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COMMUNITY ECOLOGY – ORIGINAL RESEARCH

Viral transmission and infection prevalence in a cannibalistic host–pathogen system

Benjamin G. Van Allen[1] · Forrest Dillemuth[2] · Vanja Dukic[3] · Bret D. Elderd[2]

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Abstract

Cannibalism, while prevalent in the natural world, is often viewed as detrimental to a cannibal's health, especially when they consume pathogen-infected conspecifics. The argument stems from the idea that cannibalizing infected individuals increases the chance of coming into contact with a pathogen and subsequently becoming infected. Using an insect pest, the fall armyworm (*Spodoptera frugiperda*), that readily cannibalizes at the larval stage and its lethal pathogen, we experimentally examined how cannibalism affects viral transmission at both an individual and population level. Prior to death, the pathogen in the system stops the larval host from growing, resulting in infected individuals being smaller than healthy individuals. This leads to size-structured cannibalism of infected individuals with the larger healthy larvae consuming the smaller infected larvae, which is commonly observed. At the individual level, we show that the probability of cannibalism is relatively high for both infected and uninfected individuals especially when the cannibal is larger than the victim. However, the probability of the cannibal becoming infected given that a pathogen-infected individual has been cannibalized is relatively low. On a population level, when cannibalism is allowed to occur transmission rates decline. Additionally, by cannibalizing infected larvae, cannibals lower the infection risk for non-cannibals. Thus, cannibalism can decrease infection prevalence and, therefore, may not be as deleterious as once thought. Under certain circumstances, cannibalizing infected individuals, from the uninfected host's perspective, may even be advantageous, as one obtains a meal and decreases competition for resources with little chance of becoming infected.

Keywords Baculovirus · Host–pathogen interactions · Fall armyworm · Size-structured cannibalism · *Spodoptera frugiperda*

Introduction

Arguments that cannibalism, which is highly prevalent (Fox 1975; Polis 1981; Richardson et al. 2010), is individually beneficial or deleterious tend to be based on a standard set of advantages and disadvantages for the cannibal. The positive benefits often focus on decreased competition and nutrition. The positive effect of nutrition derives from the fact that the victim represents an ideal food resource for the cannibal since the victim has similar stoichiometric ratios and essential nutrients (Mayntz and Toft 2006; Polis 1981). The negative aspects of cannibalism invoke arguments related to potential harm that might arise due to the victim fighting back or due to the cannibal becoming infected after consuming a parasite- or pathogen-laden victim (Pfennig 1997, 2000). Out of all the arguments, the potential negative effects associated with disease transmission often take precedence. Yet, a number of theoretical studies have shown that these potential negative effects may be exaggerated (Bolker et al. 2008; Rudolf and Antonovics 2007; Van Allen et al. 2017, but see Sadeh and Rosenheim (2016)). However, there have been few empirical studies directly examining how cannibalism affects pathogen transmission (See Table 1 in Van Allen et al. (2017)). Thus, there is a recognized gap in empirical research (Richardson et al. 2010) needed to resolve whether or not the negative consequences associated with

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✉ Bret D. Elderd
elderd@lsu.edu

[1] University of California, San Diego, CA 92093, USA

[2] Louisiana State University, Baton Rouge, LA 70803, USA

[3] University of Colorado, Boulder, CO 80309, USA

pathogen consumption are truly deleterious to the cannibal and whether or not cannibalism enhances disease spread.

While no disease can spread solely via cannibalism where a single cannibal feeds on a single victim (Rudolf and Antonovics 2007; Van Allen et al. 2017), cannibalism can either enhance or decrease disease incidence in more complex scenarios. An increase in disease incidence occurs if a cannibal who is susceptible to the disease represents a sufficiently better resource for the pathogen or parasite than the infected victim (Sadeh et al. 2016). A cannibal, whether infected or not, could also function as a sloppy predator that helps to spread the disease through messy eating via partial consumption of an infected victim or defecation of the pathogen (Cáceres et al. 2009; Strauss et al. 2016). On the other hand, by consuming either susceptible or infected victims, a cannibal may be able to decrease disease incidence and spread. If cannibalism removes enough susceptible individuals, cannibalism may drive the number of susceptible individuals below the epizootic threshold population size (Van Allen et al. 2017). Only when the number of susceptible individuals is above this threshold does the disease readily spread (Anderson and May 1991). However, this potential negative effect on transmission via a decrease in susceptible individuals below the epidemic threshold only occurs if the disease is transmitted via density-dependent processes (Lloyd-Smith et al. 2005). If cannibals prey on infected individuals more frequently for any reason, such as infected individuals being easier to catch or less able to defend themselves and pathogen transmission through cannibalism is sufficiently low, cannibalism leads to a decline in transmission and infection prevalence (Van Allen et al. 2017).

When a pathogen stops or slows host development, the pathogen effectively creates a population where the potential healthy cannibals and the potential infected victims are different sizes. In this environment, where the victims are smaller than their cannibalistic conspecifics, stage-structure cannibalism becomes possible and often occurs (see Appendix S1 in Sadeh et al. 2016). When this does occur, theoretical models show that it has the potential to decrease overall transmission along with the epidemic severity if the probability of contracting an infection during cannibalistic predation is low (Van Allen et al. 2017). However, if cannibalism effectively transfers the disease from a smaller lower quality host to a larger higher quality host, disease prevalence could increase (Sadeh et al. 2016). The above equivocal conclusions, resulting in a decrease or increase in disease incidence, rely on the results of theoretical models and call for empirical research to help resolve this issue.

Using a tractable host–pathogen system, we set out to determine the extent to which cannibalism affects a susceptible individual's probability of contracting the disease and whether cannibalism increases or decreases disease incidence within a population during an epizootic outbreak. Specifically, we empirically quantified: (1) how cannibalism affected individual probability of infection given the consumption of an infected individual; and, (2) how cannibalism alters disease transmission dynamics. The experimental results show that, on an individual level, the overall risk of infection given cannibalism can be low and that, on a population level, this results in cannibalism decreasing pathogen transmission and reducing the risk of infection. In general, for the host population, there are distinct positive effects of cannibalism since it lowers disease prevalence in the population and decreases the risk of catching a deadly pathogen.

Methods

Study system

Our system consists of a lepidopteran larval host, the fall armyworm (*Spodoptera frugiperda*), and its lethal baculovirus that creates a stage-structured system where susceptible individuals are larger than infected individuals. The polyphagous fall armyworm is a multivoltine migratory species (Richter et al. 1987; Sparks 1979). Within the continental United States, adults annually migrate from Florida and Texas, where the species overwinters, to the rest of the Southern United States in April and May. They continue northward in subsequent non-overlapping generations. The 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). During population outbreaks, fall armyworm infestations can be large, resulting in population densities as high as 59 pupae/m^2 (Pair et al. 1991). The fall armyworm is also a widespread agricultural pest and considerable problem in the soybean fields of Brazil (Peruca et al. 2018). It has recently been introduced into Africa, where it causes widespread damage (Stokstad 2017). Importantly for this study, fall armyworm larvae are highly cannibalistic (Chapman et al. 1999; Valicente et al. 2013) even when given adequate food resources (He et al. 2021). The fall armyworm becomes readily infected with a species-specific and lethal baculovirus, *Spodoptera frugiperda* multicapsid nucleopolyhedrovirus (SfMNPV). The virus prevents infected larvae from molting and infection rates can reach 50-60% in infested areas (Fuxa 1982). Thus, SfMNPV represents an important source of mortality (Richter et al. 1987).

Baculoviruses, such as SfMNPV, are ubiquitous in nature and infect a wide-range of insect species (Miller 1997). In lepidopteran systems, baculovirus epizootics begin when foliage contaminated with occlusion bodies (OBs) are consumed by a susceptible larva (Cory and Myers 2003). The OBs consist of a protein coat containing multiple copies of the double-stranded DNA virus. If enough OBs are consumed, a fatal infection occurs. If

smaller amounts of OBs are consumed, sublethal covert infections occur but at at relatively low levels (Myers et al. 2000). These covert infections may be passed vertically from the adult moths to their offspring (Roy et al. 2009). For fatal infections, the baculovirus replicates within the host until the virus triggers the host's liquefaction. While uninfected individuals continue to molt to larger instars, infected individuals do not. Since infected individuals stop molting, viral infection creates a size-structured population with early instar infected individuals and later instar healthy individuals. In our system, non-cannibalistic horizontal transmission occurs when OBs are released after host liquefaction. The released OBs contaminate the surrounding foliage on which susceptible larvae feed. Over time, the OBs degrade due to UV light exposure (Elderd 2013). At the start of an epizootic, recently hatched first instars become infected. By the time infected first-instar larvae die, healthy individuals have grown to the fourth instar.

Since non-cannibalistic horizontal transmission occurs when the larvae consume leaf tissue contaminated with the OBs, we used soybeans (*Glycine max* cv. Gasoy 17), which the fall armyworm readily feeds upon (Richter et al. 1987; Sparks 1979), for the population-level transmission experiments. Additionally, the variety that we chose, Gasoy 17, when induced to produce secondary chemical defensive compounds, has no affect on overall mortality due to viral infection (Shikano et al. 2017). Thus, we did not have to consider the effect of how induced defenses arising from larval feeding may change mortality. However, artificial induction via the spraying of jasmonic acid (JA) does result in decreased feeding (Shikano et al. 2017) and, in turn, can increase cannibalism (Orrock et al. 2017), which we explore in the discussion. Since soybeans self-pollinate and produce genetically similar offspring, plant uniformity across treatments allows us to focus on the host–pathogen interaction and how cannibalism changes disease transmission dynamics without needing to account for any differences in resource quality across experimental treatments.

For the experiments, all larvae were obtained as eggs from Benzon Research, Inc. (Carlisle, PA) and reared on artificial diet (Southland Products, Inc., Lake Village, AR) at 28.9 C° and 16L:8D in individual one oz. cups until they had reached the appropriate instar needed for the individual-level and the population-level experiments outlined below. Virus was obtained from field-collected samples in Louisiana.

Individual-level methods

To test whether or not size differences between larval instars or infection status affected cannibalism incidence, we reared both infected and uninfected larvae while varying the developmental instar. To infect third through fifth instar larvae, we starved recently molted larvae for one day and then fed each larvae a 95% lethal dose of virus for fifth instar larvae in a 3 μl DI water droplet (10^5 OBs, *unpublished data*) on a cube of artificial diet. Larvae were only used if they ate the entire cube along with the full dose of virus. If the larvae consumed the entire diet cube, they were placed on individual one oz. cups containing artificial diet until needed. Uninfected larvae were starved then fed a cube of diet inoculated with a control solution of 3 μl of DI water only. Given the much smaller size of the first- and second-instar larvae, the infection procedure was different. Groups of smaller larvae were allowed to feed on artificial diet in a 2 oz. cup that had been inoculated with 9 μl of DI water containing 3 X 10^5 OBs. Uninfected first- and second-instar larvae were also reared in a 2 oz. cup that had been inoculated with a control solution of 9 μl of DI water.

The individual-level experiment consisted of placing two fall armyworm larvae in a 8 cm petri dish without other food resources, which is a similar experimental set up to Boots (1998). For each trial, we systematically varied both the larval instar and infection status. Larval instar ranged from the first through the fifth instar. In terms of infection status, both individuals could be infected, only the smaller larval instar was infected, only the larger larval instar was infected, or both individuals were uninfected. Sample size for each combination varied based on instar availability. In total, 488 individual trials were conducted (See Appendix A, Statistical Analysis, Table A1). For each trial, the petri dish was monitored for 24 hours and all incidences of cannibalism were recorded to quantify the probability of cannibalism. If the cannibal consumed an infected victim, we reared the individual until death or pupation to quantify the probability of infection given cannibalism. Death by virus was confirmed by checking the larval hemolymph for OBs, which are large enough to see under a light microscope (Miller 1997). It should be noted that for trials where the individual larvae differed in instar, the cannibal was always larger than the victim.

To analyze the individual-level data on cannibalism, we constructed a Bayesian logistic model with difference in instar, infection status of the cannibal, and the infection status of the victim as the main effects. Instar differences were treated as a numerical value. All other main effects were categorical. We also considered all of the interactions between the main effects. When an uninfected cannibal consumed an infected victim, the corresponding data were used to examine how likely cannibals were to become infected. The logistic model examining the probability of infection given cannibalism only included differences in instar as a predictor variable. Models were compared using both the Watanabe Akaike Information

Criteria (WAIC) and the Deviance Information Criterion (DIC) with the best fit model having the lowest score (Hobbs and Hooten 2015). The probability of cannibalism and the probability of infection given cannibalism were analyzed using JAGS (Plummer et al. 2003) and the R2JAGS package (Yu-Sung and Masanao 2015). The details of the Bayesian aspect of the analysis are included in Appendix A, Statistical Analysis. Together, these analyses at the individual level allow us to quantify both the probability of cannibalism and the probability of infection given cannibalism.

Population-level disease models

To model disease transmission dynamics in populations, ecologists have long relied on a variety of compartmental models based on the classic Susceptible-Infected-Recovered (SIR) system of differential equations (Anderson and May 1991; Keeling and Rohani 2008). A variation of these same models when combined with empirical data can be used to understand how cannibalism affects disease transmission. Focusing on the susceptible class, the change in susceptibles over time can be described by:

$$\frac{dS}{dt} = -\beta SI, \tag{1}$$

where β is the transmission rate, S represents the susceptible class, and I represents the infected class. In the above equation, all individuals within the susceptible class are equally susceptible to the pathogen and, thus, are equally likely to become infected. This assumption results in per capita transmission (i.e., $\frac{1}{S}\frac{dS}{dt}$) being linear (Elderd 2018). Yet, disease dynamics can be decidedly non-linear (Dwyer et al. 1997; Hochberg 1991). Taking a phenomenological approach to fitting non-linear data, one could model the non-linearity by raising either S or I to a power (e.g., S^a). Using a more mechanistic approach that accounts for heterogeneity in susceptibility among individuals rather than assuming all individuals are equally susceptible (Dwyer et al. 2000, 1997), eqn. 1 becomes:

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

$S(t)$ and $S(0)$ are the number of susceptibles at time t and at the beginning of the epizootic, respectively. The transmission rate no longer consists of a single number, β, but follows a probability distribution with a mean $\bar{\beta}$ and a coefficient of variation C (See Appendix B, Including Heterogeneity Using the Moment-Closure Approach for a derivation of eqn. 2). In eqn. 2, heterogeneity in susceptibility is represented by a transmission scaling factor, $\left[\frac{S(t)}{S(0)}\right]^{C^2}$, that starts at one at the beginning of the outbreak and decreases as the number of susceptibles declines over the course of the outbreak. Essentially, the transmission rate is highest at the beginning of the outbreak when individuals who are more susceptible to the disease become infected first. Towards the end of the outbreak, transmission declines since the least susceptible individuals in the population remain uninfected (Reilly and Elderd 2014). As the coefficient of variation goes to zero, the non-linear model, eqn. 2, simplifies to the linear model, eqn. 1 (Dwyer et al. 1997). In a series of population-level transmission experiments, we use the integrated versions of the above equations (See eqn. 3 and 4, which are the integrated forms of eqn. 1 and 2, respectively) to test how cannibalism affects transmission either through the changes in transmission rate (β or $\bar{\beta}$) or heterogeneity (C).

Population-level methods

For the population-level experimental treatments, we grew soybeans until each soybean had four to five true leaves. The individual plants were then re-planted into 58.5 cm by 58.5 cm wooden flats. A 50-cm tall clear plastic cylinder of Dura-Lar® measuring 25 cm in diameter was placed around each plant. The plastic cylinder guaranteed that all larvae used in the experiment were confined to a single plant. Each plant was assigned to either the cannibalism or control treatment.

For the control/non-cannibalism treatment, first instars were infected with baculovirus by placing them on a 2 oz. diet cup that had been inoculated with 9 μl of DI water containing 3×10^5 OBs. Two days after infection, infected individuals were placed onto their assigned plants at densities of 0, 15, 30, or 60 infected first instars and evenly distributed across the plant's leaves. Within four days of being placed on the plant, the infected first-instars died due to viral infection and, subsequently, contaminated the plant's leaf tissue with OBs. Sixteen healthy fourth-instar larvae were then placed on the plants and allowed to feed for two days (Fig. 1A). After two days, the fourth instars were removed, placed on individual diet cups, and reared until death or pupation. Death by the virus was easily confirmed as the virus causes hosts to liquify in their diet cups. Additionally, the OBs are large enough to be seen under a light microscope, which can be used to check larvae for virus infection (Miller 1997) when infection was not immediately apparent. The use of the first and fourth instars mimics naturally-occurring outbreaks where first instars become infected after emerging from their egg cases. By the time the first instars have died, individuals who are not infected grow to third or fourth instar (Elderd 2013).

For the cannibalism treatment, 16 healthy fourth-instar larvae were placed on the plant 2.5 hours after the

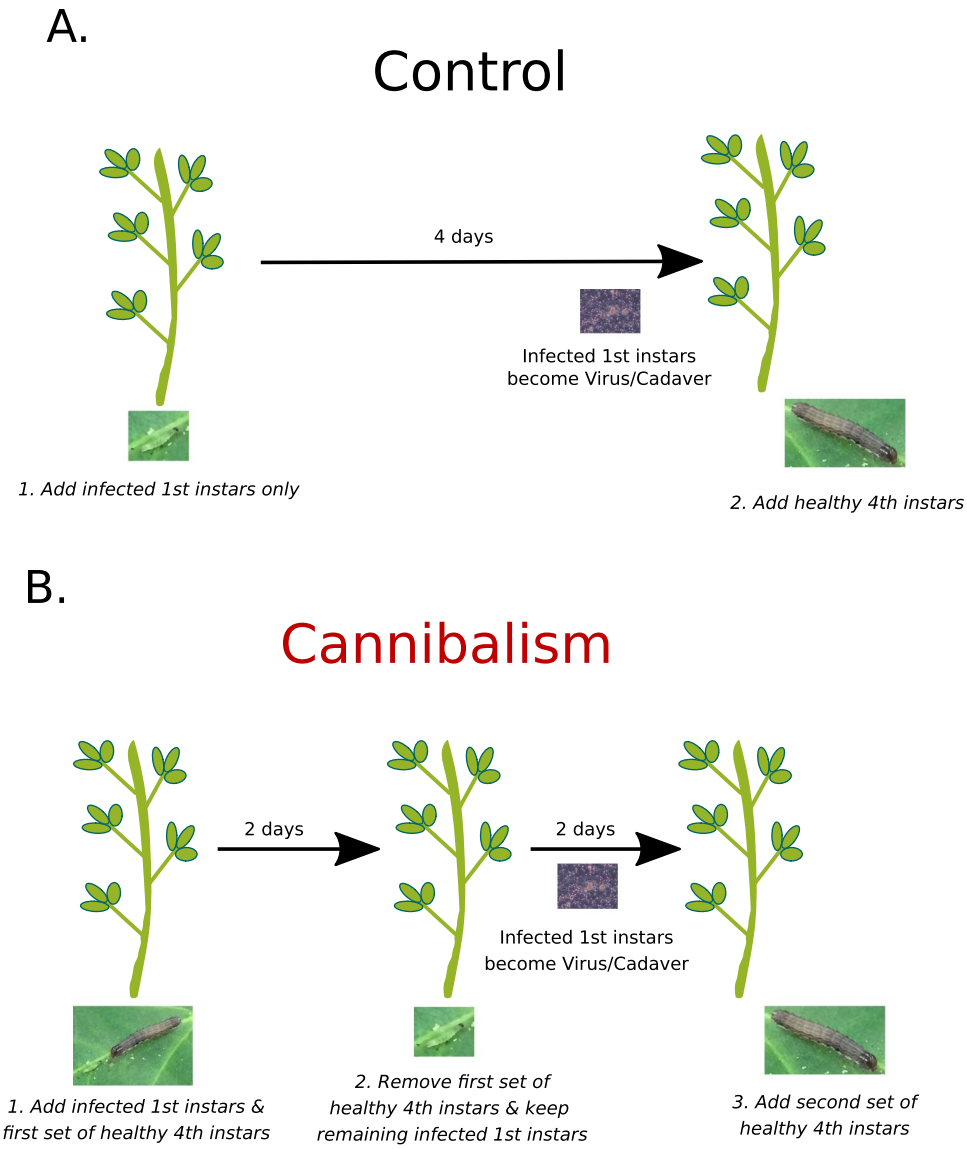

Fig. 1 **A** The Control/Non-Cannibalism treatment consisted of adding a varying number of infected first instars (0, 15, 30, or 60) and then waiting four days for the infected larvae to die due to viral infection. After the infected first instars had died, we added 16 healthy fourth instars to measure transmission without the possibility of cannibalism. **B** The Cannibalism treatment consisted of first adding a varying number of infected first instars (0, 15, 30, or 60) along with 16 healthy fourth instars at the same time. The first set of fourth instars were allowed to feed on the plant and cannibalize the infected first instars. Second, after two days, these fourth instars were collected to measure transmission due to cannibalism. The remaining infected first instars stayed on the plant and after two days died due to viral infections. Third, we added a second set of healthy fourth instars that were allowed to feed for two days

infected first-instars were evenly distributed across the plant (Fig. 1B). These fourth instars fed on the plant for two days and were also allowed to cannibalize the living infected first instars since the first instars would not have succumbed to the infection while these healthy individuals were feeding. This first set of fourth instars were removed after two days, placed on diet cups, and reared until death or pupation. The non-cannibalized infected first instars remained on the plant. After an additional two days, during which any non-cannibalized infected first instars liquify due to viral infection, a second set of 16 healthy fourth-instar larvae were placed on the plant. This second set was also allowed to feed for two days after which they were collected. Thus, both sets of larvae were exposed to the virus for the same period of time, 2 days. While we would have liked to count the remaining uncannibalized first instars post-cannibalism after they have died due to infection before the second set of healthy larvae began feeding, we could not due to the size of the first instars and the need to destructively sample the plant to obtain an accurate count. Additionally, we would not have been able to quantify any virus that had been spread due to "messy eating" during a cannibalism event. Instead, we used the infection data from the second set of healthy larvae along with the transmission estimates from the control/non-cannibalism treatment to estimate the number of infected first instar larvae remaining on the plant. This estimate was used to calculate the reduction in risk for the second set of larvae (methods outlined below).

To determine whether or not cannibalism affected transmission dynamics, we fit the integrated versions of the linear (i.e., homogeneity in susceptibility) and non-linear

(i.e., heterogeneity in susceptibility) transmission equations (Table 1, Eqs. 1, 2) to the data from the control/non-cannibalism treatments and the first set of healthy fourth-instar larvae that were allowed to cannibalize infected first instars. To do this, we first integrated the linear (eqn. 1) and non-linear (Eq. 2) equations from 0 to time T where T is the total number of days (i.e., two) that the healthy fourth instars were exposed to the virus either via consuming the virus-infected cadavers on the leaf tissue or cannibalism. When integrated, the linear equation (Eq. 1) becomes:

$$-\ln\left[\frac{S(T)}{S(0)}\right] = \beta I(0)\,T. \tag{3}$$

The non-linear equation (eqn. 2) becomes:

$$-\ln\left[\frac{S(T)}{S(0)}\right] = 1/C^2(\ln(1+\bar{\beta}C^2 I(0)\,T)). \tag{4}$$

Note that $S(T)/S(0)$ can be rewritten as $1-i$, where i is the percent infected. $I(0)$ is the number of infected larvae that are added onto an individual plant at the beginning of the experiment. Equations 3 and 4 show vastly different dynamics - linear and non-linear, respectively - as the density of infected individuals increase (Fig. 2). The data were fit to eqn. 3 and 4 using a Bayesian framework (Appendix A, Statistical Analysis). Population-level models were compared using both WAIC and DIC (Hobbs and Hooten 2015). A total of three to four replicates for each treatment and larval density were used in the analysis. The varying number of replicates depended on the availability of soybean plants. This analysis allowed us to compare changes in transmission rate and/or the coefficient of variation associated with transmission due to cannibalism.

To quantify relative reduction in the host's infection risk post-cannibalism, we estimated the number of infected live larvae consumed via cannibalism by solving for the initial number of infected larvae, $I(0)$, that the second set of healthy larvae were exposed to using eqn. 4 and the Bayesian posterior estimates of transmission. First, we quantified the percent infected, i, from the second set of healthy instars that were allowed to feed on the plants post-cannibalism (Fig. 1B). We then assumed that transmission is similar for both the control treatments and the post-cannibalism second set of healthy larvae. Given this reasonable assumption, we used the posterior parameter estimates of the best-fit model from the control treatment and the percent infected, i, from the second set of healthy individuals in the cannibalism treatment to estimate the number of uncannibalized cadavers on each plant in the cannibalism treatment after the first-set of cannibalistic larvae had finished feeding. The relative reduction in risk due to cannibals consuming infected larvae was calculated as one minus the ratio of the estimated number of cadavers divided by the initial number of infected larvae placed on the plant at the start of the experiment. This allowed us to directly quantify the extent to which cannibalism reduced disease prevalence and infection risk.

Results

Individual-level Results

On the individual level, only the main effects of instar difference and infection status were included in the best-fit model (Table 1, *Individual-level Cannibalism*). As instar differences increased between the potential cannibal and the victim, the probability of cannibalism increased rapidly (Fig. 3A). When differences were greater than two instars, the probability of cannibalism was near 1.0. Interestingly,

Table 1 WAIC and DIC values for the four models for the individual-level cannibalism experiment, the two models for the infection given cannibalism experiment, and the six models considered for the population-level transmission experiment

Model	WAIC values	DIC values
Individual-level cannibalism		
1. No difference	459.53	459.49
2. Main effects only	**310.48**	**310.59**
3. Main effects & two-way interactions Only	313.72	314.57
4. Main effects & all interactions	312.64	318.50
Individual-level infection given cannibalism		
1. No difference	103.90	103.89
2. Main effect	**94.85**	**94.58**
Population-level transmission		
1. Linear, no difference	105.24	103.02
2. Non-linear, no difference	103.76	101.34
3. Both linear	81.67	79.84
4. Both non-linear	**79.82**	**78.49**
5. Cannibalism non-linear, Ctrl linear	81.32	79.72
6. Cannibalism linear, Ctrl non-linear	80.17	78.66

The best fit models are in bold. For the individual-level cannibalism experiment, the four models considered include a null model of no differences and three models where either the main effects (i.e., difference in instar between the cannibal and victim, infection status of the cannibal, and infection status of the victim), the main effects plus the two-way interactions, or the main effects along with the two-way and three-way interactions were included. For the individual-level infection given cannibalism analysis, the two models consist of a null model of no effect and a model that considered the single main effect of the difference in the instar of the cannibal and its victim. For the population-level transmission experiment, linear and non-linear refer to the solved linear equation (eqn. 3) that assumes that individuals do not differ in susceptibility and the heterogenous equation (eqn. 4) that assumes individuals do vary. The first two models for the population-level transmission data assume no difference between cannibalism and non-cannibalism treatments

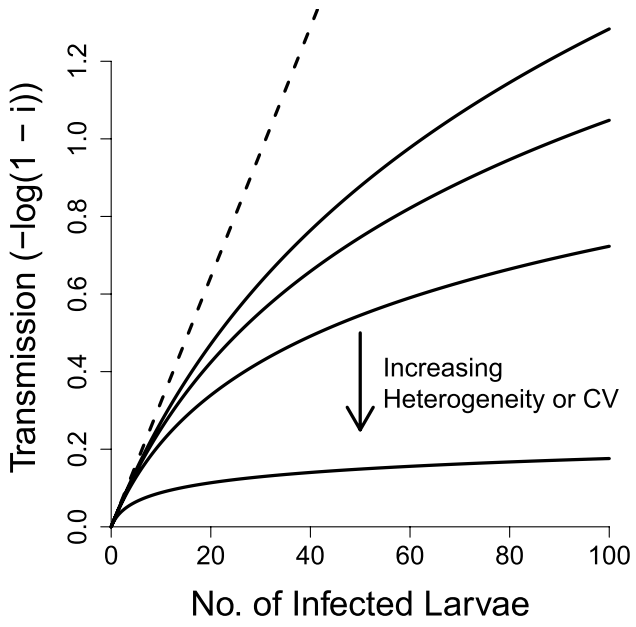

Fig. 2 Effect of heterogeneity in susceptibility amongst individuals on the disease transmission as measured by the negative natural log of the fraction infected. The dashed line assumes that all individuals are equally at risk and increases linearly as the number of infected individuals increase (Eq. 3). The solid line shows that as heterogeneity increases that transmission declines and asymptotes at a smaller fraction of infections in the population (eqn. 4)

there was a higher probability of cannibalism when the cannibal was infected perhaps due to physiological stress or nutritional state, which can result in increased cannibalism rates (Fox 1975; Polis 1981). There was also a decreased chance of cannibalism when the victim was infected (Fig. 3A). When the cannibal and the infected victim were the same instar, the median probability of being cannibalized was close to a 50:50 chance (0.46 [0.34, 0.58]; median [95% Credible Interval (CI)]). However, when instar differences were upwards of three instars, which is the difference often seen in the field between infected and uninfected individuals (Elderd 2019; Elderd and Reilly 2014), the probability of cannibalism was nearly certain (0.95 [0.92, 0.98]). Overall, the general trend showed that as instar differences increased, the probability of being cannibalized increased rapidly.

If a healthy cannibal consumed an infected victim, the probability of infection given cannibalism decreased as the differences in the number of instars between the cannibal and the victim increased (Table 1, *Individual-level Infection given Cannibalism* and Fig. 3B). When there were no differences in larval stage between the cannibal and the victim, the probability of infection was below 1.0 with the median probability (95% CI) being 0.62 (0.38, 0.81). As a comparison, when there was a difference of three instars, the probability of infection was much lower with the median probability

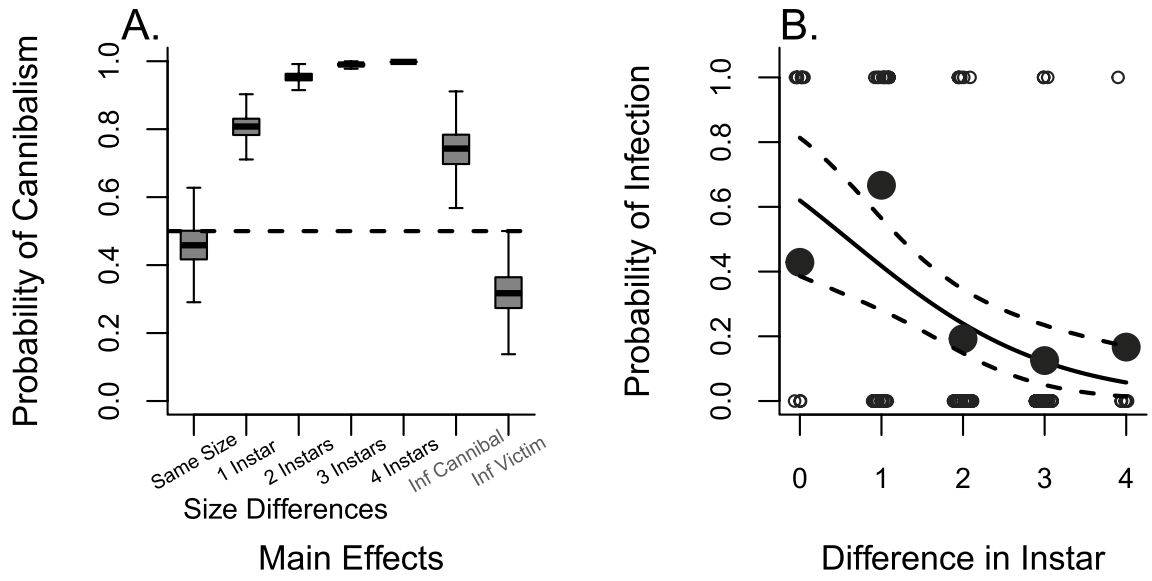

Fig. 3 **A** Boxplot of the probability of cannibalism given the main effects of the logistic regression model based on difference in instar, whether the cannibal is infected, and whether the victim is infected. In the box-and-whisker plots, the mid-lines are median values, the box limits show the first and third quartiles, and the whiskers extend beyond the box limits by 1.5 times the interquartile range. A dashed line is placed at a probability of 0.5 for reference. On the x-axis, the first five terms are the instar differences between the cannibal and victim. The last two terms in gray are whether the cannibal was infected (Inf Cannibal) or the victim was infected (Inf Victim). **B** Probability of infection if the cannibal consumed an infected individual given the differences in instar between the cannibal, which was always larger if there was a difference in instar, and the victim. The median predicted (solid line) and the 95% Credible Intervals (dashed lines) are plotted. The large solid points represent the mean response for each difference in instar and the small open points are the individual data. The data are jittered for ease of presentation

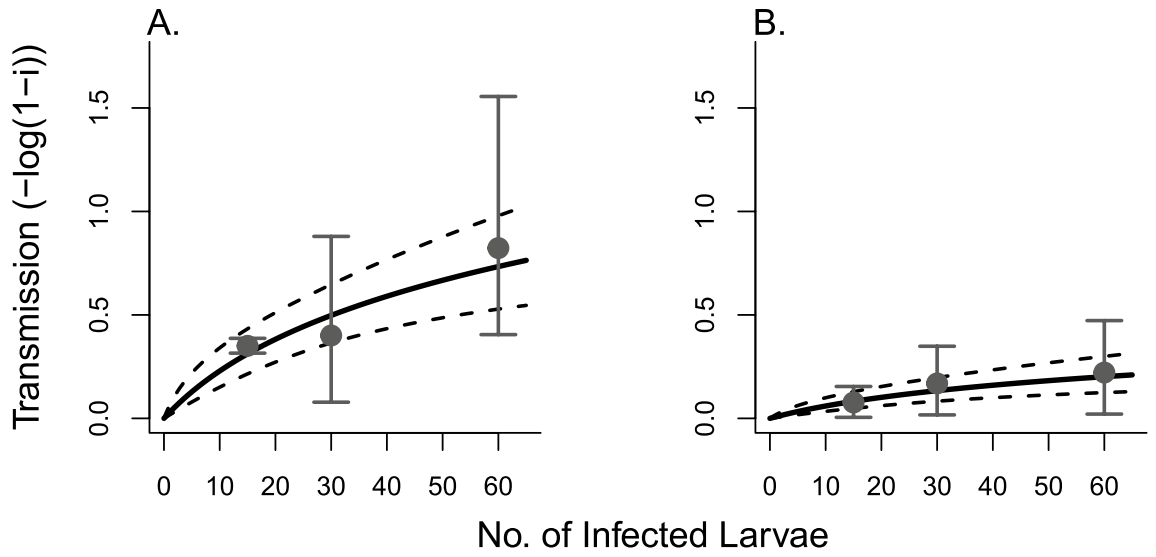

Fig. 4 Best-fit model of effects of cannibalism on transmission where **A** is the control/non-cannibalism treatment fit to the solved non-linear model (eqn. 4) and **B** is the cannibalism treatment fit to the solved non-linear model (eqn. 4). The non-linear model assumes heterogeneity in susceptibility. Transmission is measured as the negative natural log of one minus the percent infected, *i*. The points are the median values and the error bars represent one standard deviation. The solid line is the predicted median and the dashed lines are the 95% Credible Intervals

(95% CI) being 0.12 (0.05, 0.24). Thus, when a cannibal consumes an infected victim, the infection does not readily spread to the cannibal.

For the individual-level experiments, differences in the inoculation methods for the smaller instars (first and second instars) as compared to the larger instars (third through fifth instars) could potentially impact the percentage of individuals that become infected when a healthy larva feeds on an infected larva (See Appendix A, Statistical Analysis, Table A2). To test for this potential affect, we examined whether healthy second instars that consumed infected second and first instars were more or less likely to become infected than healthy fourth instars that consumed infected fourth or third instars using a simple logistic regression. Thus, both sets of healthy instars consumed individuals who were either of the same size or one instar smaller but differed in terms of inoculation methods. We found that there was no difference in percent infected between the two groups with the median estimate of the difference close to zero and the 95% CI overlapping zero (− 0.08 [− 0.57, 0.41]). Given the data, we can infer that inoculation methods are likely to have little impact on the results.

Population-level results

Given the relatively low chance of the infection being passed onto the cannibal, it should not be surprising that disease transmission decreased in cannibalism treatments as compared to the control/non-cannibalism treatments (Fig. 4). The null models that pooled the data across treatments had very little support in comparison to all other models considered (Table 1, *Population-level Transmission*). Both the cannibalism and control/non-cannibalism treatments were

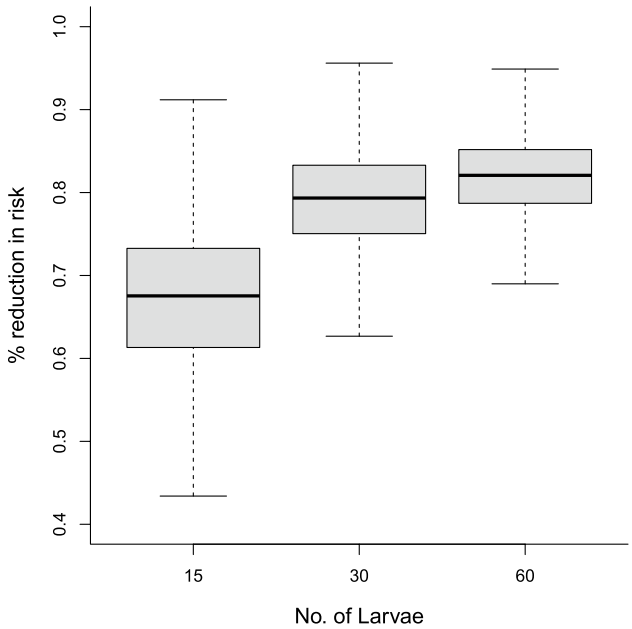

Fig. 5 Box plot of the associated percent reduction in risk due to cannibalism where reduction is measured as one minus the percent of infected cadavers remaining in the cannibalism treatment post-cannibalism for each of the infected larvae treatments. Here the bold solid mid-line is the median, the box limits represents the first and third quartiles, and the whiskers are 1.5 times the interquartile range

best fit by the non-linear model that assumes heterogeneity in susceptibility amongst the hosts (Table 1, *Population-level Transmission* and Fig. 4). When comparing parameter estimates for the non-linear model (eq. 4), estimates of the mean transmission rate $\bar{\beta}$ overlapped slightly given the 95% CI (cannibalism: 0.0037 [0.0019, 0.0095], no cannibalism: 0.0147 [0.0083, 0.0357], median [95% CI]); whereas, estimates of the coefficient of variation C overlapped considerably (cannibalism: 2.65 [1.44, 4.36], no cannibalism: 1.46 [0.829, 2.24]). Overall, the estimates of the mean transmission rate were much lower for the cannibalism treatment. This resulted in a dramatic decrease in transmission for cannibalistic healthy larvae as compared to the healthy larvae exposed to the virus-laden cadavers in the control/no-cannibalism treatment (Fig. 4A compared to 4B).

Given that the cannibalistic healthy larvae consumed live infected first-instar larvae, how much did they consume and, potentially, reduce the risk of infection, post-cannibalism, for the second-set of healthy larvae exposed to the remaining virus? The number of larvae consumed via cannibalism increased as the number of larvae initially placed on the plant increased (Treatment: Median [95% CI] – 15 infected: 4.9 [2.57, 7.84], 30 infected: 6.2 [3.04, 10.24], 60 infected: 10.8 [5.77, 17.24]). As a result, fewer of the live infected first-instar larvae subsequently died of viral infection on the plant. This resulted in a decrease in exposure risk for the second set of healthy larvae (Fig. 5). The reduction in risk was substantial with anywhere between 62.5% to 82.0% percent median reduction across all treatments. While the reduction in relative risk increased as the number of live infected first-instar larvae used in the experiment increased, there was considerable overlap between the infected larvae treatments (Fig. 5).

For the population-level transmission experiments, the first set of healthy fourth-instar larvae, the cannibals, experienced much lower transmission as compared to the control/non-cannibalistic larvae (Fig. 4A and 4B). The second set of healthy fourth instars added to the cannibalism treatments fed in an environment with a large reduction in infection risk (Fig. 5).

Discussion

Historically, the frequency of cannibalism was thought to be limited by its disadvantages (Pfennig 1997, 2000). One such disadvantage arose from the cannibal consuming pathogen- or parasite-infected conspecifics and becoming infected oneself. Yet, theoretical work has highlighted that these disadvantages to a cannibalistic host may not be as great as once thought (Bolker et al. 2008; Rudolf and Antonovics 2007; Van Allen et al. 2017, but see Sadeh et al. 2016). In fact, cannibalism may result in a decline in the spread of a disease, particularly in size-structured populations (Van Allen et al. 2017). Our empirical results (Fig. 4 and 5) support this idea. The population-level results showing a decrease in transmission and subsequent decrease in disease risk (Fig. 4 and 5) stem from the individual-level results (Fig. 3). While individuals readily cannibalize conspecifics of a smaller instar, the risk of infection in a size-structured system is relatively low (Fig. 3). Thus, by removing infected individuals before they become virus-laden cadavers, overall transmission should decline, which it does (Fig. 4) as does infection risk (Fig. 5).

Given our results, cannibalism clearly decreased disease transmission (Fig. 4), especially given the lack of fit of the two null models (Table 1). The decline in transmission for the best-fit model was driven by a lower transmission rate when cannibalism occurred. As a result of healthy fourth-instar larvae consuming infected first-instar larvae, there was a decrease in the amount of virus in the system and a reduction in infection risk via non-cannibalistic transmission as well (Fig. 5). There was also support for the second best-fit model where the cannibalism treatment was fit to the linear model, which assumes all individuals are equally susceptible (Eq. 3) and the control/non-cannibalism treatment was fit to the non-linear model, which assumes heterogeneity in susceptibility (Eq. 4). Given the relatively low values of the coefficient of variation for the cannibalism treatment, the fact that a linear model fit the data well is not unexpected since the non-linear model converges to the linear model as the coefficient of variation approaches zero (Dwyer et al. 1997). The differences between the two treatments for the second best-fit model also support the conclusion that cannibalism lowers the transmission rate since the estimate of the transmission rate for the linear model fit to the cannibalism data (0.0021 [0.001, 0.0029], median [95% CI]) is much less than the non-cannibalism treatment (0.0147 [0.0083, 0.0357], median [95% CI]). The transmission dynamics are also similar when comparing the non-linear and linear model fit to the cannibalism data (See Fig. A4 in Appendix A, Statistical Analysis).

From the pathogen perspective, one could argue that by infecting a larger host the amount of virus eventually produced will increase. The resulting increase in the pathogen population could result in an increase in epizootic intensity (Sadeh et al. 2016). Yet, when the probability of infection given cannibalism is relatively low, which is true for this system (Fig. 3B) and others (e.g., Boots 1998), the benefit of finding a better host from the pathogen's perspective may not make up for the fact that very little transmission occurs between the cannibal and the infected host. However, the experiments conducted only examine the transmission dynamics over the course of a single outbreak. Finding a more fit host could have an effect on the long-term dynamics when looking across multiple outbreaks.

Within the population-level experimental design, we essentially constrained cannibalism such that only healthy fourth-instar larvae could cannibalize infected first-instar larvae. In the field, there is a possibility that fourth-instar larvae that have become infected via cannibalism may be consumed by a healthy conspecific of the same size. Given that infected fourth instars contain a magnitude or more of infectious particles (Dwyer et al. 1997), cannibalism may result in the pathogen infecting a more competent host (Sadeh et al. 2016). Using the results from our individual-level experiments (Fig. 3), we can calculate the probability that a healthy fourth instar will consume a infected fourth instar as well as the probability that the cannibalism of the infected fourth instar will result in an infection. The median estimate of the probability of cannibalism of the same size instar that is infected is 32% with a 95% CI of 20% to 46%. The probability of this resulting in an infection is 62% (39%, 81%). Taken together, the median probability of both cannibalism and that the cannibalism results in an infection is approximately 20%. This is relatively low but given the fact that fourth instars contain magnitudes more viral particles, it could result in an increase in the amount of virus in the environment. However, the above does not account for the fact that healthy fourth-instar larvae will molt into the next instar within two to three days and eventually pupate within approximately six to seven days (Elderd, *unpublished data*). At even larger instars, the probability of cannibalism resulting in infection declines since larger instars have to consume more infectious particles (Dwyer et al. 1997). Within this system, there is a limited window of contact between the infected fourth instar, which becomes infected via cannibalism of a infected first instar, and a healthy fourth instar. However, the cannibalism of an infected fourth instar by a healthy fourth instar essentially replaces a single infected individual with another infected individual. Given that this is not 100 percent certain, cannibalism of an infected fourth instar by a healthy fourth instar will not necessarily help spread the pathogen (Van Allen et al. 2017).

The transmission dynamics that we examined take place over a single generation for this insect species. Given that the fall armyworm has non-overlapping generations (Elderd 2019), we may be able to make some inference about the ecological dynamics across multiple generations. Historically, a number of host–pathogen interactions in Lepidoptera result in long-term population cycles, particular for species that are considered silvicultural and agricultural pests (Anderson and May 1980; Bjørnstad et al. 2010; Hutchison et al. 2010). In fact, baculoviruses can be used as biocontrol to lessen the damage associated with severe outbreaks and these additions may result in dampening of the long-term cycles (Reilly and Elderd2014). Cannibalism, since it decreases transmission, would lessen the effectiveness of these agents especially when sprayed earlier in the season, which infects the smaller instar larvae. Perhaps, spraying later in the season and infecting the later instar larvae would be more beneficial since the later instar larvae produce more baculovirus when infected (Dwyer et al. 1997). The question still remains with regard to the long-term impacts of cannibalism. While we can make some inference, this comes with the caveat that the population-level experiment took less than a week to conduct. Greater inference can be had be looking at long-term dynamics in the field over multiple generations either through experimental or observational means. For the latter, research in other systems have combined these short-term experiments with observational data to make inference about what drives long-term cycles in outbreaking insects (Dwyer et al. 2000; Elderd et al. 2013) but, sometimes, with mixed success (Mihaljevic et al. 2020).

Taking an even longer-term view from the host's perspective, if there was a long-term disadvantage to cannibalism for the host with respect to disease transmission, cannibalism should decline over evolutionary time or better avoidance of infected hosts should evolve. However, in this system, where the pathogen readily infects and kills the host, there may be selective pressure that ensures cannibalism is maintained. This may be driven by the relative low cost of cannibalism as measured by the probability of becoming infected when cannibalizing an infected individual (Fig. 3B), whereas, in other systems, cannibalism may have an increased cost arising from a much higher probability of becoming infected (Pfennig et al. 1998). Thus, there may be different adaptive peaks with regard to maintaining cannibalism across species just as there may be multiple adaptive peaks for cannibalistic traits within species in non-disease driven systems (Stevens 1989).

While induction of the soybean variety Gasoy 17, the variety that we used in the population-level experiments, has no effect on mortality, it does reduce feeding and, subsequently, larval weight gain when induced (Shikano et al. 2017). Reduction in feeding due to changes in resource quality via induction increases cannibalism (Orrock et al. 2017, 2022). For the second set of healthy fourth instars in the cannibalism treatments at the population level (Fig. 1B), the plants on which they fed may have been induced due to prior feeding and this may have increased cannibalism among the second set of healthy fourth instars. In our population-level experiment, we found a difference in the number of fourth-instar larvae that we recovered when comparing the larvae recovered from plants in the control treatments as well as the first set of healthy larvae recovered in the cannibalism treatments (89%, [85%, 92%]) to the second set of larvae recovered from plants in the cannibalism treatments (70%, [62%, 76%]), which is a 19% reduction (11%, 27%) in recovery. Although recovery only serves as a proxy since lack of recovery could be due to cannibalism or other factors (e.g., natural mortality or larval escape). Unfortunately, given the

experimental design, we can not tease apart the potential interaction between induction, cannibalism, and disease transmission.

Thus, a potential avenue of future research could focus on how resource quality affects the interaction between cannibalism and disease transmission. As resource quality declines or resources become scarce, cannibalism increases (Orrock et al. 2017; Simpson et al. 2006). Amongst *Spodoptera exigua*, the beet armyworm, induction of secondary chemical defenses in tomato plants led to an increase in cannibalism (Orrock et al. 2017). In *S. frugiperda*, induction of chemical defenses in soybean led to a decrease in transmission (Elderd 2019). Change in transmission dynamics due to changes in chemical defenses also impacts other baculovirus-driven systems (Elderd et al. 2013). However, the manner in which transmission dynamics change depend upon the secondary chemical induced (Elderd 2019). How the interaction between cannibalism and disease transmission changes as a result of changes in resource quality remains an open question.

While we focused on a host–pathogen system, cannibalism also plays an important role in other empirical systems ranging from plant-herbivore to predator-prey. For instance, in a plant-herbivore system, egg cannibalism in the pest species, *Helicoverpa armigera*, decreases larval densities leading to a decline in leaf tissue consumption (Sigsgaard et al. 2002). When *H. zea* are placed on an induced tomato, *Solanum lycopersicum*, cannibalism increases and this has the same magnitude of impact on herbivore populations as a lethal disease. Thus, cannibalism in this instance is equivalent in strength to a host–pathogen interaction (Orrock et al. 2022). Here, cannibalism in an herbivore population has a positive impact on the plant. Similarly, in a host-parasitoid system, preferential cannibalistic feeding on parasitized individuals by the host may result in the extinction of the parasitoid from the system (Reed et al. 1996). For a salamander species (*Hynobius retardatus*), cannibals grow more quickly and are able to consume larger prey items than smaller non-cannibalistic salamanders (Takatsu and Kishida 2020). In fact, in this system, stage-structured cannibalism may help stabilize the population dynamics for the cannibal (Ohlberger et al. 2014; Richardson et al. 2010). For agricultural systems, these cannibalistic interactions can either serve to decrease pest populations (e.g., egg cannibalism by the pest species) or decrease the effectiveness of a biocontrol agent (e.g., parasitoid extinction, declines in disease transmission as shown in this study). From the relatively limited number of empirical studies, the effects of cannibalism on system dynamics clearly have important consequences (Fox 1975; Polis 1981; Richardson et al. 2010).

Overall, cannibalism decreased transmission and disease risk. On an individual level, the chance of cannibalism increases as instar differences increase and the chance of infection decreases as instar differences increase (Fig. 3). On a population level, infection prevalence drastically decreases when cannibalism occurs due to lower levels of transmission (Fig. 4) and further reduces the risk of infection in the population (Fig. 5). Consequently, in this highly cannibalistic species, the presence of a pathogen does not result in an increase in disease incidence but rather decreases infection prevalence. Thus, the negative consequences of cannibalism via consuming an infected individual may instead lessen a pathogen's impact.

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Data availability Upon publication, the data will be made uploaded to Dryad, a data repository. Data and code associated with the analysis are provided as private-for-peer review at Dryad via the following link: https://doi.org/10.5061/dryad.5hqbzkh61.

Code availability Upon publication, the code will be made uploaded to Dryad, a data repository.

Declarations

Conflict of interest The authors have no competing interests.

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