1-1-2019

Bottom-up trait-mediated indirect effects decrease pathogen transmission in a tritrophic system

Bret D. Elderd
Louisiana State University

Recommended Citation

Elderd, B. (2019). Bottom-up trait-mediated indirect effects decrease pathogen transmission in a tritrophic system. *Ecology, 100* (1) https://doi.org/10.1002/ecy.2551

Bottom-up trait-mediated indirect effects decrease pathogen transmission in a tritrophic system

Bret D. Elderd*

Department of Biological Sciences,
Louisiana State University, Baton Rouge, LA 70803
elderd@lsu.edu

* E-mail: elderd@lsu.edu

Running Title: TMIEs and disease transmission

October 4, 2018

Manuscript received 21 August 2018; revised 21 September 2018; accepted 2 October 2018. Corresponding Editor: John L. Orrock.

Abstract

A plant's induction of secondary defenses helps to decrease herbivore damage by changing resource quality. While these chemical or physical defenses may directly decrease herbivory, they can also have indirect consequences. In a tritrophic system consisting of a plant, an insect herbivore, and an insect pathogen, plant based trait-mediated indirect effects (TMIEs) can alter host-pathogen interactions and, thereby, indirectly affect disease transmission. In a series of field experiments, individual soybean plants (*Glycine max*) were sprayed with either a Jasmonic Acid (JA) solution to trigger induction of plant defenses or a similar control compound. Fall armyworm (*Spodoptera frugiperda*) larvae along with varying amounts of a lethal baculovirus were placed on the plants to measure transmission. Induction of plant defenses decreased viral transmission due to increased population heterogeneity arising from changes in individual susceptibility. The change in susceptibility via TMIEs was driven by a decrease in feeding rates and an increase viral dose needed to infect larvae. While the induction against herbivore attack may decrease herbivory, it can also decrease the efficacy of the herbivore's pathogen potentially to the plant's detriment. While TMIEs have been well-recognized for being driven by top-down forces, bottom-up interactions can dictate community dynamics and, here, epizootic severity.

Keywords: Baculovirus; Bayesian analysis; epizootics; Fall armyworm; Induced plant defenses; *Spodoptera frugiperda*; Variability in transmission rate.

Introduction

In tritrophic systems, trait-mediated indirect effects (TMIEs) occur when changes in a species trait directly alters the interactions between two trophic levels and indirectly affects

the third (Werner and Peacor 2003). From a top-down perspective, non-consumptive effects (NCEs) between predator and prey can initiate this cascade. NCEs happen when the mere presence of a predator changes prey behavior; thereby, affecting the prey's development, growth, or survival (Peckarsky et al. 2008). NCEs can, in fact, be even larger than those dictated by changes due to direct predator consumption of the prey (Bolker et al. 2003; Preisser et al. 2005). In turn, changes in the prey's behavior can have cascading consequences on the resources that the prey consume resulting in a TMIE between the predator and the basal resource in the system. Most work on TMIEs focuses on how predator presence changes prey dynamics via this top-down perspective (e.g., Beckerman et al. 1997; Peckarsky et al. 2008; Schmitz et al. 2016). From a disease ecology perspective, this can lead to changes in infection risk (Buck and Ripple 2017). Yet, increasing evidence shows that bottom-up processes, due to changes in resource type and resource quality via TMIEs, may be equally important for determining transmission dynamics (Elderd et al. 2013; Hall et al. 2009; Tao et al. 2015).

Plants, as basal resources in a tritrophic system, may change the chemical composition of their leaves via induced defenses to decrease herbivore consumption (Karban and Baldwin 1997). The advantage of employing an induced defense is that induction reduces the cost of the defense as compared to constitutive defenses and that increasing variability in the resource can lead to a decline in herbivore performance (Karban et al. 1997). For specialists, chemical defenses, either constitutive or induced, can be sequestered as an anti-predator defense (e.g., Agrawal et al. 2012; Bowers 1980) or detoxified (e.g., Ratzka et al. 2002), which serves little benefit for the plant. While for generalists, changes in resource quality due to induction may decrease herbivore performance, induced plant

defenses can have important indirect effects on upper trophic levels and may change the interaction between a predator and its prey (Kersch-Becker and Thaler 2015; Ohgushi 2012) as well as between a host and its pathogen (Elderd et al. 2013; Hall et al. 2009). Thus, changes in plant resource quality can have cascading consequences for other actors in the system via TMIEs (Ohgushi et al. 2012; Stephens et al. 2017; Werner and Peacor 2003). For instance, insect host-pathogen interactions may be dictated by the resource quality of the plant on which the insect feeds such that resource quality determines whether the insect becomes infected or not (Cory and Hoover 2006; Elderd et al. 2013; Hunter and Schultz 1993).

To determine how changes in resource quality affect host-pathogen interactions, I used a tritrophic system consisting of a lethal insect virus, an herbivore, and a plant variety that varies in its production of secondary chemical defenses (Shikano et al. 2017). Using both field and laboratory experiments, I show that resource quality changes disease transmission by increasing the variability in host susceptibility. This results in a decrease in epizootic severity at higher virus levels when plant defenses have been induced. Thus, while plant defenses may decrease herbivory, they can also decrease pathogen efficacy potentially to the plant's detriment.

Methods

Study system

A species-specific baculovirus, *Spodoptera frugiperda* multicapsid nucleopolyhedrovirus (SfMNPV) represents the upper trophic level in this insect pathogen, herbivore, and plant

system. Baculoviruses are ubiquitous in nature and infect a wide-range of insect species (Miller 1997). Epizootics begin when foliage contaminated with baculovirus occlusion bodies (OBs) are consumed by a susceptible larva (Cory and Myers 2003). The OBs contain multiple virions surrounded by a protein coat, which dissolves in the host midgut. If enough OBs are consumed, a fatal infection occurs. The virus then replicates within the host until the baculovirus triggers the host's liquefaction. While uninfected individuals continue to molt to larger instars, infected individuals do not. Horizontal transmission occurs when OBs are released and contaminate the foliage on which susceptible larvae feed (Elderd 2013). Over time, the virus particles degrade due to UV light exposure.

The polyphagous fall armyworm, which serves as the host for SfMNPV, is a multivoltine migratory species with non-overlapping generations that feeds on a number of different crop plants (Richter et al. 1987; Sparks 1979). Adults annually migrate to the Southern United States in April and May from Florida and Texas, where the species overwinters, and continue northward in subsequent generations. Adult females lay eggs in clusters of up to a few hundred (Sparks 1979). After the eggs hatch, there are six larval instars before pupation (Pitre and Hogg 1983). Outbreaks of the fall armyworm, which have been recorded as early as 1845 (Hinds and Dew 1915), can be quite large and wide-spread and can result in as many as 59 pupae/m^2 (Pair et al. 1991). Additionally, the armyworm/spodoptera complex does substantial damage to soybean and other crops in the Southeast (Musser et al. 2018). The fall armyworm is a particular problem in the soybean fields of Brazil (Peruca et al. 2018). Recently, *S. frugiperda* has been introduced into Africa and is causing widespread damage (Stokstad 2017). For the fall armyworm, SfMNPV

infection rates are quite high, reaching 50-60% in infested areas (Fuxa 1982). Thus, SfMNPV represents an important source of mortality (Richter et al. 1987).

Soybean isolines, the basal resource in this tritrophic system, vary in the amounts of constitutive and induced defenses that they produce (Bi and Felton 1995; Underwood et al. 2000). Feeding by chewing insects on soybean leaves triggers a large number of defense-related changes locally and systemically (Shikano et al. 2017; Underwood et al. 2002). For the fall armyworm, which readily feeds on soybeans (Richter et al. 1987; Sparks 1979), the production of plant defense compounds may have varying effects (Paulillo et al. 2000; Pitre and Hogg 1983). Given the wide-diet breadth of *S. frugiperda*, the herbivore could be considered a generalist and its response to *G. max* defenses may be indicative of a wide-range of species that feed upon the plant. Additionally, larval weight gain on the soybean isoline used in these experiments, Stonewall, declines when the plants have been induced as does leaf area eaten (Shikano et al. 2017). These differences between feeding on induced and non-induced plants were not driven by changes in total phenolics or peroxidase activity. Most likely, the differences in performance and consumption were driven by the chemical composition of the induced leaf tissue (Shikano et al. 2017). Overall, these induced changes affect insects directly by altering feeding behavior, damaging midgut tissues, and interfering with digestive processes (Shikano 2017). These changes may also affect insects indirectly by interacting with baculoviruses in insect midguts (Elderd et al. 2013; Hoover et al. 1998).

Population-level dynamics

The baculovirus transmission cycle governing a single outbreak or epizootic can be described by a series of differential equations (Dwyer et al. 1997; Reilly and Elderd 2014) that track the number of susceptible individuals, the infected individuals, and the pathogen in the system. The equation for the change in susceptible individuals over time t takes the form:

$$\frac{dS}{dt} = -\bar{\beta}\left[\frac{S(t)}{S(0)}\right]^{C^2} SV \ , \tag{1}$$

where $\bar{\beta}$ is the mean transmission rate and C is the coefficient of variation given the mean transmission rate. S and V are the number of susceptibles and the amount of virus in the system, respectively. During an epizootic, the transmission rate $\bar{\beta}$ is scaled by the fraction of susceptibles in the population at time t and the number of susceptibles at the beginning of the epizootic, $[S(t)/S(0)]$. The degree of scaling is determined by C^2. This transmission scaling factor, $[S(t)/S(0)]^{C^2}$, starts at one and declines over time resulting in lower transmission as the epizootic progresses (Elderd and Reilly 2014). The above equation assumes that disease transmission is governed by a distribution with a mean and some variation about the mean. At the beginning of the epizootic, transmission is relatively high as the highly susceptible individuals become infected first and is governed solely by the mean transmission rate $\bar{\beta}$. As the epizootic begins to wane, the only uninfected individuals left have a low susceptibility to the disease and overall transmission drops rapidly or slowly depending upon the degree of heterogeneity in the population (See Fig. 1A in Dwyer et al. 1997). As the coefficient of variation decreases to zero, such that all individuals are equally

susceptible, the above equation becomes $dS/dt = \beta SV$ (Dwyer et al. 1997). For eq. 1, either the mean transmission rate and/or the coefficient of variation can differ given the induction status of the plant. That is, the resource quality of the plant as determined by a trait-mediated induced defense can indirectly affect transmission dynamics.

Eq. 1 can be integrated from time 0 to time T, where T corresponds to either a point of time in the epizootic or the end of an experiment. The integrated equation is:

$$\frac{S(T)}{S(0)} = (1 + C^2\bar{\beta}V(0)T)^{-1/C^2} , \tag{2}$$

where $S(T)/S(0)$ is the fraction of larvae that survive at the end of the experiment and $V(0)$ is the virus-killed cadaver density at the beginning of the experiment. The above equation can be easily fit to field data to estimate both the mean transmission rate $\bar{\beta}$ and the associated coefficient of variation C. If instead, there is no heterogeneity in the system and all individuals are equally susceptible, the corresponding differential equation integrates to $S(T)/S(0) = \exp(-\beta V(0)T)$ and only the transmission rate β needs to be estimated.

To understand how up regulation of plant secondary metabolites affects disease transmission, a series of experiments were conducted that manipulated plant induction status and virus-killed cadaver density. Induction was initiated by spraying either a solution containing 1mM of Jasmonic Acid (JA) dissolved in ethanol or distilled water on a group of similar sized soybean plants from the inducible Stonewall isoline (Underwood et al. 2000, 2002) every other day for one week. The period was chosen to ensure the defenses were induced over the course of the entire field experiment given that levels of defenses in soybeans can decline after 72 hours when exposed to herbivores (Underwood et al. 2000)

and was comparable to other long-term studies on soybeans (Accamando and Cronin 2012). Another group of plants were sprayed with a control solution that did not contain JA. Using herbivores rather than JA to induce the plants would present an additional challenge as it is often difficult to control for the amount of leaf material consumed across replicates (Baldwin 1996; Cipollini et al. 2003). Soybean plants were germinated and grown in a walk-in chamber at 28°C until being translocated to the field (LSU's Burden Center, Baton Rouge, LA). Virus density was manipulated by placing a varying number of infected first instars (0, 10, 20, or 40) on the plant. An infected first instar will produce 1.1 x 10^5 baculovirus occlusion bodies (95% Credible Interval (CI), [0.8 x 10^5, 1.3 x 10^5]) when the instar succumbs to the virus. By placing first instars on the plant rather than spraying virus, the virus would be distributed across the leaf tissue closer to what occurs naturally. To infect the first instars, 9 μL of 10^6 OBs per 3 μL solution were spread across two-ounce cups filled with an artificial diet (Southland Productions, Lake Village, AR). Recently hatched first instar larvae (Benzon Research, Carlisle, PA) were placed on the diet cup and allowed to feed. Infected larvae do not molt to the next instar and, thus, are easily identified. The infected larvae were then placed on soybean plants covered in a mesh bag to ensure that the larvae did not escape. The mesh bag also prevents virus degradation due to UV light exposure (Elderd and Reilly 2014). After four days, during which the first instars had died, 20 healthy fourth-instar larvae were placed on each plant and allowed to feed for an additional four days. The fourth instars were then removed and placed on individual diet cups where they were reared until they had either pupated or died. Recovery varied between replicates due to healthy fourth instars being cannibalized when the mesh bag was opened for recovery. Cannibalism occurred during recovery when the

larvae dropped from the leaves and fell to the bottom of the experimental bag covering the plant. When confined to a small space, the larvae would begin consuming nearby conspecifics. Death due to viral infection was confirmed by either liquefaction of the host or under a light microscope where OBs are visible (Cory and Myers 2003).

A total of 20 replicates were used across all control and infected instar treatments. In the plots where no virus was added, no infected individuals were recovered. Thus, there was no need to correct the data for background infection (Morgan 1992) due to environmental contamination. Additionally, given the lack of infection in the zero virus treatments and that having no virus in the system would result in zero transmission, the estimates for the linear and non-linear models were placed through the origin. The data collected were then fit to the non-linear heterogenous equation (eq. 2) or its linear counterpart. The models (Table 1) tested whether the non-linear or linear transmission model best fit the data depending on plant induction treatment and, thus, whether or not TMIEs via induction of secondary metabolites affected transmission dynamics.

Individual-level dynamics

While the above focuses on the epizootic from a population perspective, it is also useful to examine how TMIEs affect individual probability of infection. In baculovirus-driven systems, an individual larva has to both consume a lethal viral dose and become infected (Dwyer et al. 2005; Elderd et al. 2008) in order for horizontal transmission to occur. This process encompasses the two main components of transmission, which include contact rate and infection probability given contact with the virus (Hawley et al. 2011; McCallum et al. 2017). The probability of consumption and infection can be written as

$P(I, C) = P(I|C)P(C)$ where $P(I, C)$ represents the joint probability of infection I and virus consumption C. $P(C)$ is the probability of consumption and $P(I|C)$ is the conditional probability of infection given consumption. $P(I|C)$ and $P(C)$ essentially break down the transmission process into two separate actions that incorporate individual behavior and physiology.

To test whether the probability of infection given consumption, $P(I|C)$, changes due to plant induction status, a dose-response experiment was performed. In the experiment, a known amount of virus, either 0, 300, 3,000, or 30,000 OBs, was placed on a 9.5 mm diameter leaf disk from either induced or non-induced plants. Thirty recently molted fourth-instar larvae that had been starved for 24 hours were presented with a leaf disk containing one of the experimental viral doses. Only larvae that consumed the entire leaf disk were included to ensure that the larvae received a full dose of the virus and the associated secondary plant metabolites. The number of larvae (mean [standard deviation]) that failed to consume the entire disk did not vary by induction treatment (control discs - 5.0 larvae [0.82]; JA-treated discs (5.8 larvae [0.50])). A Bayesian Poisson t-test using methods outlined in the statistical analysis section showed that there was an increase in the number of individuals that failed to consume the JA-treated leaf disc but that the estimate of this increase was centered around zero given the 95% CIs (0.13, [-0.461,0.763]). After consuming the leaf disk and the virus, the larvae were placed on individual diet cups and reared to pupation or death. Death resulting from a viral infection was confirmed using the same techniques as the field experiment. None of the controls became infected during the course of the dose-response experiment. Thus, there was no need to correct the dose-response data for background infection (Morgan 1992) due to laboratory

contamination. The data were then analyzed using a logit regression (Collett 2003) where either the slope and/or intercept term differed due to induction treatment. In estimating the slope and intercept associated with the logit function only the non-zero dose data were used as is common practice since the logit function is undefined at zero (Collett 2003). The dose-response experiment directly tested whether TMIEs due to resource quality affected the likelihood of transmission given contact between the host and virus.

To quantify the probability of consumption, $P(C)$, a preference test was preformed. Individual fourth-instar larvae were placed in a petri dish with two pre-measured 9.5 mm diameter leaf disks, one of which was from an induced plant and the other from a control plant. The larvae were allowed to feed for up to four hours and the disks were re-measured and amount eaten calculated. All measurements were conducted using ImageJ (Schneider et al. 2012). The analysis of the model associated with fall armyworm preference included a random effect for each individual given the experimental set-up. While the above experiment does not directly measure consumption, it does serve as a proxy for how secondary plant metabolites affect feeding behavior of the host.

Statistical Analysis

All analyses were conducted using a Bayesian framework with vague priors. The individual-level data from the preference test and the dose-response experiment were analyzed using JAGS (Plummer et al. 2003) and the R2JAGS package (Yu-Sung and Masanao 2015). JAGS (Plummer et al. 2003) was also used to test for differences in cannibalism rates in the field between induced and control treatments when recovering the fourth instars since insect cannibalism rates are known to vary depending upon the level of

induced defenses produced by the plant (Orrock et al. 2017). A Poisson t-test with vague priors was used to quantify the difference between the control and induced treatments. For the population-level field tranmission data, a Bayesian Markov chain Monte Carlo (MCMC) was constructed in R (R Core Team 2015). For each of the analyses, three chains were run from different starting points. The first 10,000 draws were removed to account for the transient dynamics at the start of the chain. The remaining 100,000 MCMC draws were retained to ensure precise estimates of the associated parameters (Link and Eaton 2012). After visually inspecting the chains, multiple tests for convergence were used including the Gelman-Rubin and the Hiedelberg-Welch test (Gelman et al. 2014). The chains were then combined to construct a posterior distribution. Afterwards, a posterior predictive check was performed and Bayesian p-values were calculated to determine whether the model being considered fit the data collected (Gelman et al. 1996). Bayesian p-values near 0.50 indicate that the model does a reasonable job of fitting the data considered (Kéry 2010). All of the models considered passed each of the individual tests. The associated Watanabe Akaike Information Criteria (WAIC) and Deviance Information Criterion (DIC) scores were calculated (Hobbs and Hooten 2015). Model comparisons were done by comparing both the WAIC and DIC scores across models. The model with the lowest score represented the best fit model given the data.

Results

The best fit models used to measure population-level dynamics in the field showed that the transmission process differed between induction treatments (Table 1). For the best-fit

model, transmission was non-linear for both induced and non-induced plants due to differences in both mean transmission $\bar{\beta}$ (JA-induced: 0.144 [0.056, 0.374]; Control: 0.004 [0.003, 0.006] – median [95% CIs]) and the heterogeneity parameter C (JA-induced: 4.719 [3.661, 6.131]; Control: 0.007 [0.005, 0.012]). Note that the estimates of the coefficient of variation for the control treatments were extremely low and very close to zero. As C approaches zero, the non-linear fit becomes linear and, in this instance, the non-linear model for the control treatment was essentially linear. The second highest ranking model, which had very similar scores as compared to the best-fit model, also showed that transmission dynamics differed due to induction treatments (Fig. 1). These differences arose from whether the linear or non-linear model was used to fit the data. JA-induced plants showed decidedly non-linear transmission. In contrast, host-pathogen transmission dynamics on control plants were decidedly linear. At lower viral loads on the JA-induced plants, transmission increased more rapidly due to an increase in the transmission rate as is to be expected given the heterogeneity model (Dwyer et al. 1997). However, at higher viral loads, transmission declined due to the increase in population-level heterogeneity arising indirectly from the plant's induction of secondary defenses. When comparing the ratio of percent survival on induced versus non-induced plants, under low viral/cadaver loads survival is less on the induced plant. However, under higher viral loads induction increases survival (Fig. 1C). Overall, host-pathogen transmission and host survival were governed by changes in resource quality due to bottom-up TMIEs.

For the fourth instars recovered at the end of the field experiment, there were no difference in cannibalism rates. The median difference between cannibalism on induced

plants as compared to control plants was positive, albeit rather small, and the 95% Credible Intervals overlapped zero (0.07 [-0.428, 0.587], Appendix S1: Fig. S1).

In terms of individual-level dynamics, there were differences in both the dose-response and preference tests due to the induction treatment (Fig. 2). For the dose response, the best-fit model included differences in the intercept term. Although the second ranked model, which included differences in the slope term, had a relatively similar score (Table 1). The difference in the intercept shifted the curve for JA-induced plants to the right (Fig. 2A and 2B) such that the lethal dose at which 50% of the population would be infected was much higher for larvae fed virus on JA-induced plants than control plants (Fig. 2C). See Fig. S2 for the associated logit plot of the relationship. The general result did not change when taking the second ranked model into account (Fig. S3). The logit model can also be used to calculate a coefficient of variation associated with the dose-response experiment, which can be used as a measure of variability (Elderd et al. 2013). Given the logit model's estimates of the intercepts, there was considerable overlap between treatments in the coefficient of variation for the dose-response experiments (JA-induced: 0.196 [0.147, 0.274]; Control: 0.226 [0.168, 0.323]). In terms of larval preference, fall armyworm larvae clearly preferred to feed on the control leaf disk as compared to the JA-induced leaf disk (Fig. 2D, Table 1). Thus, induction of plant secondary metabolites decreased both the $P(I|C)$ and the $P(C)$ for individual larvae.

Discussion

Trait-mediated indirect effects driven by changes in the basal resource clearly affected transmission dynamics. However, the change in dynamics was not entirely due to changes in the mean transmission rate but arose from changes in the variability about the transmission rate. This led to a decrease in infection risk as viral density increased (Fig. 1). From a direct benefit/cost perspective, there are clear direct benefits to the induced defense as fall armyworm feeding declines when defenses are induced (Shikano et al. 2017). There are also clear costs to induction in terms of biomass and seed production (Accamando and Cronin 2012). The indirect costs stem from increasing herbivore survival in the presence of a lethal virus when viral and herbivore densities are high. At high herbivore densities, increased survival due to a decrease in transmission (Fig. 1) will increase defoliation, which will further exacerbate biomass loss. Given the estimates for the transmission rate and its associated CV along with the fact the 50-60% of the population becomes infected during an epizootic (Fuxa 1982), the estimate of virus load or cadaver number can be readily calculated. The median number of first-instar cadavers needed to result in these infection levels is between 90 and 120, which is well above the point where induction of defenses results in lower infection rates as compared to control plants (Fig. 1C). While the induction of chemical defenses may have a direct positive effect on the plant by decreasing herbivory (Karban and Baldwin 1997), the TMIE by decreasing transmission negatively affects the plant and, thus, the induction of the defense in a tritrophic system may do more harm than good from the plant's perspective.

While TMIEs are increasingly recognized as important for driving species dynamics (Bolker et al. 2003; Bolnick and Preisser 2005), the focus of the research often centers on shifts in the mean population response (Flick et al. 2016) and how responses are driven by top-down forces (Beckerman et al. 1997; Peckarsky et al. 2008). Yet, these effects may not solely impact the mean response but also variability about the mean. For disease transmission, changes in the variability may be driven by either variability in the contact rate or infection risk after contact. While infection risk decreases after induction, there was no measurable change in the CV given the best-fit dose-response model (Table 1, Fig. 2). On the other hand, feeding behavior, which influences contact rate, may be driving the pattern. However, feeding rates were not directly measured in the preference test conducted. Yet others have shown that the feeding rate of fall armyworm declines when soybean defenses in the same isoline used in these experiments are induced (Shikano et al. 2017). Regardless of the exact mechanism, the increase in variability can have important consequences for both the short-term epizootic and, potentially, the long-term population dynamics of the host (Elderd et al. 2013).

For the fall armyworm-soybean system, the field transmission results contrast tranmission dynamics observed in a baculovirus system where the insect host is the gypsy moth (*Lymantria dispar*) and the virus is consumed on JA-induced or non-induced red oak (*Quercus rubra*). For the gypsy moth, baculovirus transmission on non-induced branches resulted in a non-linear model best fitting the data and the JA-induced branches resulted in linear tranmission (Elderd et al. 2013). The difference between the two systems likely arises because the plants involved produce very different defenses when confronted with an herbivore. When induced, oaks increase the production of hydrolyzable tannins (Hunter

and Schultz 1993). Soybeans, in contrast, produce protease inhibitors and peroxidases (Shikano et al. 2017; Underwood et al. 2002). For the gypsy moth, increases in tannins increased leaf consumption (Elderd et al. 2013) as they readily consume leaves that have increased tannin concentrations (Dwyer et al. 2005). An increase in tannins also increased the amount of virus that the larval host needed to consume in order to become infected (Elderd et al. 2013). The increase in consumption increased the probability that a host contacted the pathogen but induction decreased the probability of infection given consumption. For the fall armyworm, induction of soybean defenses decreases larval consumption and interfers with larval weight gain (Shikano et al. 2017). It also increased the amount of virus needed to infect a larva (Fig. 2C). The similar shifts in the dose-response curves may stem from how the different defenses impact infection probability within the gut of the host. Further research needs to be done to pinpoint the exact mechanism. Given the similar shift in dose-response curves in the two systems, the changes in dynamics most likely arises from differences in consumption. The increase in consumption that was observed in the gypsy moth may have ensured that all individuals contacted the virus, thereby reducing or eliminating the inter-individual differences or heterogeneity that result in non-linear transmission. The reduction in consumption that was observed in the fall armyworm (Shikano et al. 2017), which corresponds to the preference data (Fig. 2D), may have increased differences among individuals, thereby strengthening transmission non-linearities. Thus, the indirect effect mediated by the plant's induction status depends upon the defense being induced and the herbivore's response.

With regards to how secondary metabolites change resource quality and, subsequently, affect epizootic severity, a direct measurement of secondary chemical metabolites was not

conducted for this study. However, it is well known that induction of secondary metabolites in soybeans affects the production of foliar phenolics and their composition within the leaf tissue (Shikano et al. 2017). Induction of these defenses can be costly for the plant as it reduces biomass, seed set, and germination (Accamando and Cronin 2012). Indirectly, these secondary metabolites may decrease baculovirus efficacy by interferring with the dissolution of the outer protein coat of the baculovirus, which is needed in order for the infectious viral particles to be released in the insect's midgut (Felton and Duffey 1990). The compounds may also increase the sloughing rate of the midgut along with any infected midgut cells, which decreases the likelihood of a systemic infection occurring (Hoover et al. 2000). In general, the induction of secondary metabolites within the plant's leaf tissue can result in a decrease in infection risk both in the lab and the field.

While the focus of these experiments was on the direct and indirect effects of induced plant defenses on the host-pathogen interactions, there are other avenues by which induced defenses may affect disease transmission. The induction of plant defenses can increase cannibalism rates (Orrock et al. 2017), which, according to theory, may either increase (Sadeh and Rosenheim 2016) or decrease transmission (Van Allen et al. 2017). Since baculoviruses stop infected larvae from molting (Elderd 2013), the pathogen affects the population's size structure, where healthy larvae are much larger than infected larvae (see Fig. 4 in Van Allen et al. 2017). This change in size structure occurs in a number of different parasite systems (Harrison and Hoover 2012). For the fall armyworm, cannibalism rates increase as the difference in size increases (*Van Allen and Elderd, unpublished data*). Since only 10% of size-based cannibalistic encounters between healthy fourth-instar larvae and infected first-instar larvae result in infection, cannibalism in a size-structured

391 population can decrease transmission (Van Allen et al. 2017). On the other hand,
392 cannibalism may allow for the parasite to infect a new host that contains a greater amount
393 of resources than the current host. In this instance, cannibalism benefits the parasite
394 (Sadeh and Rosenheim 2016). Given that induction of plant defenses can change
395 cannibalism rates (Orrock et al. 2017) and can affect disease transmission (Sadeh and
396 Rosenheim 2016; Van Allen et al. 2017), these two processes may well interact to influence
397 transmission in systems where they both arise. In the field experiments reported here,
398 there was no difference in the number of individuals cannibalized between induced and
399 control plants due to cannibalism (Fig. S1). However, cannibalism was not size structured
400 as all individuals were the same instar. How cannibalism affects transmission dynamics by
401 either increasing or decreasing transmission represents a logical avenue to explore
402 experimentally (Orrock et al. 2017; Van Allen et al. 2017).

403 On a larger spatial scale, since induction changes resource quality, it can affect herbivore
404 movement (Underwood et al. 2005) and may even result in hypermobility of the infected
405 host (Goulson 1997; Vasconcelos et al. 1996). This, in turn, may affect the spatial spread of
406 the disease (Dwyer 1992). The potential interaction between induced plant defenses,
407 herbivore movement, and the herbivore's infection status also represents a ripe avenue for
408 further exploration.

409 Due to changes in the secondary metabolites, induced plant chemical defenses indirectly
410 decreased baculovirus efficacy. Therefore, in a tritrophic system, the production of these
411 defenses could indirectly have a deleterious effect on the plant. To determine the
412 magnitude of this effect, future research needs to quantify the demographic costs associated
413 with the TMIE from the plant's perspective. While the research examined the differences

in outcome during a single epizootic, the long-term dynamics and the potential consequences for host population cycles are less apparent but have been shown in other systems (Elderd et al. 2013). These questions not only arise when thinking about temporal issues but also when considering how pathogen-host-resource dynamics are affected at a larger spatial scale as related to changes in the phytochemical landscape (Elderd et al. 2013; Hunter 2016). While the focus on TMIEs often comes from a top-down perspective, the importance of bottom-up interactions dictated by resource quality is clearly evident and can have wide ranging consequences.

Acknowledgments

I would like to thank Kyle McCauley and William Vial for their help in the field and in the lab, Jim Fuxa and Art Richter for providing the initial inoculum of virus used to conduct the experiments, and Michael Garvey along with Matthew Faldyn for providing comments. Additionally, I would like to thank the two anonymous reviewers whose comments and suggestions helped to improve this work. This work was funded by NSF grant 1316334 as part of the joint NSF-NIH-USDA Ecology and Evolution of Infectious Diseases program and by the Louisiana Board of Regents.

References

Accamando, A. K. and J. T. Cronin. 2012. Costs and benefits of jasmonic acid induced responses in soybean. Environmental Entomology, **41**:551–561.

Agrawal, A. A., G. Petschenka, R. A. Bingham, M. G. Weber, and S. Rasmann. 2012. Toxic cardenolides: Chemical ecology and coevolution of specialized plant–herbivore interactions. New Phytologist, **194**:28–45.

Baldwin, I. T. 1996. Methyl jasmonate-induced nicotine production in *Nicotiana attenuata*: Inducing defenses in the field without wounding. Entomologia Experimentalis et Applicata, **80**:213–220.

Beckerman, A. P., M. Uriarte, and O. J. Schmitz. 1997. Experimental evidence for a behavior-mediated trophic cascade in a terrestrial food chain. Proceedings of the National Academy of Sciences of the United States of America, **94**:10735–10738.

Bi, J. L. and G. W. Felton. 1995. Foliar oxidative stress and insect herbivory - primary compounds, secondary metabolites, and reactive oxygen species as components of induced resistance. Journal of Chemical Ecology, **21**:1511–1530.

Bolker, B., M. Holyoak, V. Krivan, L. Rowe, and O. Schmitz. 2003. Connecting theoretical and empirical studies of trait-mediated interactions. Ecology, **84**:1101–1114.

Bolnick, D. I. and E. L. Preisser. 2005. Resource competition modifies the strength of trait-mediated predator-prey interactions: A meta-analysis. Ecology, **86**:2771–2779.

Bowers, M. D. 1980. Unpalatability as a defense strategy of *Euphydryas phaeton* (Lepidoptera: Nymphalidae). Evolution, **34**:586–600.

Buck, J. C. and W. J. Ripple. 2017. Infectious agents trigger trophic cascades. Trends in Ecology & Evolution, **32**:681–694.

Cipollini, D., C. B. Purrington, and J. Bergelson. 2003. Costs of induced responses in plants. Basic and Applied Ecology, **4**:79–89.

Collett, D. 2003. Modelling binary data. Chapman & Hall/CRC, Boca Raton, FL.

Cory, J. S. and K. Hoover. 2006. Plant-mediated effects in insect-pathogen interactions. Trends in Ecology & Evolution, **21**:278–286.

Cory, J. S. and J. H. Myers. 2003. The ecology and evolution of insect baculoviruses. Annual Reviews of Ecology and Systematics, **34**:239–272.

Dwyer, G. 1992. On the spatial spread of insect pathogens - theory and experiment. Ecology, **73**:479–494.

Dwyer, G., J. S. Elkinton, and J. P. Buonaccorsi. 1997. Host heterogeneity in susceptibility and disease dynamics: Tests of a mathematical model. American Naturalist, **150**:685–707.

Dwyer, G., J. Firestone, and T. E. Stevens. 2005. Should models of disease dynamics in herbivorous insects include the effects of variability in host-plant foliage quality? American Naturalist, **165**:16–31.

Elderd, B. D. 2013. Developing models of disease transmission: Insights from the ecology of baculovirus-driven systems. PLoS Pathogens, **9**:e1003372.

Elderd, B. D., J. Dushoff, and G. Dwyer. 2008. Host-pathogen dynamics, natural selection for disease resistance, and forest-defoliator outbreaks. American Naturalist, **172**:829–842.

Elderd, B. D., K. Haynes, B. Rehill, and G. Dwyer. 2013. Interactions between an induced plant defense and a pathogen drive outbreaks of a forest insect. Proceedings of the National Academy of Sciences of the United States of America, **110**:14978–14983.

Elderd, B. D. and J. Reilly. 2014. Warmer temperatures increase disease transmission and outbreak intensity in a host-pathogen system. Journal of Animal Ecology, **83**:838–849.

Felton, G. W. and S. S. Duffey. 1990. Inactivation of baculovirus by quinones formed in insect-damaged plant-tissues. Journal of Chemical Ecology, **16**:1221–1236.

Flick, A. F., M. A. Acevedo, and B. D. Elderd. 2016. The negative effects of pathogen-infected prey on predators: A meta-analysis. Oikos, **125**:1554–1560.

Fuxa, J. R. 1982. Prevalence of viral infections in populations of fall armyworm, *Spodoptera frugiperda*, in Southeastern Louisiana. Environmental Entomology, **11**:239–242.

Gelman, A., J. B. Carlin, H. S. Stern, and D. B. Rubin. 2014. Bayesian data analysis. Taylor & Francis.

Gelman, A., X. L. Meng, and H. Stern. 1996. Posterior predictive assessment of model fitness via realized discrepancies. Statistica Sinica, **6**:733–760.

Goulson, D. 1997. Wipfelkrankheit: Modification of host behaviour during baculoviral infection. Oecologia, **109**:219–228.

Hall, S. R., C. J. Knight, C. R. Becker, M. A. Duffy, A. J. Tessier, and C. E. Caceres. 2009. Quality matters: Resource quality for hosts and the timing of epidemics. Ecology Letters, **12**:118–128.

Harrison, R. and K. Hoover. 2012. Baculoviruses and other occluded insect viruses. In F. Vega and H. Kaya, editors, Insect Pathology. Academic Press.

Hawley, D. M., R. S. Etienne, V. O. Ezenwa, and A. E. Jolles. 2011. Does animal behavior underlie covariation between hosts' exposure to infectious agents and susceptibility to infection? Implications for disease dynamics. Integrative and Comparative Biology, **51**:528–539.

Hinds, W. and J. Dew. 1915. The grass worm or fall army worm. Technical Report Bulletin no. 186, Alabama Agricultural Experiment Station.

Hobbs, N. and M. Hooten. 2015. Bayesian models: A statistical primer for ecologists. Princeton University Press.

Hoover, K., K. T. Kishida, L. A. DiGiorgio, J. Workman, S. A. Alaniz, B. D. Hammock, and S. S. Duffey. 1998. Inhibition of baculoviral disease by plant-mediated peroxidase activity and free radical generation. Journal of Chemical Ecology, **24**:1949–2001.

Hoover, K., J. O. Washburn, and L. E. Volkman. 2000. Midgut-based resistance of *Heliothis virescens* to baculovirus infection mediated by phytochemicals in cotton. Journal of Insect Physiology, **46**:999–1007.

Hunter, M. 2016. The phytochemical landscape: Linking trophic interactions and nutrient dynamics. Princeton University Press, Princeton, NJ.

Hunter, M. D. and J. C. Schultz. 1993. Induced plant defenses breached - phytochemical induction protects an herbivore from disease. Oecologia, **94**:195–203.

Karban, R., A. A. Agrawal, and M. Mangel. 1997. The benefits of induced defenses against herbivores. Ecology, **78**:1351–1355.

Karban, R. and I. T. Baldwin. 1997. Induced responses to herbivory. University of Chicago Press, Chicago, IL, New York.

Kersch-Becker, M. F. and J. S. Thaler. 2015. Plant resistance reduces the strength of consumptive and non-consumptive effects of predators on aphids. Journal of Animal Ecology, **84**:1222–1232.

Kéry, M. 2010. Introduction to WinBUGS for ecologists. Academic Press, Boston, MA.

Link, W. A. and M. J. Eaton. 2012. On thinning of chains in MCMC. Methods in Ecology and Evolution, **3**:112–115.

McCallum, H., A. Fenton, P. J. Hudson, B. Lee, B. Levick, R. Norman, S. E. Perkins, M. Viney, A. J. Wilson, and J. Lello. 2017. Breaking beta: Deconstructing the parasite transmission function. Philosophical Transactions of the Royal Society B-Biological Sciences, **372**:20160084.

Miller, L. K., editor. 1997. Baculoviruses. Kluwer Academic.

Morgan, B. 1992. Analysis of quantal response data. Chapman & Hall, Boca Raton, FL.

Musser, F. R., A. Catchot, S. P. Conley, J. A. Davis, C. DiFonzo, J. Greene, G. M. Lorenz, D. Owens, T. Reed, D. D. Reisig, P. Roberts, T. Royer, N. J. Seiter, S. D. Stewart, S. Taylor, K. Tilmon, and M. O. Way. 2018. 2017 soybean insect losses in the United States. Midsouth Entomologist, **11**:1–23.

Ohgushi, T. 2012. Community consequences of phenotypic plasticity of terrestrial plants: Herbivore-initiated bottom-up trophic cascades. In T. Ohgushi, O. Schmitz, and R. Holt, editors, Trait-mediated indirect interactions: Ecological and evolutionary perspectives, pages 161–185. Oxford University Press.

Ohgushi, T., O. Schmitz, and R. Holt, editors. 2012. Trait-mediated indirect interactions: Ecological and evolutionary perspectives. Oxford University Press.

Orrock, J., B. Connolly, and A. Kitchen. 2017. Induced defences in plants reduce herbivory by increasing cannibalism. Nature Ecology & Evolution, **1**:1205.

Pair, S. D., J. R. Raulston, J. K. Westbrook, W. W. Wolf, and S. D. Adams. 1991. Fall armyworm (Lepidoptera: Noctuidae) outbreak originating in the Lower Rio-Grande Valley, 1989. Florida Entomologist, **74**:200–213.

Paulillo, L. C., A. R. Lopes, P. T. Cristofoletti, J. R. P. Parra, W. R. Terra, and M. C. Silva-Filho. 2000. Changes in midgut endopeptidase activity of *Spodoptera frugiperda* (Lepidoptera: Noctuidae) are responsible for adaptation to soybean proteinase inhibitors. Journal of Economic Entomology, **93**:892–896.

Peckarsky, B. L., P. A. Abrams, D. I. Bolnick, L. M. Dill, J. H. Grabowski, B. Luttbeg, J. L. Orrock, S. D. Peacor, E. L. Preisser, O. J. Schmitz, and G. C. Trussell. 2008.

Revisiting the classics: Considering nonconsumptive effects in textbook examples of predator-prey interactions. Ecology, **89**:2416–2425.

Peruca, R. D., R. G. Coelho, G. G. da Silva, H. Pistori, L. M. Ravaglia, A. R. Roel, and G. B. Alcantara. 2018. Impacts of soybean-induced defenses on *Spodoptera frugiperda* (Lepidoptera: Noctuidae) development. Arthropod-Plant Interactions, **12**:257–266.

Pitre, H. N. and D. B. Hogg. 1983. Development of the fall armyworm (Lepidoptera, Noctuidae) on cotton, soybean and corn. Journal of the Georgia Entomological Society, **18**:182–187.

Plummer, M. et al. 2003. JAGS: A program for analysis of Bayesian graphical models using Gibbs sampling. In Proceedings of the 3rd international workshop on distributed statistical computing, volume 124, page 125. Technische Universit at Wien Wien, Austria.

Preisser, E. L., D. I. Bolnick, and M. F. Benard. 2005. Scared to death? The effects of intimidation and consumption in predator–prey interactions. Ecology, **86**:501–509.

R Core Team. 2015. R: A language and environment for statistical computing. R Foundation for Statistical Computing, Vienna, Austria.

Ratzka, A., H. Vogel, D. J. Kliebenstein, T. Mitchell-Olds, and J. Kroymann. 2002. Disarming the mustard oil bomb. Proceedings of the National Academy of Sciences, **99**:11223–11228.

Reilly, J. R. and B. D. Elderd. 2014. Effects of biological control on long-term population dynamics: Identifying unexpected outcomes. Journal of Applied Ecology, **51**:90–101.

Richter, A. R., J. R. Fuxa, and M. Abdelfattah. 1987. Effect of host plant on the susceptibility of *Spodoptera frugiperda* (Lepidoptera, Noctuidae) to a nuclear polyhedrosis virus. Environmental Entomology, **16**:1004–1006.

Sadeh, A. and J. A. Rosenheim. 2016. Cannibalism amplifies the spread of vertically transmitted pathogens. Ecology, **97**:1994–2002.

Schmitz, O. J., A. E. Rosenblatt, and M. Smylie. 2016. Temperature dependence of predation stress and the nutritional ecology of a generalist herbivore. Ecology, **97**:3119–3130.

Schneider, C. A., W. S. Rasband, and K. W. Eliceiri. 2012. NIH Image to ImageJ: 25 years of image analysis. Nature methods, **9**:671–675.

Shikano, I. 2017. Evolutionary ecology of multitrophic interactions between plants, insect herbivores and entomopathogens. Journal of Chemical Ecology, **43**:586–598.

Shikano, I., K. L. Shumaker, M. Peiffer, G. W. Felton, and K. Hoover. 2017. Plant-mediated effects on an insect–pathogen interaction vary with intraspecific genetic variation in plant defences. Oecologia, **183**:1121–1134.

Sparks, A. N. 1979. Review of the biology of the fall armyworm (Lepidoptera, Noctuidae). Florida Entomologist, **62**:82–87.

Stephens, J. P., K. A. Altman, K. A. Berven, S. D. Tiegs, and T. R. Raffel. 2017. Bottom-up and trait-mediated effects of resource quality on amphibian parasitism. Journal of Animal Ecology, **86**:305–315.

Stokstad, E. 2017. New crop pest takes Africa at lightning speed. Science, **356**:473–474.

Tao, L., C. D. Gowler, A. Ahmad, M. D. Hunter, and J. C. de Roode. 2015. Disease ecology across soil boundaries: Effects of below-ground fungi on above-ground host-parasite interactions. Proceedings of the Royal Society B-Biological Sciences, **282**.

Underwood, N., K. Anderson, and B. D. Inouye. 2005. Induced vs. constitutive resistance and the spatial distribution of insect herbivores among plants. Ecology, **86**:594–602.

Underwood, N., W. Morris, K. Gross, and J. R. Lockwood. 2000. Induced resistance to mexican bean beetles in soybean: Variation among genotypes and lack of correlation with constitutive resistance. Oecologia, **122**:83–89.

Underwood, N., M. Rausher, and W. Cook. 2002. Bioassay versus chemical assay: Measuring the impact of induced and constitutive resistance on herbivores in the field. Oecologia, **131**:211–219.

Van Allen, B. G., F. P. Dillemuth, A. J. Flick, M. J. Faldyn, D. R. Clark, V. H. W. Rudolf, and B. D. Elderd. 2017. Cannibalism and infectious disease: Friend or foe? American Naturalist, **190**:299–312.

Vasconcelos, S. D., J. S. Cory, K. R. Wilson, S. M. Sait, and R. S. Hails. 1996. Modified behavior in Baculovirus-infected lepidopteran larvae and its impact on the spatial distribution of inoculum. Biological Control, **7**:299–306.

Werner, E. and S. Peacor. 2003. A review of trait-mediated indirect interactions in ecological communities. Ecology, **84**:1083–1100.

Yu-Sung, S. and Y. Masanao. 2015. R2jags: Using R to Run 'JAGS'. R package version 0.5-7.

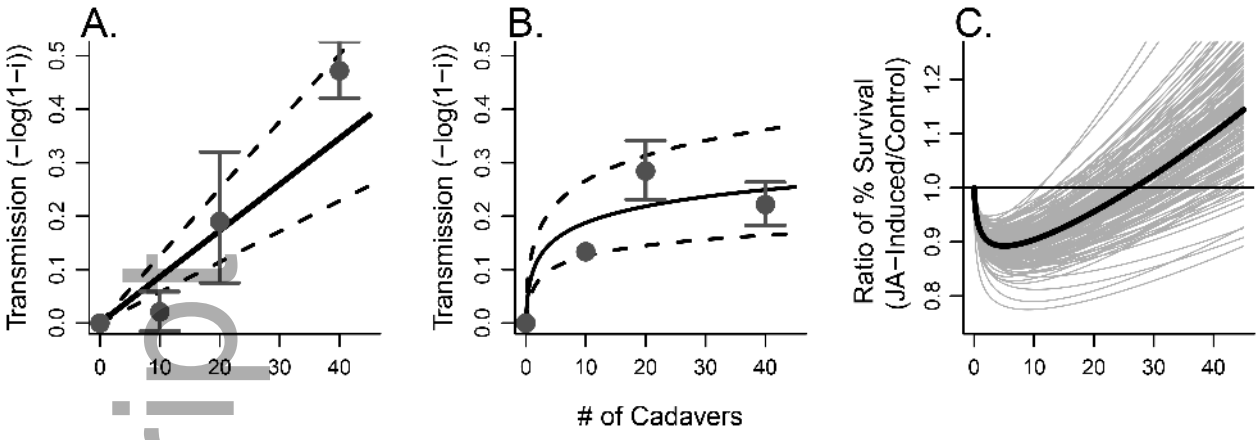
Data Availability

Data are available from the Dryad Digital Repository: https://doi.org/10.5061/dryad.p9d27nh

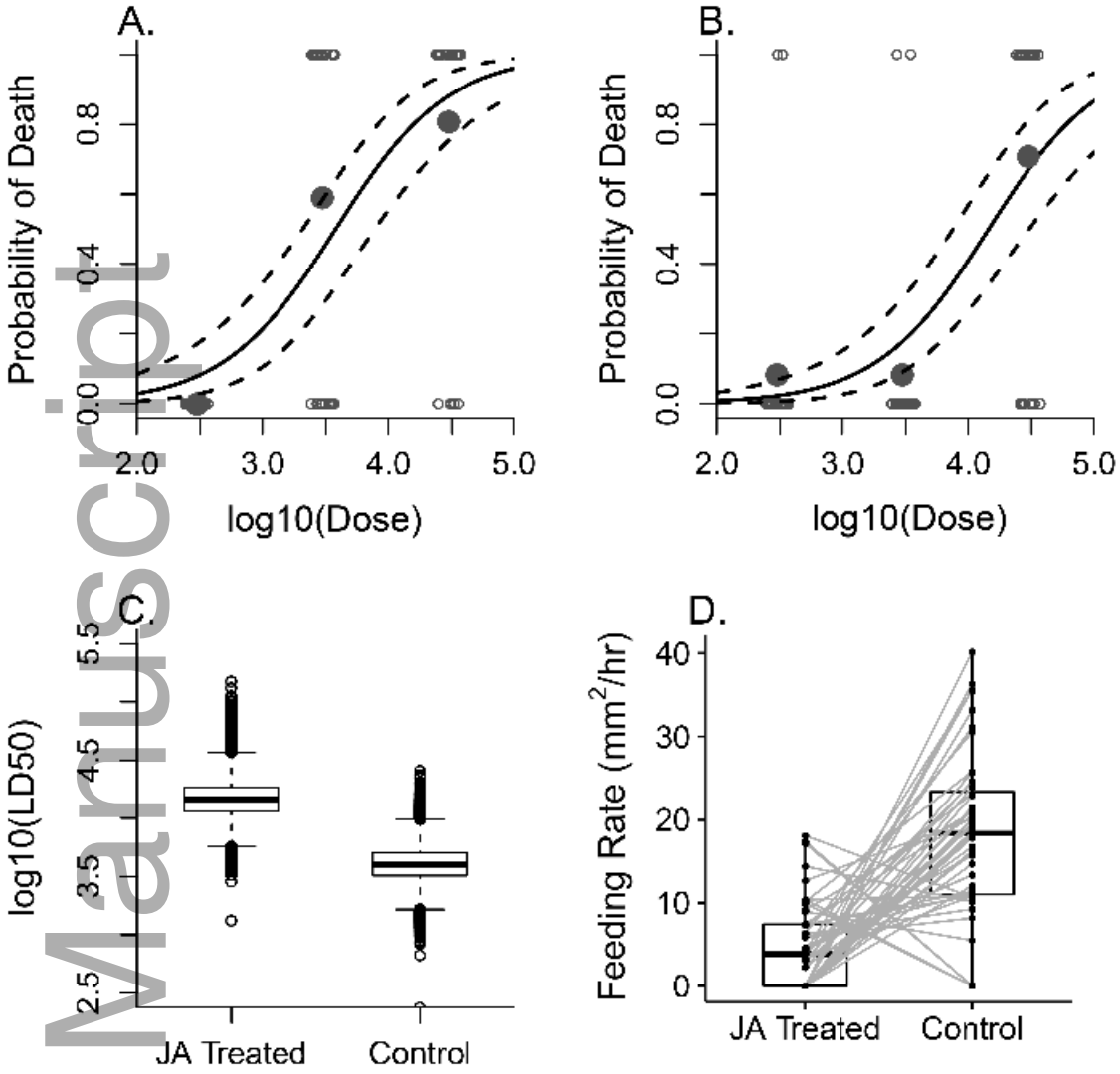
Table 1. WAIC and DIC values for the six models considered for the field transmission data, the four models for the dose-response experiments, and the two models for the preference experiment.

Model	WAIC values	DIC values
Field transmission experiment		
1. Linear, no difference	48.98	49.09
2. Non-linear, no difference	48.96	49.11
3. Both linear	50.71	50.71
4. Both non-linear	**46.74**	**47.33**
5. JA non-linear, control linear	**46.76**	**47.34**
6. JA linear, control non-linear	50.69	50.70
Dose response experiment		
1. No difference	134.40	140.51
2. Intercept only differs	**129.43**	**129.33**
3. Slope only differs	129.59	131.42
4. Both intercept and slope differ	132.16	133.18
Preference experiment		
1. No difference	765.72	763.31
2. Treatment difference	**722.38**	**718.84**

Notes: For the field transmission data, the two best-fit models are in bold. Non-linear and linear refer to the solved heterogenous equation (eq. 2) and its linear counterpart that assumes that individuals do not differ in susceptibility. The first two models for the field transmission data considered assume no difference in JA-induced and control plants. For the dose-response experiments, the four models considered include a null model of no differences and three models where either the intercept and/or the slope differs between JA-induced and control plants. The best-fit model is highlighted for the dose-response and preference experiments.



ecy_2551_f1.tif



ecy_2551_f2.tif