in disease transmission when predators are present (Biever et al. 1982, Young and Yearian 1986). In the experiment, I observed spined soldier bugs defecating on both leaf tissue and the ground. These events may lead to continued transmission and increased soil reservoirs of virus for future epizootics. Soil reservoirs and contaminated leaf tissues are important factors for determining
future baculovirus outbreaks (Thompson and Scott 1979, Fuxa 1982). Thus, rather than culling the sick in terms of the "healthy herds" hypothesis (Packer et al. 2003), the spined soldier bug better fits a system in which a messy eater helps to spread the pathogen and infection rates increase (Caceres et al. 2009).

In support of mechanically spreading virus, spined soldier bugs prefer infected live larvae, suggesting that the virus alters a behavioral aspect of the soybean looper making it easier to capture (Lafferty 1992, Bell et al. 2004). For instance, different lepidopteran species have varying levels of aggressiveness toward predator attack (Marston et al. 1978), and a pathogen may increase the likelihood of its host being consumed by a generalist predator by decreasing the aggressiveness of the prey (e.g., Ruberson and et al. 1991). The predator’s preference for live infected individuals as compared to dead individuals also suggests there is not a chemical or physical cue that soldier bugs use to determine larval quality. Here, the impact of the predator on the prey’s pathogen is positive by spreading pathogen throughout the system.

However, the impact of the pathogen on the prey’s predator is generally negative (Fig. 3.2). The results support previous meta-analytical findings that infected prey represent low quality prey for predators (Flick et al. 2016). This can have important consequences for the long-term dynamics of IGP systems from a theoretical perspective. When prey are distributed in the environment in patches of high quality (healthy) and low quality (infected) prey; this can lead to system stability in classic IGP theory (Holt and Polis 1997, Borer et al. 2007, Sieber and Hilker 2011). This suggests that predator preference in this system may result in long-term stability. Thus, to effectively use IGP theory to understand predator, pathogen, and prey communities, it is important to understand interactions of each community member from an empirical perspective.
The best-fit model from the field study investigating predator effects on pathogen transmission supports consumptive effects over non-consumptive effects and is over twice as likely to better fit the data as the second ranked model (Table 3.1, Fig 3.4). This appears to be driven by a relatively high infection rate of non-consumptive predators at low virus density. Models 3 through 16 have ΔAIC scores of 2.3 to 6.0, suggesting these models do not fit the data as well as the top two models. Finally, the two null models (models 17 and 18) have ΔAIC scores greater than 40, suggesting they fit the data poorly (Table 3.1).

The results, with regards to the effects of infected prey on predator fitness, differed from some past studies. Lee & Fuxa (2000) found that soldier bugs reared on infected caterpillars had similar survival to those reared on healthy caterpillars, while Abbas & Boucias (1984) found that consuming infected prey did not significantly reduce soldier bug developmental times. This may arise from differences in the experimental design. The methods vary from these studies in that I did not feed soldier bugs ad libitum. When soldier bugs are fed ad libitum, differences in nutritional value may be overwhelmed by increased feeding on infected prey; for instance, soldier bugs fed for shorter periods of time on each infected prey compared to each healthy prey (Abbas and Boucias 1984) and consumed significantly more infected prey (Bell et al. 2004). However, other studies that did not take an ad libitum approach found similar results to the own. For example, De Nardo et al. (2001) found that Podisus nigrispinus had severely reduced fitness when consuming one larva of infected prey compared to one larva of healthy prey per day after three generations. Down et al. (2004) found that spined soldier bugs that consumed one infected larva every third day had reduced fitness when compared to one healthy larva every third day.

Previous studies show that non-consumptive effects of the predator are important when considering disease transmission in prey species (Raffel et al. 2010, Rohr et al. 2015). One
possible factor for the lack of non-consumptive effects in the study may be the response exhibited by the prey species when confronted with a predator. Soybean loopers exhibit relatively low aggressiveness toward predators (Marston et al. 1978), and perhaps prey with high predator aggression would show a clear differentiation in disease transmission in the presence of a non-consumptive predator. Fear in the prey species can also increase disease transmission (Ramirez and Snyder 2009). Prey behavior is likely important in determining the diet choice of the predator as predators choose infected prey that are easier to catch (Arthurs and Thomas 2001, Lafferty et al. 2008), even though they experience reduced fitness. Therefore, the magnitude and importance of non-consumptive TMIEs may be determined by the behavioral response or range of behavioral responses displayed by the prey/host.

Given the results, consumptive effects are important to disease transmission. First, predators directly change the proportion of healthy and infected prey items (Packer et al. 2003, Ostfeld and Holt 2004). Second, predators may mechanically spread the virus after consumption (Caceres et al. 2009). For example, in the absence of predators, plants that have been skeletonized by moribund herbivores will serve as a sink for the pathogen, as new hosts are unlikely to explore areas without food. However, predators may find and consume these easy to capture prey items and move to areas with higher densities of uninfected hosts, thus increasing pathogen transmission. Determining the strength of these indirect effects on a broader spatial scale will inform the strength and direction of IGP interactions.

In the community, the intraguild predation interactions are counterintuitive. Predators increase the transmission rate of the virus thus increasing virus fitness. At the same time, predator fitness is reduced when consuming infected prey. Therefore, it appears that the virus is the top predator, the predator is the intermediate predator, and the prey is the resource, in this
IGP community. This result is driven primarily by the spread of virus when predators are present. The strength of effects on predator fitness and pathogen transmission rate will dictate if the system is best described by unidirectional IGP (with either predator or pathogen as top predator) or mutual IGP where predators reduce pathogen transmission while suffering fitness consequences.

By using IGP theory I can better predict the dynamics of integrated pest management (IPM) programs. Models suggest that a generalist predator being added to a host-pathogen system will decrease the likelihood of pathogen persistence in the community (Packer et al. 2003, Ostfeld and Holt 2004, Roy and Holt 2008). Therefore, generalist predators combined with pathogens should result in persistence of the pest over time. However, the results suggest predators and pathogens may have an additive or even stronger effect on controlling their shared prey. Given the above, it would be interesting to test specialist predators, which are often the focus of IPM programs, combined with pathogens which may yield different results (Roy and Holt 2008, Flick et al. 2016).

Increasing evidence points to the need to invoke IGP in a multitude of systems, including those where pathogens play a strong role (Thomas et al. 2006, Borer et al. 2007, Caceres et al. 2009, Rohr et al. 2015). While I focus on the short-term dynamics of a single epizootic, the long-term dynamics remain to be tested empirically. Although theoretical perspectives can help to guide the way. We found no support for non-consumptive effects in the community, though consumptive effects continue to play a major role by either culling the sick and decreasing disease transmission or, as others have shown along with this study, spreading the pathogen and increasing pathogen transmission (Caceres et al. 2009, Duffy et al. 2011). This suggests that
predators exist on a consumptive continuum between reducing and increasing pathogen abundance.
CHAPTER 4.
MODELING INTRAGUILD PREDATION BETWEEN A PREDATOR
AND A PATHOGEN OF A SHARED RESOURCE

INTRODUCTION

Predators and pathogens influence the population dynamics of their prey and hosts, respectively. As these two groups are ubiquitous in nature, understanding the interactions between them will shed light on how natural communities fluctuate over time. Predator removal experiments have found mixed results, with prey populations either increasing or decreasing when predators are removed (Sih et al. 1985, Curtis and Barnes 1994). Pathogens can also control the dynamics of their prey, for example, ruffed grouse populations crash at high densities due to *Leucocytozoon bonasae* (Erickson et al 1949). When combined in a single system where both predators and pathogens exert influence on a single prey/host, pathogens can play an important role in these predator removal experiments, exerting stronger control of their host populations when host density is increased through predator removal (Packer et al. 2003). To understand short-term dynamics, ecologists will often turn toward empirical experiments based in the field or the laboratory. Tracking long-term dynamics from an empirical perspective becomes complicate due to space and time constraints. Thus, ecologists will lean on theoretical tools to understand how interactions drive cycles or co-existence. Often these two methods of exploration intersect such that short-term experiments are used to inform long-term dynamical models.

As seen from an empirical perspective, many important factors influence the ways in which predators and pathogens interact. For example, predators may exhibit preference for healthy prey (Flick et al. 2016), which can decrease the number of available hosts for the pathogen and lead to pathogen burnout. Alternatively, predators can prefer infected prey (Vaughan and Coble 1975, Temple 1987, Lafferty 1992), which can lead to extinction of the
pathogen when predators are unsuitable hosts. Predators can also consume prey and secondarily release pathogen into the environment, increasing the overall pathogen transmission (Cáceres et al. 2009). These interactions demonstrate the importance of considering the way in which predators spread disease in the community.

However, we cannot examine these interactions separately and need to consider them as part of a community module (Holt and Polis 1997) in which the predators and pathogens along with their prey hôsts interact via intraguild predation (IGP). Intraguild predation requires a top predator that consumes both a bottom predator, and a shared resource. The bottom predator, in turn, must be a better competitor for the resource than the top predator (Holt and Polis 1997). Predator-prey communities where a pathogen splits the prey into healthy and infected groups can be classified as a special case of IGP. In communities where the pathogen does not infect the predator, predators and pathogens consume the same resource (i.e., healthy prey) and predators can consume infected prey (see Bairigi et al. (2007) for infected prey that are lethal for predators, and Venurino (2002) for parasites that can infect the predators). Examples of these communities are abundant in agricultural systems, including but not limited to parasitoid and fungus interactions (Powell et al. 1986, Akalach et al. 1992) and predator and fungus interactions (Pell et al. 1997, Roy et al. 1998). Communities made up of infected and healthy prey are well suited for intraguild predation theory.

Besides predator preference for healthy versus infected prey, there are other important factors that influence how pathogens spread in an IGP community that include the predator functional response and the density of predators. Functional responses define the relationship between prey abundance and number of prey captured. A type I functional response is a linear relationship, as prey abundance increases, the number of prey captured increases linearly;
however, in a type III response, as prey abundance increases, prey capture increases non-linearly. Here, we examine the importance of functional responses, predator preference (from strong preference for healthy to strong preference for infected), and the ability of the predator to increase or inhibit disease spread when consuming infected prey. We use a system of differential equations to model healthy prey, infected prey, and pathogen populations under the influence of predation.

First, we consider predator functional response (Fig. 4.1). Generally, there three types of predator responses to prey are considered. Under a Holling type I response, predators consume prey at a constant rate regardless of prey density. In this response, predators can always find prey at low densities, and do not become satiated. This is a fairly simple assumption of predator-prey interactions. A Holling type II response models predators that become satiated at high prey densities. This still assumes that predators can always find prey at low prey densities, which is indicative of some specialist predators. Holling type III predators either have a hard time finding prey at low densities or exhibit prey switching, while still becoming satiated at high prey densities, which is indicative of generalist predators. These different predator functional responses have important consequences on equilibrial dynamics of predator, prey, and pathogen models.

Insect predator-prey communities have shown each of the Holling responses. For example, a type I response fits adult ladybird beetles consuming monarch butterfly eggs (Koch et al. 2003) or the aphid *Rhopalosiphum prunifoliae* (Luo et al. 1987). A type II response fits several different ladybird beetle predators consuming aphids (He et al. 1994, Lee and Kang 2004, Pervez 2005) or ladybird larva consuming monarch butterfly eggs (Koch et al. 2003). It is also a good fit for *Podisus maculiventris* consuming *Spodoptera exigua* at relatively low temperatures (Clercq
A type III response fits well when ladybird beetles consume the aphid *Cinara* sp. (Hu et al. 1989), or when *P. maculiventris* consume *S. exigua* in relatively warm conditions (Clercq 2001). Along with prey species, age class of the predator, and temperature, the plant species can also alter the predator response type; in sweet pepper and eggplant, *Podisus nigrispinus* exhibited a type II response, whereas on tomato, *P. nigrispinus* had a type III response (De Clercq et al. 2000). These empirical studies support the use of functional responses to understand how predators and prey interact.

![Figure 4.1](image)

**Figure 4.1.** Different functional responses of predators. A Holling type I response assumes that predators consume prey in density-independent manner, the more prey there are, the more prey are consumed. A Holling type II response assumes that predators become satiated at high prey densities, predators cannot continue feeding past satiation. The Holling type III response assumes that predators become satiated and exhibit prey switching or have difficulty finding prey at low densities.

While predators can have various effects on the prey density, predators can also inhibit or spread disease in prey populations; on this topic, there are two alternate hypotheses. First, predators are expected to reduce disease transmission by decreasing prey densities or specifically removing infected prey that are easier to capture from the community (healthy herds hypothesis - Packer et al. 2003). Reducing the predators associated with game animals can increase game
animal population densities and secondarily the transmission rate of their pathogens (Millán et al. 2004, Zeman and Benes 2004, Gortázar et al. 2006). Second, predators may actually increase disease transmission when they are messy and release disease from the infected prey into an environment where the pathogen can spread to new hosts (predator spreaders hypothesis - Cáceres et al. 2009). For example, many species of predators defecate infective polyhedral bodies of baculoviruses at biologically meaningful levels after consuming infected prey (e.g., Lautenschlager and Podgwaite 1979, Lautenschlager et al. 1979, Biever et al. 1982). In insects, true predators such as wolf spiders, generally do not have preference; whereas parasitoids avoid infected prey (Caballero et al. 1991, Flick et al. 2016). In communities that exhibit prey defense, predators are more likely to attack infected prey that do not contribute to prey defense (Bate and Hilker 2014). Therefore, predator preference extends the entire continuum from high preference for healthy prey, to no preference, to high preference for infected prey.

Finally, the model is unique in that we explicitly model the pathogen population. This is particularly useful when determining the potential for a new epizootic to occur, especially when using a pathogen for biocontrol. It is vital for land managers to understand the likelihood of new infections based on the amount of pathogen left in the system after an epizootic has run its course. Below we present the model, non-dimensionalize, parameterize the model from the studies and the literature, and examine equilibria and stability via the Jacobian.

Example communities we use throughout are soybean fields with both predators and pathogens present to control crop pests. The empirical work focused on the generalist predator, the spined soldier bug (*Podisus maculiventris*), its lepidopteran prey the soybean looper (*Pseudoplusia includens*), and a lethal baculovirus that only infects lepidoptera (*AcMNPV, Autographa californica Multicapsid Nuclear Polyhedrovirus*). The *AcMNPV* infects susceptible
larvae, which do not transmit the pathogen until the larvae die and liquefy on the plant material (Elderd 2013), which we then refer to as the pathogen or cadaver class. We also found, through meta-analysis, that predators and parasitoids in these communities interact differently with infected prey (Flick et al. 2016), and therefore examine two models, one for specialist predators (HIP1) and one for generalists (HIP3).

**MODEL**

\[
\frac{dH}{dt} = rH \left( \frac{k - H}{k} \right) - \alpha_p HP - \frac{1}{\lambda} \alpha_Q Q f(H, I) \tag{1}
\]

\[
\frac{dI}{dt} = \alpha_p HP - \mu_p I - \lambda \alpha_Q Q g(H, I) \tag{2}
\]

\[
\frac{dP}{dt} = \delta \mu_p I - d_p P + \beta \lambda \alpha_Q Q g(H, I) \tag{3}
\]

Below we examine two models to investigate the role of predator functional response on community dynamics in a healthy prey (H), infected prey (I), and pathogen or cadaver (P) community. Figure 2 shows how these classes interact. Within each model we vary the ability for the predators (Q) to spread pathogen when consuming infected prey.

The first model (HIP1) again considers predators that are relatively long-lived compared to their prey, however, are specialist predators; therefore, we use a Holling type I response, which does not assume predators become satiated and does not assume they have difficulty finding prey at low densities. Since the predators are long lived, we also do not directly model predator dynamics but assume that they are a parameter in the model. The second model (HIP3) assumes that predators are generalists that are long lived and do not fluctuate with their prey. In this way, we do not directly model predators, but only assume a predator abundance that we can change to investigate the effects of predators on their prey and pathogens of their prey. This
method has been used before to model predator, prey, pathogen dynamics (e.g., Packer et al. 2003, Roy and Holt 2008). Generalist predators tend to become satiated at high prey densities and exhibit prey switching at low prey densities, resulting in a Holling type III response (Holling 1959). By examining dynamics using a generalist and specialist framework, we can gain insight into how predatory responses affect long-term host-pathogen dynamics.

Another aspect to consider is the effect of infected prey on predators. Predators can suffer deleterious effects such as reduced fitness when consuming only infected prey (Flick et al. 2016). As we are considering long-lived predators here, we make the simplifying assumption that the reduction in prey quality for the predator is overwhelmed by the difference in generational times between the predator and the prey and thus, treat predators as a parameter, rather than a state variable.

In both models, we assume that healthy prey grow logistically at a rate of \( r \) to a carrying capacity \( k \). Infected prey are created following a mass action function when healthy prey interact with pathogen at a rate of \( \alpha_P \). Infected prey transition into the pathogen class at a rate of \( \mu_P \) and pathogen breaks down in the environment at a rate of \( d_p \). Predators (Q) remove prey with either a Holling type I or Holling type III response, and can spread pathogen at a rate of \( \beta \) when consuming infected prey. We assume predators have variable attack rates on healthy versus infected prey; to account for this, we include a term \( \lambda \) as a proxy for preference. When \( \lambda \) is greater than one, predators prefer infected prey, when it is less than one, predators prefer healthy prey. We explore parameter space of \( \lambda \) between 0.1 (predators attack healthy prey 100 times more often than infected prey) to 10 (predators consume infected prey 100 times more often than healthy prey). The baseline attack rate for predators is \( \alpha_Q \) when \( \lambda \) is equal to one and, thus, the predators exhibit no preference between infected and healthy prey.
Intraguild predation between a predator, a pathogen, and a prey/host. Predators consume both infected and healthy prey. Pathogen particles only infect healthy prey and convert healthy prey into infected prey. Infected prey die and create more pathogen particles. Predators release pathogen back into the environment when consuming infected prey.

**Holling type I**

In the Holling type I model, $f(H,I)$ is defined as simply $H$ and $g(H,I)$ is $I$. This creates a linear, density dependent response. The Holling type I response models the predator functional response where predators can always find prey and do not become satiated. This model is more appropriate for long-lived (compared to their prey), specialist predators.

**Holling type III**

In the Holling type III model, $f(H,I)$ is defined as $\frac{H(H+I)}{a^2+(H+I)^2}$ and $g(H,I)$ is $\frac{I(H+I)}{a^2+(H+I)^2}$. This creates a non-linear, density dependent response. Predation is modeled following a Holling type III functional response on both the healthy and infected prey classes. The shape of the response is governed by $a$. 
In both models we use software R packages deSolve and rootSolve to graph population dynamics and solve equilibria, respectively (Soetaert 2009, Soetaert et al. 2010). Reproducible code is presented in appendix 1. Finally, in both models, we define the total number of prey at equilibrium ($N^*$), the proportion of infected individuals at equilibrium ($\phi^*$), and the reproductive potential of the virus ($R_0$), where $H^*_\text{DFE}$ is the disease free equilibrium of the healthy group, following Roy and Holt 2008:

\[
N^* = I^* + H^*;
\]

\[
\phi^* = I^*/N^*;
\]

\[
R_0 = \frac{H^*_\text{DFE}}{H^*}.
\]

**Parameterization**

Table 4.1. Parameters below are the default values used in figures. Growth rate is the same as used in Roy & Holt (2008). Each infected individual creates one cadaver (or one pathogen). Other values were arbitrarily chosen from within biologically realistic ranges (see parameterization section for details).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Definition</th>
<th>Default Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>R</td>
<td>growth rate of susceptible prey</td>
<td>10</td>
</tr>
<tr>
<td>K</td>
<td>carrying capacity</td>
<td>1000</td>
</tr>
<tr>
<td>$\alpha_P$</td>
<td>infection rate</td>
<td>0.5</td>
</tr>
<tr>
<td>$\alpha_Q$</td>
<td>predation rate</td>
<td>0.2</td>
</tr>
<tr>
<td>Q</td>
<td>predator density</td>
<td>0-10</td>
</tr>
<tr>
<td>a</td>
<td>shape of predator response</td>
<td>10</td>
</tr>
<tr>
<td>$\mu_P$</td>
<td>conversion of infected to pathogen</td>
<td>0.1</td>
</tr>
<tr>
<td>$\lambda$</td>
<td>predator preference</td>
<td>0.1-10</td>
</tr>
<tr>
<td>$\delta$</td>
<td>pathogen created per infected prey</td>
<td>1</td>
</tr>
<tr>
<td>$\delta_P$</td>
<td>pathogen break down rate</td>
<td>0.5</td>
</tr>
<tr>
<td>$\beta$</td>
<td>pathogen created from predation of infected</td>
<td>0, 0.5</td>
</tr>
</tbody>
</table>

Parameters were chosen from the literature or picked randomly from biologically realistic ranges (Table 4.1). The growth rate is the same as used in Roy & Holt (2008). Predation is varied from 100 times more preference for infected prey to 100 times more preference for healthy prey. Each infected individual creates one cadaver (or one pathogen particle). The carrying capacity of
healthy prey is set at 1000, which is often used and recently used as the carrying capacity for insects in vineyards (Silva et al. 2017). A functional response parameter of 10 is used by Cordoleani et al. (2013) to investigate the use of accurate parameters in modeling Holling functional responses. A conversion rate of infected individuals into pathogen equal to 0.1 implies that the pathogen takes ten days to break down a host completely, which is close to the time from infection to death of nuclear polyhedroviruses (e.g., Trang and Chaudhari 2002, Choi et al. 2009, Rios-Velasco et al. 2011).

**Equilibria and stability**

We examine different initial conditions based on the parameterized model. There are generally three equilibria, a trivial equilibrium at (0, 0, 0), a disease free equilibrium at (H*, 0, 0), and an equilibrium with all three states at (H*, I*, P*). Next, we analyze the stability of these equilibria. We use the Jacobian to determine eigenvalues using function stode in the deSolve package of R (R Core 2013). See Table 4.2 for the definition of each type of equilibrium using the eigenvalues. Finally, we plot example populations at and near the equilibrium to show how populations behave over time. The code used to find equilibria, stability, and example populations can be found in Appendix C.

**RESULTS**

Table 4.2. The definitions of stability with respect to the real and imaginary parts of the eigenvalues. The color is with respect to Fig. 4.7.

<table>
<thead>
<tr>
<th>Equilibrium Type</th>
<th>Real Part</th>
<th>Imaginary Part</th>
<th>Color</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stable node</td>
<td>Negative</td>
<td>= 0</td>
<td>Blue</td>
</tr>
<tr>
<td>Stable focus-node</td>
<td>Negative</td>
<td>≠ 0</td>
<td>Green</td>
</tr>
<tr>
<td>Unstable saddle</td>
<td>Mixed</td>
<td>= 0</td>
<td>Yellow</td>
</tr>
<tr>
<td>Unstable saddle-focus</td>
<td>Mixed</td>
<td>≠ 0</td>
<td>Pink</td>
</tr>
<tr>
<td>Unstable node</td>
<td>Positive</td>
<td>= 0</td>
<td>Orange</td>
</tr>
<tr>
<td>Unstable focus-node</td>
<td>Positive</td>
<td>≠ 0</td>
<td>Red</td>
</tr>
</tbody>
</table>
In both models, at low levels of predation, the total abundance of prey is roughly at 1/10th of the carrying capacity. Differences arise as the density of predators increase. In the HIP3 model, the total abundance of prey decreases 0.2 percent, while predation increases from zero to ten (Fig. 4.3). However, in the HIP1 model, the abundance of prey changes 20 percent with the same increase in predator density (Fig. 4.5).

**Holling type III**

In Fig. 4.3, we show various characteristics of the infection model plotted against predator abundance ($Q$) in the Holling type III model (HIP3) when predators do not spread pathogen ($\beta = 0$). When predators exhibit preference for infected prey compared to healthy prey, there are more infected individuals present ($I^*$), more total individuals ($N^*$), a lower proportion of infected individuals to the total ($\phi^*$), and a lower reproductive potential of the pathogen ($R0$). As predator abundance increases, the number and proportion of infected individuals decreases linearly, as well as the reproductive potential of the pathogen (non-linearly), regardless of predator preference. However, when predators prefer infected prey, the total abundance of prey increases; while predators that prefer healthy prey decrease the overall prey abundance, and predators that don’t exhibit preference only slightly decrease overall abundance (Fig. 4.3).

In Fig. 4.4, we show various characteristics of the infection model plotted against predator abundance ($Q$) in HIP3 when predators spread pathogen ($\beta = 0.5$). When predators exhibit preference for infected prey compared to healthy prey, there are fewer infected individuals present ($I^*$), fewer total individuals ($N^*$), and a lower proportion of infected individuals to the total ($\phi$). At low levels of predation, predators that prefer healthy prey lead to a higher reproductive potential of the pathogen, whereas at high levels of predation predators that prefer infected prey lead to a higher reproductive potential of the pathogen ($R0$). As predator
abundance increases, the number of infected prey, total abundance of prey, and the reproductive potential of the pathogen all decrease non-linearly; while the proportion of infected individuals decreases linearly regardless of predator preference (Fig. 4.4).

Figure 4.3. Here, I use the Holling type III model and parameters from Table 1 and $\beta = 0$. Black lines indicate preference for healthy prey (4:1); gray lines indicate no preference, and blue lines indicate preference for infected prey (4:1). In all graphs, the x-axis is the number of predators from zero to ten ($Q$). In a) $I^*$ represents the number of infected individuals; in b) $N^*$ represents the population size of the combined infected and healthy classes; in c) $\phi^*$ represents the proportion of infected individuals in the whole population; in d) $R_0$ indicates the reproductive potential of the pathogen. Above $R_0 = 1$ the pathogen persists, below it goes extinct. Note the scale of the y-axes. While the results are inherently interesting, the differences at these parameter values are biologically meaningless and do not alter community dynamics.

The predator’s ability to spread pathogen does not play an important role in the stability of the community. The HIP3 model is unstable at the disease free equilibria (DFE). When all three species coexist, the equilibria are also unstable (Fig. 4.5, panels a and b). Parameter space that leads to extinction of all three states is unstable when initial populations are either zero, or
relatively large. Populations exhibit point stability at equilibrium, but exhibit boom and bust followed by extinction when perturbed, regardless of whether predators spread pathogen (Figs. 4.6 & 4.7).

Figure 4.4. Here, I recreate Figure 1 from Roy & Holt (2008) using the Holling type III model and parameters from Table 1 and $\beta = 0.5$. Black lines indicate preference for healthy prey (4:1); gray lines indicate no preference, and blue lines indicate preference for infected prey (4:1). In all graphs, the x-axis is the number of predators from zero to ten (Q). In a) $I^*$ represents the number of infected individuals; in b) $N^*$ represents the population size of the combined infected and healthy classes; in c) $\phi^*$ represents the proportion of infected individuals in the whole population; in d) $R0$ indicates the reproductive potential of the pathogen. Above $R0 = 1$ the pathogen persists, below it goes extinct. Note the scale of the y-axes. While the results are inherently interesting, the differences at these parameter values are biologically meaningless and do not alter community dynamics.
Figure 4.5. Stability plots using equilibrial values produced for each model with or without predators spreading pathogen ($\beta$). In all models, when all three states are extinct or in the disease free equilibrium, the equilibria are unstable saddles. The Holling type III model is shown in a) and b) and the Holling type I model is shown in c) and d). Predators do not spread pathogen in a) and c) ($\beta = 0$), and do spread pathogen in b) and d) ($\beta = 0.5$). Predator preference is on the y-axes (increasing $\lambda$ increases preference for infected), and predator density is on the x-axes. When all three states are present in the equilibrium, the models generally exhibit an unstable saddle-focus (pink). However, in the Holling type I model, when predators are abundant and prefer infected prey (but cannot spread pathogen), the equilibria are stable focus-nodes (green). At high levels of predation and strong preference for healthy prey the trivial equilibrium is a stable node (blue). See table 2 for explanation of stability based on eigenvalues.
Figure 4.6. Example populations over time using the Holling type III model from Fig 7 panel a. In both graphs, the black line indicates healthy prey, the red line indicates infected prey, and the green line indicates cadavers. At equilibrium, the population is unchanged over time (left). The right panel is an example of adding or subtracting five individuals from each state, in this case, \((H^*+5; I^*+5; P^*+5)\). This equilibrium is unstable with boom and bust followed by extinction. Parameters are as defined in Table 4.1, with \(Q = 7.99\), \(\lambda = 8.00\), and \(\beta = 0.5\).

Figure 4.7. Example populations over time using the Holling type III model from Fig 7 panel a. In both graphs, the black line indicates healthy prey, the red line indicates infected prey, and the green line indicates cadavers. At equilibrium, the population is unchanged over time (left). The right panel is an example of adding or subtracting five individuals from each state, in this case, \((H^*+5; I^*+5; P^*+5)\). This equilibrium is unstable with boom and bust followed by extinction. Parameters are as defined in Table 4.1, with \(Q = 7.99\), \(\lambda = 8.00\), and \(\beta = 0.5\).
Holling type I

Figure 4.8. We recreate Figure 1 from Roy & Holt (2008) using the Holling type I model and parameters from Table 4.1 and $\beta = 0$. Black lines indicate preference for healthy prey (4:1); gray lines indicate no preference, and blue lines indicate preference for infected prey (4:1). In all graphs, the $x$-axis is the number of predators from zero to ten ($Q$). In a) $I^*$ represents the number of infected individuals; in b) $N^*$ represents the population size of the combined infected and healthy classes; in c) $\phi^*$ represents the proportion of infected individuals in the whole population; in d) $R0$ indicates the reproductive potential of the pathogen. Above $R0 = 1$ the pathogen persists, below it goes extinct.

In Fig. 4.8, we show various characteristics (e.g., the abundance of infected prey) plotted against predator abundance in the Holling type I model (HIP1) when predators do not spread pathogen ($\beta = 0$). When predators exhibit preference for infected prey compared to healthy prey, there are more infected individuals present ($I^*$), more total individuals ($N^*$), a lower proportion of infected individuals to the total ($\phi^*$), and a lower reproductive potential of the pathogen ($R0$). As predator abundance increases, the number and proportion of infected individuals decreases, as well as the
reproductive potential of the pathogen, regardless of predator preference. However, predators that prefer infected prey increase the total abundance of prey while predators that prefer healthy prey decrease the overall prey abundance (Fig. 4.8).

Figure 4.9. We recreate Figure 1 from Roy & Holt (2008) using the Holling type I model and parameters from Table 4.1 and $\beta = 0.5$. Black lines indicate preference for healthy prey (4:1); gray lines indicate no preference, and blue lines indicate preference for infected prey (4:1). In all graphs, the x-axis is the number of predators from zero to ten (Q). In a) $I^*$ represents the number of infected individuals; in b) $N^*$ represents the population size of the combined infected and healthy classes; in c) $\phi^*$ represents the proportion of infected individuals in the whole population; in d) $R0^*$ indicates the reproductive potential of the pathogen. Above $R0 = 1$ the pathogen persists, below it goes extinct.

In Fig. 4.9, we show various characteristics of the infection model plotted against predator abundance in HIP1 when predators spread pathogen ($\beta = 0.5$). When predators exhibit preference for infected prey compared to healthy prey, there are fewer infected individuals present, fewer total individuals, a lower proportion of infected individuals to the total, and a lower reproductive potential of the pathogen. As predator abundance increases, the number and
proportion of infected individuals decreases, the total number of prey decreases, and the reproductive potential of the pathogen decreases, regardless of predator preference (Fig. 4.9).

The predator’s ability to spread pathogen plays an important role in the stability of the community. The HIP1 model is unstable at the DFE. When all three species co-exist with predators capable of spreading disease when consuming infected prey, the equilibria are also unstable (Fig. 4.5, panel c). When predators do not spread pathogen when consuming infected prey and predators prefer infected prey, there exists stable equilibria at high values of predator abundance (Fig. 4.5, panel d). In the HIP1 model with predators that do not spread pathogen, increasing predator preference for infected prey (exacerbated by increasing predator density) increases the equilibrial value of healthy prey (Figs. 4.10 & 4.11). However, when predators can spread pathogen, increasing preference for infected prey drives the healthy prey toward zero, and increases the equilibrial value of infected prey (Fig. 4.12). Except at high values of predator abundance and predator preference for healthy prey, parameter space that leads to extinction of all three states is unstable. Populations exhibit point stability at equilibrium, and either dampening oscillations or boom and bust cycles when perturbed (Figs. 4.13 & 4.14).
Figure 4.10. Plots of population equilibria versus predator preference from the HIP1 model without predator transmission ($\beta = 0$). Across predator densities (greater than 0), increasing predator preference for infected prey reduces the abundance of infected individuals and increases the abundance of healthy individuals. Healthy prey are the dominant state at high predator preference for infected prey.

Figure 4.11. Plots of population equilibria versus predator density from the HIP1 model without predator transmission. Across predator preference for infected prey (greater than 1:1), increasing predator density reduces the abundance of infected individuals and increases the abundance of healthy individuals. Healthy prey are the dominant state at high predator abundance and high preference for infected prey.
Figure 4.12. Plots of population equilibria versus predator preference from the HIP1 model with predator transmission (β = 0.5). Across predator densities (greater than 0), increasing predator preference for infected prey reduces the abundance of infected individuals and increases the abundance of pathogen. Pathogen is the dominant state at high predator preference for infected prey.

Figure 4.13. Example populations over time using the Holling type I model from Fig 4.5 panel c (pink region). In both graphs, the black line indicates healthy prey, the red line indicates infected prey, and the green line indicates cadavers. At equilibrium, the population is unchanged over time (left). The right panel is an example of adding or subtracting five individuals from each state, in this case, (H*+5; I*-5; P*-5). This equilibrium is unstable with boom and bust cycles. Parameters are as defined in Table 4.1, with Q = 4.98, λ = 4.97, and β = 0.
Figure 4.14. Example populations over time using the Holling type I model from Fig 4.5 panel d (pink region). In both graphs, the black line indicates healthy prey, the red line indicates infected prey, and the green line indicates cadavers. At equilibrium, the population is unchanged over time (left). The right panel is an example of adding or subtracting five individuals from each state, in this case, \((H^*+5; I^*; P^*)\). This equilibrium is unstable with boom and bust cycles. Parameters are as defined in Table 4.1, with \(Q = 7.99\), \(\lambda = 8.00\), and \(\beta = 0.5\).

**DISCUSSION**

The models suggest that the functional response of the predator is the overwhelming force driving differences in dynamics in the healthy prey, infected prey, and pathogen classes. In the HIP3 model, the predator parameters are important for determining community stability and composition. When predators do not spread pathogen when consuming infected prey, predator preference for infected prey and density play important roles in community dynamics. Increasing preference for infected prey and predator density both increase the proportion and abundance of healthy prey. When predators do spread pathogen, predator density plays an important role when predators prefer healthy prey. However, when predators prefer infected prey, predator density is less important.
**Holling type III**

In the Holling type III model, parameters associated with predator preference ($\lambda$) and density ($Q$) did not play a large role in community stability (Fig. 4.5, panels a & b). Interestingly, along a gradient from predators that prefer infected prey four to one (Fig. 4.3 blue lines), to predators that prefer healthy prey four to one (Fig. 4.3 black lines), the abundance of infected prey decreases when predators do not spread pathogen. This seems to be caused by a decrease in transmission due to a density-dependent transmission rate. However, the proportion of infected prey to the total increases along the preference gradient. Finally, the differences between low and high predator densities are not biologically meaningful. For example, the reproductive potential of the pathogen (when greater than one the pathogen persists) drops from 1000 to 996 when predators increase from zero to ten and the total number of prey drops from 100.9 to 100.7. However, when values of $Q$ are much larger than realistic, interesting changes in community dynamics occur.

When predators do spread pathogen, the abundance of infected prey and the total abundance of prey patterns switch. That is to say, along a gradient from those that prefer infected prey to those that prefer healthy prey, the abundance of infected prey increases along with the total prey abundance. This switching also occurs in the Holling type I model, lending support to the importance of the ability of predators to spread pathogen. Again, the differences between extreme values of predator densities are not biologically significant. However, when predator density is increased to over 500, differences exist between models where predators do or do not spread pathogen (see Appendix Fig C.1).
Holling type I

In the Holling type I model, parameters associated with predator preference ($\lambda$) and density ($Q$) played a large role in community stability (Fig. 4.5, panels c & d). For example, when predators that prefer infected prey are relatively abundant and do not spread pathogen, the community exhibits a stable focus-node equilibrium (Fig. 4.5 panel d). Similar dynamics are found in a number of natural systems such as among *Agelaius phoeniceus*, the gypsy moth (*Lymantria dispar*), and a baculovirus (Lautenschlager and Podgwaite 1979, Lautenschlager et al. 1979). Larvae exhibit unusual behavior when infected by viruses (van Houte et al. 2012), and this behavior can lead to increased predation of infected compared to healthy individuals (Hoover et al. 2011, Clem and Passarelli 2013). The bird consumes the prey and flies away, thus removing the virus from the community. While supplementing the bird populations would be difficult, farmers could plant trees in or near the fields to increase local diversity (Greenberg et al. 2000, Philpott et al. 2008); which may help avoid population booms.

As in the HIP3 model, along a gradient from predators that prefer infected prey two to one, to predators that prefer healthy prey two to one, the abundances of infected prey and total prey decrease when predators do not spread pathogen when predation is modeled using a Type I functional response (Fig. 4.8). The proportion of infected prey to the total increases along the preference gradient. The reproductive potential of the pathogen increases from predators that prefer infected prey to predators that prefer healthy prey. Unique to this model, the decrease in reproductive potential is nonlinear. These parameters are biologically meaningful as increasing the predator density decreases the total number of prey by 20% when they prefer healthy prey compared to an equal increase in total prey abundance when predators prefer infected prey.
When predators do spread pathogen (Fig. 4.9), the abundance of infected prey and the total abundance of prey patterns switch. That is to say, along a gradient from preferring infected prey to those that prefer healthy prey, the abundance of infected prey increases along with the total prey abundance. However, the relationship with predator abundance switches from linear to non-linear. The effect of predator density on the reproductive potential of the pathogen is also interesting. As predator density increases, the highest reproductive potential switches from the model with preference for healthy prey to the model with preference for infected prey.

Conclusions

We examined four total models, two types of predator response with two types of predator-pathogen interactions (i.e., predators that do and do not spread pathogen). We found that when predators exhibit a Holling type III response, changing various predator related parameters does not play a large role. For example, increasing the predator density had little effect on the community, and changing between predators that spread pathogen and those that do not also played nearly no role. However, in the Holling type I model, predator parameters were important indicators of community stability. When predators do not spread pathogen, areas of parameter space lead to stable equilibria. Correctly quantifying the Holling functional response of predators is key to predicting community dynamics.

Many things influence the functional response of the predators. Biotic factors such as predator grouping behavior or prey refuges can change functional responses (Cosner et al. 1999). Community dynamics may also change predator functional responses, for example, interference or facilitation among predators may occur at varying densities, changing the shape of the response (Soluk 1993), or alternative prey may be available changing a type I or II response into a type III response (Holling 1959, Miller et al. 2006). Abiotic factors can also influence predator
responses. For example, increasing the temperature in a crop pest community can change a Holling type II response to a Holling type III response (De Clercq et al. 2000). Understanding what factors influence the predator response can improve biocontrol efforts, for example, supplementing predators may keep prey below economic thresholds in the early growing season when temperatures are lower, but may lead to boom and bust cycles in warmer temperatures.

Within the Holling type I model, we found important differences between predators that spread pathogen and those that do not. Many factors influence whether a predator can spread a pathogen. Pathogen traits can alter if and where their hosts become prey. For example, pathogens increase the predation of mule deer by mountain lions which consume their prey in kill dens (Krumm et al. 2010), therefore, taking the prey away from the area where the pathogen is likely to infect new hosts. Additionally, baculoviruses cause their hosts to alter their behavior such that they become easier targets to predators (Hoover et al. 2011, Clem and Passarelli 2013); however, recombinant baculoviruses tend to cause their hosts to fall from the plants to the ground (Hoover et al. 1995), where a different suite of predators occurs. Pathogen type, as well as predator type, influence the likelihood of pathogen spread after predation. Podisus maculiventris passes baculovirus during defecation (Biever et al. 1982) and female Apanteles telengai can transmit granulovirus after parasitizing infected hosts (Caballero et al. 1991); however, male parasitoids contain little to no baculovirus after feeding on infected prey (Caballero et al. 1991). Even among parasitoids, the ability to spread pathogen is variable. In the parasitoid Microplitis croceipes, no emergent parasitoids from infected prey were found to contain viral particles (Smith et al. 2000). Directly testing the ability of predators to spread pathogen will determine outcomes from the Holling type I model.
In the current study, we focus only on Holling type III and Holling type I models. However, the results indicate that the most important consideration when modeling a predator, prey, and pathogen community is the predator response function. This is evident from the stability analyses, where the HIP1 model had areas of stable equilibria while the HIP3 model did not. Clearly, using the function best suited for the biology of the system will inform management decisions more than just estimating the associated parameters for the predators such as density and preference. The large amount of parameter space associated with stable or unstable equilibria indicates that the parameters used are not overly sensitive and rough estimation is likely sufficient for desired outcomes.
CHAPTER 5.
DISCUSSION

In my dissertation I examine the interactions in an intraguild predation (IGP) community between predators and pathogens that share a resource. I used a meta-analysis to examine how pathogens negatively influence predator life-history traits, lab studies to expand on the meta-analysis results, field studies to investigate how predators change disease transmission of their prey, and mathematical models to explore the various factors associated with predators that change disease transmission in their prey. In the meta-analysis, I found that predators had reduced fitness when consuming infected prey compared to healthy prey. I found strong support for predators altering disease transmission in their prey in field experiments, and reinforced those results with mathematical models that show predators can alter community dynamics depending on their functional response.

First, in chapter 2., I used meta-analytic methods to complete a comprehensive literature review of predators, prey, and pathogens in agricultural communities. I found that, in general, predators had reduced longevity, fecundity, and survival. I also found that predators preferred healthy prey. Preference was driven by parasitoids that preferred healthy prey, while true predators (i.e., wolf spiders) preferred infected prey. This difference in preference can drive the community that exhibits IGP interactions when predators consume infected prey to a community that is purely competition based when predators avoid infected prey. Tight coevolution, like that between a specialist parasitoid and its host, may drive this preference for healthy prey. In other words, parasitoids that lay eggs in infected hosts that die before the larvae finish development will be selected against. Therefore, selection will favor parasitoids that attack relatively healthy hosts. With respect to true predators, prey have varying levels of aggressiveness (Marston et al. 1978), and infected prey are often easier to capture (Thomas et al. 2006). Therefore, I would
expect predators to prefer infected prey as long as reduced fitness associated with low-quality prey is relatively low compared to the increase in ease of capture, or more are available to overwhelm the reduced quality (Holmes and Bethel 1972, Lafferty 1992, Johnson et al. 2010).

The studies used in this meta-analysis exclusively test differences in life-history parameters associated with predators that consume only infected or only healthy prey. Field conditions will vary by community, but are unlikely to contain only infected prey. Pathogens can alter host movement patterns (Vasconcelos et al. 1996a) which may result in a landscape made up of regions with low-quality and regions with high-quality prey. This kind of habitat complexity may increase long-term stability of an IGP system consisting of predators and pathogens (Janssen et al. 2007, Okuyama 2008). Predators interact with infected prey and healthy prey differently, regardless of predator type. For example, parasitoids will spend more time examining infected prey before parasitization (Jiang et al. 2014) and predators reduce handling time of infected prey compared to healthy prey (Abbas and Boucias 1984). These differences will also play a role in the long-term stability of the community. Therefore, more studies are needed investigating the long-term outcomes of communities of mixed infected and healthy prey. These long-term studies will shed light on the inter-generational effects of predators on disease transmission, the long-term effects of consuming a diet made up of infected and healthy prey, as well as the likelihood of the pathogen going extinct.

In chapter 3., I conducted lab studies to build off the meta-analysis and field studies to explore how predators change disease transmission in their prey. I found that predators (*Podisus maculiventris*) again had reduced life-history parameters when consuming baculovirus infected *Pseudoplusia includens*. Interestingly, I found that this true predator preferred infected prey over healthy prey two to one. This result was contingent on the prey being alive. When prey died
before the preference experiment, predators did not exhibit preference. This suggests that prey behavioral traits make them easier to capture when infected, and predators do not use chemo-sensing to determine prey quality before attacking and consuming them in this community. I also conducted a field study to investigate how predators influence the pathogens of their prey.

Using the same system, I set up a field experiment using soybean plants, *Ps. includens*, virus-infected larvae, and *Po. maculiventris*. I had four virus densities and three predator treatments; plots with predators, plots with predators with no mouthparts, and plots without predators. I surgically removed the proboscis of *Po. maculiventris* in order to test the effects of predator-induced behavioral changes in altering disease dynamics. I found that predators increased disease transmission through consumptive effects only (i.e., plots with predators with no mouthparts had the same level of infection as plots without predators) and that predators changed the heterogeneity of disease transmission. When predators were not present, the best fit model to the data was one that included heterogeneity in susceptibility of prey. However, when consumptive predators were present, the best fit for the data was the model without host heterogeneity in susceptibility. Predators may increase pathogen transmission through prey processing or defecating infective virus after consuming infected prey, thus increasing virus dispersal (Down et al. 2004, Caceres et al. 2009, Reilly and Hajek 2012). Virus dispersal is associated with heterogeneity of pathogen infection such that clumps can increase heterogeneity in transmission compared to evenly distributed virus (D'Amico et al. 2005).

Predator preference for infected or healthy prey and the ability of a predator to spread pathogen are two important aspects that can influence dynamics in intraguild predator, pathogen, and prey communities. In chapter 4., I compare and contrast four models testing which factors are most important in these communities. I start with two separate functional responses of
predators. A Holling type I response models predators that always increase their consumption of prey with an increase in prey abundance. This model does well under certain conditions, but generalist predators often exhibit prey switching at low prey densities and become satiated at high prey densities; in this case, the Holling type III model fits well. Next, I split each model between predators that spread pathogen and predators that do not spread pathogen. I compare these models across a range of predator densities and preference for healthy or preference for infected prey. In the Holling type III model I found that predator preference and ability to spread pathogen do not influence community dynamics. For example, in communities with predators that prefer infected prey compared to those that prefer healthy prey, the prey exhibit boom and bust cycles followed by extinction, regardless if predators can spread pathogens.

However, in the Holling type I model I found important differences between the models. For example, in the model without predators spreading pathogens, I found that the community exhibits stable equilibria when predators prefer infected prey and are relatively abundant. However, when predators spread pathogens, the dynamics always lead to boom and bust cycles in the parameter space I tested. Many biotic and abiotic factors can influence which Holling response fits a community best, from temperature to predator characteristics (De Clercq et al. 2000, Koch et al. 2003). Understanding which type of functional response best describes a predator, and when they exhibit that response will inform management and biocontrol decisions.

In my dissertation I used meta-analytic methods, empirical studies, and a mathematical model to explore the dynamics of predators, pathogens, and their shared prey in intraguild communities. I found that predators are negatively influenced when consuming infected prey compared to healthy prey, may exhibit preference for infected prey, can increase disease transmission while decreasing heterogeneity in the field, and the type of predator functional
response is important to understanding community dynamics. These results lend themselves well to be used in species management, particularly in systems of economic importance, such as coyote, deer, and chronic wasting disease communities or crop fields dominated by pests. These results lend themselves well to be used in species management, particularly in systems of economic importance, such as crop fields dominated by pests or understanding chronic wasting disease dynamics in communities where predators like coyotes are present. These results are also well suited for pollinator communities, where bee larvae consume not only pollen, but yeast that also feed on the pollen (Steffan et al. 2017). Many non-traditional intraguild predation communities exist and a better incorporation of microbes (pathogen or otherwise) into this body of work will push forward our understanding of how ecological interactions affect community dynamics both across space and time.
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Hoch, G., A. Schopf, and J. V. Maddox. 2000. Interactions between an entomopathogenic Microsporidium and the endoparasitoid Glyptapanteles liparidis within their host, the gypsy moth larva. Journal of Invertebrate Pathology 75:59-68.


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parasitoid of the cabbage moth, *Plutella xylostella* (Lepidoptera, Yponomeutidae). Journal of Invertebrate Pathology 74:120-126.


## APPENDIX A. SUPPLEMENTARY MATERIAL FOR CHAPTER 2.

### TRIM AND FILL ANALYSIS OF META-ANALYTIC DATA

Table A.1. Overall mean effect size of each treatment. Trim and fill analyses were used to calculate the number of missing studies and the adjusted mean for each group.

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Mean</th>
<th>Missing Studies</th>
<th>Adjusted Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fecundity</td>
<td>-0.6132</td>
<td>2</td>
<td>-0.4934</td>
</tr>
<tr>
<td>Choice</td>
<td>-1.0171</td>
<td>2</td>
<td>-0.7570</td>
</tr>
<tr>
<td>Longevity</td>
<td>-0.3108</td>
<td>0</td>
<td>-0.3108</td>
</tr>
<tr>
<td>Development</td>
<td>0.0819</td>
<td>1</td>
<td>0.0803</td>
</tr>
<tr>
<td>Survival</td>
<td>-0.6643</td>
<td>0</td>
<td>-0.6643</td>
</tr>
</tbody>
</table>
## MEAN EFFECT SIZES OF PREDATOR BY PATHOGEN TREATMENTS

Table A.2. Mean effect sizes (± 95% confidence interval) for each IG\textsubscript{Pred} (predator and parasitoid) by each IG\textsubscript{Prey} (fungus and virus). Negative values indicate a decrease from the control group while positive values represent an increase from control groups. NA's signify groups that had either 0 or 1 study and thus could not be analyzed separately. Degrees of freedom for each variable are in parentheses.

<table>
<thead>
<tr>
<th>Pathogen</th>
<th>Predator</th>
<th>Parasitoid</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Development</strong></td>
<td><strong>Fungus</strong></td>
<td>1.2423 ± 2.6139 (1)</td>
</tr>
<tr>
<td></td>
<td><strong>Virus</strong></td>
<td>-0.0518 ± 0.0954 (26)</td>
</tr>
<tr>
<td><strong>Longevity</strong></td>
<td><strong>Fungus</strong></td>
<td>NA</td>
</tr>
<tr>
<td></td>
<td><strong>Virus</strong></td>
<td>-0.2331 ± 0.1739 (23)</td>
</tr>
<tr>
<td><strong>Fecundity</strong></td>
<td><strong>Fungus</strong></td>
<td>-0.5383 ± 3.2265 (1)</td>
</tr>
<tr>
<td></td>
<td><strong>Virus</strong></td>
<td>-0.6400 ± 0.6870 (12)</td>
</tr>
<tr>
<td><strong>Survival</strong></td>
<td><strong>Fungus</strong></td>
<td>-5.4701 ± 3.0482 (5)</td>
</tr>
<tr>
<td></td>
<td><strong>Virus</strong></td>
<td>-0.8730 ± 0.7309 (11)</td>
</tr>
<tr>
<td><strong>Choice</strong></td>
<td><strong>Fungus</strong></td>
<td>-0.1232 ± 3.0260 (4)</td>
</tr>
<tr>
<td></td>
<td><strong>Virus</strong></td>
<td>NA</td>
</tr>
</tbody>
</table>
ROSENTHAL’S VALUES FOR META-ANALYTIC DATA

Table A.3. Rosenthal's values ($N_R$) for figures 2 and 3. $N_R$ is the hypothetical number of unpublished studies with null results necessary to create a non-significant result.

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Overall</th>
<th>Predator</th>
<th>Parasitoid</th>
<th>Fungus</th>
<th>Virus</th>
</tr>
</thead>
<tbody>
<tr>
<td>Development</td>
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<td>NA</td>
<td>NA</td>
<td>101</td>
<td>NA</td>
</tr>
<tr>
<td>Longevity</td>
<td>207</td>
<td>70</td>
<td>108</td>
<td>120</td>
<td>65</td>
</tr>
<tr>
<td>Fecundity</td>
<td>320</td>
<td>13</td>
<td>168</td>
<td>NA</td>
<td>262</td>
</tr>
<tr>
<td>Survival</td>
<td>105</td>
<td>202</td>
<td>NA</td>
<td>NA</td>
<td>126</td>
</tr>
<tr>
<td>Choice</td>
<td>148</td>
<td>NA</td>
<td>179</td>
<td>NA</td>
<td>179</td>
</tr>
</tbody>
</table>
### SOURCE OF DATA AND COMMUNITY USED

**TABLE A.4. Authors (year published), predator, pathogen, and resource from studies used in the meta-analysis.**

<table>
<thead>
<tr>
<th>Author (year published)</th>
<th>Predator</th>
<th>Pathogen</th>
<th>Resource</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abbas &amp; Boucias (1984)</td>
<td><em>Podisus maculiventris</em></td>
<td>AgNPV</td>
<td>Anticarsia gemmatalis</td>
</tr>
<tr>
<td>Alma (2007)</td>
<td><em>Dicyphus hesperus</em></td>
<td><em>Paecilomyces fumosoroseus</em></td>
<td>Trialeurodes vaporariorum</td>
</tr>
<tr>
<td>Beegle (1975)</td>
<td><em>Hyposoter exiguae</em></td>
<td>TnNPV</td>
<td><em>Trichoplusia ni</em></td>
</tr>
<tr>
<td>Caballero et al (1990)</td>
<td><em>Apanteles telengai</em></td>
<td>AsGV</td>
<td>Agrotis segetum</td>
</tr>
<tr>
<td>Caballero et al (1990)</td>
<td><em>Campoletis annulata</em></td>
<td>AsGV</td>
<td>Agrotis segetum</td>
</tr>
<tr>
<td>Caballero et al (1991)</td>
<td><em>Apanteles telengai</em></td>
<td>AsGV</td>
<td>Agrotis segetum</td>
</tr>
<tr>
<td>Caballero et al (1991)</td>
<td><em>Campoletis annulata</em></td>
<td>AsGV</td>
<td>Agrotis segetum</td>
</tr>
<tr>
<td>Caballero et al (1991)</td>
<td><em>Aleioodes gasteratus</em></td>
<td>AsGV</td>
<td>Agrotis segetum</td>
</tr>
<tr>
<td>De Nardo et al (2001)</td>
<td><em>Podisus nigrispinus</em></td>
<td>AgNPV</td>
<td>Anticarsia gemmatalis</td>
</tr>
<tr>
<td>Down (2009)</td>
<td><em>Orius laevigatus</em></td>
<td>Lecanicillium longisporum</td>
<td><em>Myzus persicae</em></td>
</tr>
<tr>
<td>Down (2009)</td>
<td><em>Orius laevigatus</em></td>
<td>Lecanicillium longisporum</td>
<td>Frankiniella occidentalis</td>
</tr>
<tr>
<td>Eller et al. (1988)</td>
<td><em>Microplitis croceipes</em></td>
<td>HzNPV</td>
<td><em>Heliotris zea</em></td>
</tr>
<tr>
<td>Escribano et al. (2000b)</td>
<td><em>Campoletis sonorensis</em></td>
<td>SfNPV</td>
<td>Spodoptera frugiperda</td>
</tr>
<tr>
<td>Escribano et al. (2000a)</td>
<td><em>Chelonus insularis</em></td>
<td>SfNPV</td>
<td>Spodoptera frugiperda</td>
</tr>
<tr>
<td>Escribano et al. (2000a)</td>
<td><em>Campoletis sonorensis</em></td>
<td>SfNPV</td>
<td>Spodoptera frugiperda</td>
</tr>
<tr>
<td>Guo et al. (2013)</td>
<td><em>Meteorus pulchricornis</em></td>
<td>SeMNPV</td>
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<tr>
<td>Hoch et al. (2000)</td>
<td><em>Glyptapanteles liparidis</em></td>
<td>Vairimorpha spp.</td>
<td><em>Lymantria dispar</em></td>
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<tr>
<td>Hoch &amp; Schopf (2001)</td>
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<td>Polydnavirus</td>
<td><em>Lymantria dispar</em></td>
</tr>
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<td>Hochberg (1991)</td>
<td><em>Apanteles glomeratus</em></td>
<td>PbGV</td>
<td><em>Pieris brassicae</em></td>
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<td>Jiang et al. (2011)</td>
<td><em>Microplitis pallidipes</em></td>
<td>SeMNPV</td>
<td>Spodoptera exigua</td>
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<tr>
<td>Jiang et al. (2014)</td>
<td><em>Microplitis pallidipes</em></td>
<td>SiNPV</td>
<td>Spodoptera litura</td>
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</table>

*(table cont’d.)*
<table>
<thead>
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<th>Author (year published)</th>
<th>Predator</th>
<th>Pathogen</th>
<th>Resource</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kaya (1970)</td>
<td>Apanteles militaris</td>
<td>PuGV</td>
<td>Pseudaletia unipuncta</td>
</tr>
<tr>
<td>Kaya &amp; Tanada (1972)</td>
<td>Apanteles militaris</td>
<td>TnGV</td>
<td>Trichoplusia ni</td>
</tr>
<tr>
<td>Kaya &amp; Tanada (1972)</td>
<td>Apanteles militaris</td>
<td>PuGV</td>
<td>Spodoptera exigua</td>
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<td>Kringle et al. (1988)</td>
<td>Oxyopes salticus</td>
<td>AgNPV</td>
<td>Anticarsia gemmatalis</td>
</tr>
<tr>
<td>Kyei-Poku &amp; Kunimi (1997)</td>
<td>Cotesia kariyai</td>
<td>Entomopoxvirus</td>
<td>Pseudaletia seperata</td>
</tr>
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<td>Lacey et al. (1997)</td>
<td>Aphelinus asychis</td>
<td>Paecilomyces fumosoroseus</td>
<td>Diuraphis noxia</td>
</tr>
<tr>
<td>Lautenschlager et al. (1979)</td>
<td>Birds</td>
<td>LdNPV</td>
<td>Lymantria dispar</td>
</tr>
<tr>
<td>Levin et al. (1981)</td>
<td>Apanteles glomeratus</td>
<td>PrGV</td>
<td>Pieris rapae</td>
</tr>
<tr>
<td>Li et al. (1999)</td>
<td>Solenopsis invicta</td>
<td>AcMNPV</td>
<td>Spodoptera frugiperda</td>
</tr>
<tr>
<td>Li et al. (1999)</td>
<td>Geocoris punctipes</td>
<td>AcMNPV</td>
<td>Heliothis virenspecs</td>
</tr>
<tr>
<td>Li et al. (1999)</td>
<td>Hippodamia convergens</td>
<td>AcMNPV</td>
<td>Heliothis virenspecs</td>
</tr>
<tr>
<td>Li et al. (1999)</td>
<td>Solenopsis invicta</td>
<td>HZNPV</td>
<td>Heliothis virenspecs</td>
</tr>
<tr>
<td>Li et al. (1999)</td>
<td>Geocoris punctipes</td>
<td>HZNPV</td>
<td>Heliothis virenspecs</td>
</tr>
<tr>
<td>Li et al. (1999)</td>
<td>Hippodamia convergens</td>
<td>HZNPV</td>
<td>Heliothis virenspecs</td>
</tr>
<tr>
<td>Marti &amp; Hamm (1985)</td>
<td>Geocoris punctipes</td>
<td>Vairimorpha spp.</td>
<td>Spodoptera frugiperda</td>
</tr>
<tr>
<td>Matthews (2004)</td>
<td>Meteorus gyrator</td>
<td>LoGV</td>
<td>Lactonia oleracea</td>
</tr>
<tr>
<td>McCutchen et al. (1996)</td>
<td>Microplitis croceipes</td>
<td>AcMNPV</td>
<td>Heliothis virenspecs</td>
</tr>
<tr>
<td>McNitt et al. (1995)</td>
<td>Polistes metricus</td>
<td>AcMNPV</td>
<td>Spodoptera frugiperda</td>
</tr>
<tr>
<td>Mesquita et al. (1997)</td>
<td>Aphelinus asychis</td>
<td>Paecilomyces fumosoroseus</td>
<td>Diuraphis noxia</td>
</tr>
<tr>
<td>Mesquita &amp; Lacey (2001)</td>
<td>Aphelinus asychis</td>
<td>Paecilomyces fumosoroseus</td>
<td>Diuraphis noxia</td>
</tr>
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<td>Nakai et al. (1997)</td>
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<td>AsEPV</td>
<td>Adoxophytes spp.</td>
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<td>Nusawardani et al. (2005)</td>
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<td>AcMNPV</td>
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<tr>
<td>Poprawski et al. (1998)</td>
<td>Hippodamia convergens</td>
<td>Paecilomyces fumosoroseus</td>
<td>Diuraphis noxia</td>
</tr>
<tr>
<td>Perez et al. (2007)</td>
<td>Delphastus pusillos</td>
<td>Lecanicillium lecanii</td>
<td>Bemisia argentifolii</td>
</tr>
<tr>
<td>Poprawski et al. (1998)</td>
<td>Serangium parcesetosum</td>
<td>Beauveria bassiana</td>
<td>Bemisia argentifolii</td>
</tr>
<tr>
<td>Poprawski et al. (1998)</td>
<td>Serangium parcesetosum</td>
<td>Paecilomyces fumosoroseus</td>
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</tr>
<tr>
<td>Author (year published)</td>
<td>Predator</td>
<td>Pathogen</td>
<td>Resource</td>
</tr>
<tr>
<td>------------------------</td>
<td>---------------------------</td>
<td>------------</td>
<td>-----------------------------------</td>
</tr>
<tr>
<td>Ruberson et al. (1991)</td>
<td><em>Nabis roseipennis</em></td>
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<td>Sajap et al. (1999)</td>
<td><em>Sycanus leucomesus</em></td>
<td>SINPV</td>
<td><em>Spodoptera litura</em></td>
</tr>
<tr>
<td>Schuld et al. (1999)</td>
<td><em>Trichogramma chilonis</em></td>
<td>Vairimorpha spp.</td>
<td><em>Plutella xylostella</em></td>
</tr>
<tr>
<td>Simelane et al. (2008)</td>
<td><em>Coccinella septempunctata</em></td>
<td>Neozygites fresenii</td>
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<td>Smith et al. (2000)</td>
<td><em>Microplitis croceipes</em></td>
<td>AcMNPV</td>
<td><em>Heliothis virescens</em></td>
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<td>Stoianova et al. (2007)</td>
<td><em>Euplectrus plathypenae</em></td>
<td>SeMNPV</td>
<td><em>Spodoptera exigua</em></td>
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<td>Stoianova et al. (2007)</td>
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<td>SFNPV</td>
<td><em>Spodoptera frugiperda</em></td>
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<td><em>Harpalus rupes</em></td>
<td>MbMNPV</td>
<td><em>Mamestra brassicae</em></td>
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<td>Vasconcelos et al. (1996b)</td>
<td><em>Pterostichus melanarius</em></td>
<td>MbMNPV</td>
<td><em>Mamestra brassicae</em></td>
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<td>Young &amp; Yearian (1987)</td>
<td><em>Nabis roseipennis</em></td>
<td>AgNPV</td>
<td><em>Anticarsia gemmatalis</em></td>
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<td>HzNPV</td>
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Author's name: Andrew J. Flick

Author's address: Dept of Biological Sciences, Louisiana State University, Baton Rouge, LA 70803, USA

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APPENDIX B. SUPPLEMENTARY MATERIAL FOR CHAPTER 3.

PREFERENCE EXPERIMENT SET-UP

Figure B.1. An example of the preference experiment petri dish with one healthy and one infected soybean looper and one spined soldier bug. Soybean loopers were frozen before the experiment. In this case, the infected soybean looper melted as the spined soldier bug dragged it across the filter paper.
APPENDIX C. SUPPLEMENTARY MATERIAL FOR CHAPTER 4.

REPRODUCIBLE CODE

#Load required packages
require(rootSolve)
require(deSolve)

#Define the two models
#HIP Holling type III
HIP3 <- function (time, y, parms) {
    H <- y[1]
    I <- y[2]
    P <- y[3]
    with(as.list(parms), {
        dHdt <- r * H * ((k-H)/k) - alphaP * H * P - (1/lambda)*alphaQ*Q*((H*(H+I))/(1+a^2*(H+I)^2))
        dIdt <- alphaP * H * P - muP * I - lambda * alphaQ*Q*((I*(H+I))/(1+a^2*(H+I)^2))
        dPdt <- delta * muP * I - dP * P + beta*lambda*alphaQ*Q*((I*(H+I))/(1+a^2*(H+I)^2))
        return(list(c(dHdt, dIdt, dPdt)))
    })
}

#HIP Holling type I
HIP1 <- function (time, y, parms) {
    H <- y[1]
    I <- y[2]
    P <- y[3]
    with(as.list(parms), {
        dHdt <- r * H * ((k-H)/k) - alphaP * H * P - (1/lambda)*alphaQ*Q*H*Q
        dIdt <- alphaP * H * P - muP * I - lambda * alphaQ * I * Q
        dPdt <- delta * muP * I - dP * P + beta * lambda * alphaQ * Q * I
        return(list(c(dHdt, dIdt, dPdt)))
    })
}

#Define ranges for some important parameters (Q - predators
#       LAMBDAM - preference for healthy or infected
Q <- seq(0, 10, length = 250)
LAMBDAM <- seq(0.1, 10, length = 125)

#DETERMINE THE EQUILIBRIA
#Create matrices to save data for the healthy, infected, and predator groups
healthy.data <- matrix(nrow = length(LAMBDAM), ncol = length(Q))
infecte.data <- matrix(nrow = length(LAMBDAM), ncol = length(Q))
pathogen.data <- matrix(nrow = length(LAMBDAM), ncol = length(Q))
# Create a matrix of initial condition vectors
# Trivial equilibrium
# Healthy only
# High and Low H/I/P densities
yinia <- matrix(nrow = 4, ncol = 3)
yinia[1,] <- c(0,0,0)
yinia[2,] <- c(1000,0,0)
yinia[3,] <- c(10,10,10)
yinia[4,] <- c(500,500,500)
# Create lists to save the different healthy, infected, pathogen matrices
list.healthy <- list()
list.infected <- list()
list.pathogen <- list()
#### See which states coexist/go extinct based on initial conditions
# Create a 2x2 graphing window
par(mfrow=c(2,2))
# For loop for each set of initial conditions
### START OF LOOP
for(j in 1:4) {
  # Create the plot space, put a point off the graph for setting it up
  plot(xlim = c(0,max(Q)), ylim = c(0, max(LAMBDA)), x = -10, y = -10,
       xlab = "Q", ylab = "lambda")
  # Loop on LAMBDA
  for(i in 1:length(LAMBDA)) {
    # Set up a vector to save the coexistence results
    # White = extinction
    # Grey = disease free equilibrium (DFE)
    # Black = coexistence
    col.vect <- vector(length = length(Q))
    # Loop on Q
    for(z in 1:length(Q)) {
      # Define the other parameters
      params.1a <- c(k = 1000, beta = 0, dP = 0.5, Q = Q[z], alphaQ = 0.2,
                     muP = 0.1, r = 10, lambda = LAMBDA[i], delta = 1, alphaP = 0.5)
      # Run function stode to find the nearest equilibrium of H/I/P
      stode.out <- stode(func = HIP1, parms = params.1a, yinia[j,], time = 1000, positive = TRUE)$y
      # IF statement to save colors (black, white, or gray) based on stode
      if(stode.out[1] < 0.0001 & stode.out[2] < 0.0001 & stode.out[3] < 0.0001) {#0s or below
        col.vect[z] <- "white"} else if (stode.out[1] > 0.0001 & stode.out[2] < 0.0001 & stode.out[3] < 0.0001) {#healthy only
        col.vect[z] <- "grey"} else {
        col.vect[z] <- "black"} # non-zero equilibrium
      # Save the values for later
      healthy.data[i,z] <- stode.out[1]
      infected.data[i,z] <- stode.out[2]
      pathogen.data[i,z] <- stode.out[3]
# Plot the colors on a graph based on LAMBDA (y-axis) and Q (x-axis)
points(x = Q, y = rep(LAMBDA[i], length(Q)), col = col. vect, cex = 0.1)

# Save the numeric values of the lists created earlier for stability later
list.healthy[[j]] <- healthy.data
list.infected[[j]] <- infected.data
list.pathogen[[j]] <- pathogen.data

### END OF LOOP

### DETERMINE THE STABILITY

# Open a new plotting window
dev.new()
par(mfrow = c(2,2))

### BEGIN LOOP
for(j in 1:4) {
    # Create a vector to save the colors later
    col.mat1 <- vector(length = length(Q))
    plot(xlim = c(0, max(Q)), ylim = c(0, max(LAMBDA)), x = -10, y = -10,
         xlab = "Q", ylab = "lambda")
    for(i in 1:length(LAMBDA)) {
        for(z in 1:length(Q)) {
            params.1a <- c(k = 1000, beta = 0, dP = 0.5, Q = Q[z], alphaQ = 0.2,
                            muP = 0.1, r = 10, lambda = LAMBDA[i], delta = 1, alphaP = 0.5)
            # Create a vector of times
            timesa <- seq(0,1000, by = 1)
            # Calculate the jacobian with function jacobian.full
            jac1 <- jacobian.full(func = HIP1, parms = params.1a, time = timesa,
                                  y = c(list.healthy[[j]][i,z], list.infected[[j]][i,z], list.pathogen[[j]][i,z]))
            # Extract Eigenvalues with function eigen
            eig1 <- eigen(jac1)$values
            # Determine stability based on eigenvalues
            if(Re(eig1)[1] < 0 & Re(eig1)[2] < 0 & Re(eig1)[3] < 0 &
              abs(Im(eig1)[1]) + abs(Im(eig1)[2]) + abs(Im(eig1)[3]) == 0) {
                col.mat1[z] <- "blue"
            } else if (Re(eig1)[1] > 0 & Re(eig1)[2] > 0 & Re(eig1)[3] > 0 &
                        abs(Im(eig1)[1]) + abs(Im(eig1)[2]) + abs(Im(eig1)[3]) == 0) {
                col.mat1[z] <- "orange"
            } else if (abs(Im(eig1)[1]) + abs(Im(eig1)[2]) + abs(Im(eig1)[3]) == 0) {
                col.mat1[z] <- "yellow"
            } else if (Re(eig1)[1] < 0 & Re(eig1)[2] < 0 & Re(eig1)[3] < 0 &
                        abs(Im(eig1)[1]) + abs(Im(eig1)[2]) + abs(Im(eig1)[3]) != 0) {
                col.mat1[z] <- "green"
            } else if (Re(eig1)[1] > 0 & Re(eig1)[2] > 0 & Re(eig1)[3] > 0 &
                        abs(Im(eig1)[1]) + abs(Im(eig1)[2]) + abs(Im(eig1)[3]) != 0) {
                col.mat1[z] <- "red"
            }
        }
    }
    # Plot the colors on a graph based on LAMBDA (y-axis) and Q (x-axis)
    points(x = Q, y = rep(LAMBDA[i], length(Q)), col = col.mat1, cex = 0.1)
}

### END OF LOOP

### DETERMINE THE STABILITY

# Open a new plotting window
dev.new()
col.mat1[z] <- "red"} else {# unstable focus-node
  col.mat1[z] <- "pink"} # unstable saddle-focus

points(x = Q, y = rep(LAMBDA[i], length(Q)), col = col.mat1, cex = 0.2)
#
### END LOOP

# EXAMPLE POPULATION GRAPHS
par(mfrow = c(1,2))
# Define initial conditions
# [j][i][z] is initial conditions (1-4), lambda (0-125), and Q (0-250)
y1 <- list.healthy[[4]][75,200]
y2 <- list.infected[[4]][75,200]
y3 <- list.pathogen[[4]][75,200]
# Define initial conditions
params.1a <- c(k = 1000, beta = 0, dP = 0.5, Q = Q[200], alphaQ = 0.2,
               muP = 0.1, r = 10, lambda = LAMBDA[75], delta = 1, alphaP = 0.5)
timesa <- seq(0,400, by = 1)
# Use function ode to run populations over time
outb <- ode(func = HIP1, parms = params.1a, time = timesa,
            y = c(y1,y2,y3))
# Plot the results
# This will create population size over time
plot(outb[,2], pch = 19, ylab = "Population size")
points(outb[,3], col = "red", pch = 19)
points(outb[,4], col = "green", pch = 19)
y1 <- y1 + 5
y2 <- y2 - 5
y3 <- y3 + 5
# This sometimes needs very small time steps to be accurate/function correctly
timesa <- seq(0,1000, by = 1)
outc <- ode(func = HIP1, parms = params.1a, time = timesa,
            y = c(y1,y2,y3))
plot(outc[,3], pch = 19, ylim = c(0,300), col = "red",
     ylab = "Population size")
points(outc[,2], pch = 19)
points(outa[,4], col = "green", pch = 19)
LARGE VALUES OF PREDATOR DENSITY AND PREFERENCE

Figure C.1. Unrealistically large parameters of the HIP3 model. Predator densities vary from 0 to 1000 and predator preference varies from 100:1 for healthy prey to 1,000,000:1 for infected prey. At these extreme values, the HIP3 model predicts stability when predators can spread pathogen (panel b), but not when they do not spread pathogen (panel a).
VITA

Andrew Flick was born and raised in Waukesha, Wisconsin, in 1989. He attended primary school in the Kettle Moraine School District, where he learned to appreciate nature at a young age. From C is for Camping class in elementary school to advanced biology courses in high school, Andrew seized each opportunity to explore nature. Cross country road trips to explore the National Parks also played a role; sitting in aI of the Grand Canyon and giant redwoods. His passions for ecological research and conservation were formalized during his undergraduate degree at Minnesota State University, Mankato, where he participated in an advanced field ecology course in Wyoming and Montana. He studied the effects of shading on sunscreen production in plants, as well as learned about behavioral patterns of buffalo in Yellowstone National Park. In August 2012, Andrew joined the Elderd lab at Louisiana State University. He focused on the interactions between crop pests and their natural enemies. He also took part in a tropical ecology course through the Organization for Tropical Studies where he studied frog calling patterns, spider and ant movement rates, and blackberry pest damage patterns. Andrew hopes that his experiences up to this point will lead to a career in the private or public sector in charge of a research program teaching the next generation of young scientists.