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Warmer temperatures increase disease transmission and outbreak intensity in a host–pathogen system

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Summary

1. While rising global temperatures are increasingly affecting both species and their biotic interactions, the debate about whether global warming will increase or decrease disease transmission between individuals remains far from resolved. This may stem from the lack of empirical data.

2. Using a tractable and easily manipulated insect host–pathogen system, we conducted a series of field and laboratory experiments to examine how increased temperatures affect disease transmission using the crop-defoliating pest, the fall armyworm (*Spodoptera frugiperda*) and its species-specific baculovirus, which causes a fatal infection.

3. To examine the effects of temperature on disease transmission in the field, we manipulated baculovirus density and temperature. As infection occurs when a host consumes leaf tissue on which the pathogen resides, baculovirus density was controlled by placing varying numbers of infected neonate larvae on experimental plants. Temperature was manipulated by using open-top chambers (OTCs). The laboratory experiments examined how increased temperatures affect fall armyworm feeding and development rates, which provide insight into how host feeding behaviour and physiology may affect transmission.

4. Disease transmission and outbreak intensity, measured as the cumulative fraction infected during an epizootic, increased at higher temperatures. However, there was no appreciable change in the mean transmission rate of the disease, which is often the focus of empirical and theoretical research. Instead, the coefficient of variation (CV) associated with the transmission rate shrunk. As the CV decreased, heterogeneity in disease risk across individuals declined, which resulted in an increase in outbreak intensity.

5. In the laboratory, increased temperatures increased feeding rates and decreased developmental times. As the host consumes the virus along with the leaf tissue on which it resides, increased feeding rate is likely to increase the probability of an individual consuming virus-infected leaf tissue. On the other hand, decreased developmental time increases the sloughing of midgut cells, which is predicted to hinder viral infection.

6. Increases in outbreak intensity or epizootic severity, as the climate warms, may lead to changes in the long-term dynamics of pests whose populations are strongly affected by host–pathogen interactions. Overall, this work demonstrates that the usual assumptions governing these effects, via changes in the mean transmission rate alone, may not be correct.

Key-words: baculovirus, climate change, epizootics, fall armyworm, *Spodoptera frugiperda*, variability in transmission rate

Introduction

As global temperatures continue to rise, the ecological impacts of climate change are becoming more apparent (Adler & HilleRisLambers 2008; Doak & Morris 2010; Milazzo *et al.* 2013). Initially, research efforts focused on how climate change will affect species in isolation and, in particular, how the range of a particular species will respond to a warmer world (Parmesan *et al.* 1999). More recent efforts recognized the importance of species interactions, which may represent potential biotic multipliers of climate change (Zarnetske, Skelly & Urban 2012). This

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led to both theoretical and empirical research on multiple topics such as plant–herbivore (Vasseur & McCann 2005; O'Connor 2009; O'Connor, Gilbert & Brown 2011), predator–prey (Harmon, Moran & Ives 2009) and multispecies interactions (Suttle, Thomsen & Power 2007; Barton, Beckerman & Schmitz 2009; Urban, Tewksbury & Sheldon 2012; Hansen *et al.* 2013). While there have also been theoretical and empirically based models examining the effects of global warming on disease transmission (Dobson 2009; Molnar *et al.* 2013; Mordecai *et al.* 2013; Thompson, Levin & Rodriguez-Iturbe 2013) as well as laboratory experiments (Paull, LaFonte & Johnson 2012; Ben-Horin, Lenihan & Lafferty 2013), few studies have addressed this question using field experiments (but see Roy, Gusewell & Harte 2004). Thus, the effects of climate change on disease transmission dynamics represent an area of research that has raised a number of questions that remain unanswered about the timing and intensity of epidemics or epizootics in a warmer world (Rohr *et al.* 2011; Altizer *et al.* 2013). This may be due, in a large part, to a lack of empirical data (Pascual & Bouma 2009).

In general, there has been considerable debate in the disease literature on whether global warming and its associated stressors will result in an increase or decrease in disease outbreak frequency and intensity (Lafferty & Holt 2003; Wilson 2009; Rohr *et al.* 2011). The focus of the debate appears to hinge on how increased temperatures will affect disease transmission rates between individuals. An underlying simplifying assumption of models used in this debate is that disease transmission rates do not vary greatly between individuals in a population (Moore *et al.* 2012; Molnar *et al.* 2013). Thus, all individuals have the same transmission rate, which results in transmission depending linearly on host and pathogen densities. We realize that this simplifying assumption is not solely relegated to the debate on climate change and disease transmission. In fact, this assumption is used throughout the pathogen and parasite literature (Anderson & May 1980; Hudson, Dobson & Newborn 1998; Grenfell, Bjornstad & Kappey 2001; Elderd, Dukic & Dwyer 2013) and can be fully justified as these linear transmission models fit the observed data. Yet, individuals vary in their risk of contracting a disease (Anderson & May 1991; Dwyer, Firestone & Stevens 2005). The degree of variability or heterogeneity in the population can have important consequences for determining the number of individuals infected and, thus, intensity of an outbreak both over the short and long term (May & Anderson 1988; Dwyer, Elkinton & Buonaccorsi 1997; Dwyer *et al.* 2000; Ben-Ami, Ebert & Regoes 2010; Tompkins *et al.* 2011).

For many insect host–pathogen or host–parasitoid interactions, as well as other systems, variability in individual infection or parasitism risk can play an important role in disease transmission or parasitism rates (Anderson & May 1980; May & Anderson 1988; Reeve, Cronin & Strong 1994; Briggs & Godfray 1996; Cronin & Strong 1999). Hassell *et al.* (1991) show that in a host–parasitoid system that heterogeneity in the parasitoid attack rate stabilizes the system. In theoretical systems where individual hosts are considered homogeneous such as in the classic Nicholson-Bailey model (Nicholson & Bailey 1935), the host–parasitoid system as a whole goes extinct. For host–pathogen systems, allowing for heterogeneity in the disease transmission rate best explains epizootic data for the invasive gypsy moth, *Lymantria dispar* (Dwyer, Elkinton & Buonaccorsi 1997; Elderd, Dushoff & Dwyer 2008; Elderd *et al.* 2013). Essentially, the addition of heterogeneity to transmission models results in a nonlinear relationship between pathogen or virus density and transmission. While we assume that heterogeneity drives this nonlinear relationship, other mechanisms can also result in nonlinear transmission (Hochberg 1991; McCallum, Barlow & Hone 2001; Fenton *et al.* 2002). Traditional models that do not include heterogeneity assume a linear relationship (Fig. 1). Thus, heterogeneity in transmission rates and how heterogeneity changes in a warmer world may have important consequences for epizootic outbreaks.

While heterogeneity in disease transmission regulates the short-term dynamics of an epizootic, it is also important for determining the system's long-term dynamics. The long-term dynamics of these host–pathogen interactions commonly exhibit boom and bust cycles, in which the host population crashes from peak levels due to disease outbreaks (Cory, Hails & Sait 1997; Liebhold *et al.* 2000). Insect species exhibiting long-term cycles are often of ecological and economic interest because the boom portion of the cycle leads to widespread forest and crop damage (Dwyer, Dushoff & Yee 2004). In fact, the ability of pathogens to decrease pest population size has led to the development and use of a number of pathogen-based biocontrol agents (Hochberg 1989; Podgwaite *et al.* 1992; Moscardi 1999; Moreau *et al.* 2005; Moreau & Lucarotti

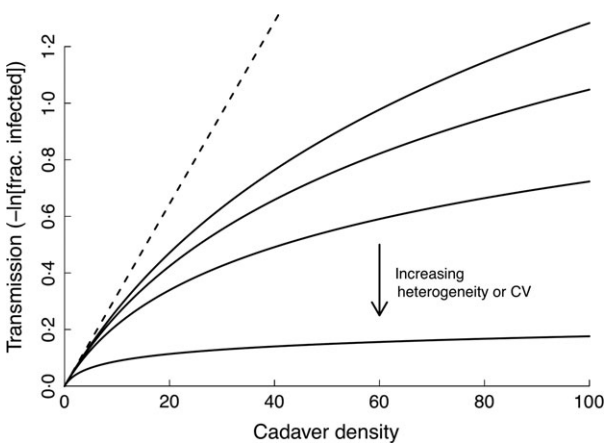

Fig. 1. Effect of increasing the transmission rate's coefficient of variation (CV) or K in eqn 2 on transmission. The solid lines represent populations in which risk varies across individuals. The dashed line represents a population in which all individuals are equally at risk (eqn 1). Each line uses the same value for the transmission rate of the virus.

2007; Gomez-Bonilla *et al*. 2013). Thus, any change in disease transmission and epizootic intensity due to climate change may have important consequences from both an ecological and economic perspective.

For many lepidopteran and other insect species, baculoviruses, which include nucleopolyhedroviruses (NPVs), comprise the pathogen source responsible for a number of large-scale epizootics (Miller 1997). Baculovirus-driven epizootics begin when a larva consumes foliage contaminated with baculovirus occlusion bodies (OBs) (Cory & 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. Prior to death, the virus replicates within the non-moulting larva until the baculovirus triggers the dissolution of the larval integument (Miller 1997). The OBs are released and contaminate the foliage on which the host is feeding. Additional larvae eat the contaminated foliage, and the infection cycle continues (Cory & Myers 2003). Over time, ultraviolet light exposure causes virus particles to degrade (Miller 1997). To investigate the effect of global warming on disease transmission, we conducted a series of field and laboratory experiments where the fall armyworm, *Spodoptera frugiperda*, serves as the host and its species-specific fatal baculovirus, Spodoptera frugiperda nucleopolyhedrovirus (SfNPV), serves as the pathogen.

Materials and methods

THE SYSTEM

The multivoltine fall armyworm is polyphagous and overwinters in Florida and Texas (Pitre & Hogg 1983). The pupae cannot survive freezing temperatures. During the spring, the fall armyworm reinvades the northern areas of its range as it migrates northward. The life cycle begins with adult females laying eggs in clusters. After the eggs hatch, there are six larval instars that collectively last 14–30 days depending upon temperature (Pitre & Hogg 1983). Fall armyworms then pupate for 7–37 days also depending upon temperature (Sparks 1979), emerge to mate and continue their life cycle. The species, like many lepidopterans, exhibits boom and bust dynamics, which have been recorded as early as 1845 (Hinds & Dew 1915). As the population increases in size during the boom phase, fall armyworm infestations occur, which can be large and widespread (Fuxa 1982).

For the fall armyworm, SfNPV represents an important mortality source (Richter, Fuxa & Abdelfattah 1987). Prior to an epizootic, a viral reservoir in the soil provides the initial inoculation of baculovirus into the system (Fuxa & Geaghan 1983). After 4–6 days, initially infected first-instar larvae die (De Oliveira 1999), while uninfected larvae grow to third or fourth instars (Pitre & Hogg 1983). The older instars become infected by consuming the contaminated foliage on which the first instars have died.

In baculovirus systems, virus transmission is primarily dependent upon the host consuming leaf tissue on which the virus resides (Miller 1997). For our experiments, we used soybean (*Glycine max*), a common food source for the fall armyworm (Richter, Fuxa & Abdelfattah 1987). Soybean plants self-pollinate and produce genetically similar offspring. Therefore, using a single soybean genotype (Gasoy 17) allowed us to examine disease transmission without being concerned about differences in plant quality.

THE FIELD EXPERIMENTS

To quantify the effects of warming temperatures on a single round of disease transmission, we manipulated temperature and virus-killed cadaver density within individual plots. The 40 1-m^2 plots, which were separated from each other by at least two metres, were each randomly assigned a temperature and virus (i.e. number of virus-killed cadavers) treatment. The experiment was conducted three times, once during 2010 and twice during 2011, at LSU's Burden Research Center, Baton Rouge, LA (30°24′N, 91°06′W). The Burden Research Center is affiliated with the LSU AgCenter and contains 440 acres of mixed forest and fields. The experimental plots were set up in a mowed field with natural grass cover.

To increase temperatures in the 20 warmed plots, we placed an open-top chamber (OTC) over the entire plot (See Fig. S1, Supporting information). The chambers were made with plexiglass plates (Solar Components Corporation, Manchester, NH, USA) that slant inward to focus solar energy within the plot (Marion *et al*. 1997). A single OTC consisted of four trapezoidal plates held together by metal brackets at each corner. Given our OTC design, a cage control would consist of the metal brackets used to attach the plexiglass plates together. Given the relatively small size of the brackets, we felt a cage control was unnecessary.

In a subset of the plots, we placed iButtons (Maxim Integrated, San Jose, CA, USA). The iButtons were placed in a small mesh bag made of the same material enclosing the individual plants used in the experiment (see below). The bag containing the iButton was then placed at the base of the plant just below the point on the stem where the mesh bag enclosing the plant ended and near the top of the plot's vegetation (See Fig. S1, Supporting information). The placement of the iButtons, along with being enclosed in a mesh bag of its own, minimized the chance that the individual iButtons were exposed to direct sunlight, which can cause unrealistic spikes in temperatures. The buttons measured temperature, and a subset measured humidity at 15-min intervals during the experiment. The iButton data allowed us to determine the extent to which the OTCs raised temperature and humidity in experimental warming plots as compared to control plots.

The experiments were designed to measure disease transmission and replicate, as closely as possible, natural virus transmission. In each plot, we placed a 4-week-old soybean plant, which had been grown at 28 °C in a laboratory growth chamber. Each plant had approximately five to six trifoliate leaves. Any extra leaves were trimmed to ensure that all plants had approximately the same general leaf area. For most plants used in our experiments, no leaves were removed while other plants had, at most, two leaves removed. In our experiments, we followed Underwood *et al*. (2000) and assumed that limited mechanical damage will not induce a soybean plant to produce chemical defences. Additionally, to err on the side of caution, we chose a soybean variety, Gasoy 17, that Underwood, Rausher & Cook (2002) classified as having no induced defences. Thus, even if the plants were induced due to any mechanical damage, their inductive response would be limited.

To manipulate the amount of virus, we varied cadaver density on each plant. Either 0, 15, 30 or 60 infected first-instar larvae

(i.e. future cadavers) were placed on each plant. The infected larvae treatments were assigned in equal proportions across control and warmed plots. By using infected first-instars rather than spraying a set amount of virus on the plant, the virus would be spread about the leaf tissue in a manner close to that seen in natural environments.

To infect larvae, we allowed recently hatched larvae to feed on artificial diet that had been inoculated with SfNPV derived from field-collected fall armyworms. The infected fall armyworms were collected from corn fields in Southeastern Louisiana near Hammond by Jim Fuxa and Art Richter (A. Richter, pers. comm.). We processed the field-collected virus by feeding fifth-instar larvae a cube of diet inoculated with a homogenized mixture of the tissue from a single armyworm. After the infected individuals died, but before they lysed, we extracted the virus from each individual by grinding the cadaver in a 1·5-mL eppendorf tube with 750 μL of deionized water. We centrifuged the cadaver mixture for 5 min at 1 G to remove the supernatant, which contains the OBs. We resuspended the course debris by adding another 750 μL of deionized water, recentrifuged the mixture and extracted the supernatant. The supernatant was then spun at 20 000 G for 10 min to pellet out the OBs. We discarded the supernatant and resuspended the OBs. A single cadaver can produce upwards of 2×10^9 OBs ml^{-1} (Valicente *et al.* 2013).

To inoculate the diet, we placed 9 μL of 10^6 OBs per 3 μL solution on the surface of two ounce diet cups. Once the solution had dried, we placed recently hatched neonates or first-instar larvae on the diet. Given the large number of OBs from a single larva, we used OBs derived from one larva for the October 2010 experiment and an additional larva for the July and September 2011 experiment. The virus from each larva used was derived from a single collected fall armyworm. Ideally, we could have used and amplified plaque-purified virus to control for differences in the pathogen across the experiments. However, none was available to us.

The first-instar larvae were, then, allowed to feed for 2 days on the virus-infected diet. After 2 days, they were checked for infection. Infected neonates can be readily distinguished from non-infected individuals as infected neonates appear bloated and non-infected neonates by that time have moulted to the next instar. Once infected larvae were placed on a plant, the plant was enclosed in a mesh bag (Econet, Hummert, Springfield, MO, USA).

After a sufficient period of time had elapsed to ensure infected larval death (2–4 days), 20 healthy laboratory-reared and recently moulted fourth instars were placed in the bag and allowed to feed. After feeding, the larvae were recaptured and reared in individual cups of artificial diet until death or pupation. The same experimental methodology has been used to gain insight into gypsy moth transmission dynamics (Dwyer, Elkinton & Buonaccorsi 1997; Elderd *et al.* 2013).

In 2010, the fourth instars were allowed to feed for 4 days. For the other two experimental trials in 2011, the larvae fed for 2 days. Although no plants were completely defoliated in 2010, which could affect the analysis, we decided to decrease the number of days spent feeding to guard against complete defoliation given the potential for warmer temperatures in July and September of 2011 as compared to October 2010. In the analysis, we account for differences in the experimental times. After bringing the larvae back to the laboratory, we recorded the number of infected and healthy larvae recovered from each plot. Infection was confirmed by either liquefaction of the host by the virus or under a light microscope where OBs were clearly visible (Cory & Myers 2003). Controls without virus-infected larvae were used to quantify background infection rates. All larvae used in the experiment were from a colony maintained by Bio-Serv (Frenchtown, NJ, USA) or by Dr. Mike Stout at LSU. As the larvae were brought back into the laboratory before they lysed, the above experiment measured a single round of disease transmission.

MECHANISTIC MODEL OF DISEASE TRANSMISSION FOR THE FIELD EXPERIMENTS

Disease ecologists have long relied on mathematical models to understand and describe disease transmission (Kermack & McKendrick 1927; Anderson & May 1980; Briggs & Godfray 1996). We use a well-tested mechanistic mathematical model of baculovirus transmission (Dwyer, Elkinton & Buonaccorsi 1997; Elderd, Dushoff & Dwyer 2008; Fuller, Elderd & Dwyer 2012) to examine how temperature affects baculovirus infection rates in fall armyworm larvae. The model is a modification of the well-known susceptible-exposed-infected-removed or 'SEIR' model (Keeling & Rohani 2008), extended to allow for host heterogeneity in larval infection risk. Given our experimental set-up, we simply needed to keep track of the change in susceptibles over the course of the experiment (See the Supporting information for the full model). Traditionally, SEIR models assume that the change is linear over time such that:

$$\frac{dS}{dt} = -\beta SV. \qquad \text{eqn 1}$$

Here, β is the transmission rate of the disease. S and V represent the density of uninfected or 'susceptible' hosts and virus-killed cadavers, respectively. Eqn 1 assumes that all individuals are equally susceptible to the virus. When individuals vary in their risk of contracting the disease, eqn 1 becomes:

$$\frac{dS}{dt} = -\bar{\beta} SV\left[\frac{S(t)}{S(0)}\right]^{K^2}. \qquad \text{eqn 2}$$

The above equation assumes that the transmission rate follows a probability distribution with mean transmission rate $\bar{\beta}$ and coefficient of variation K, which describes the variability about the transmission rate. Heterogeneity in risk is captured by the transmission scaling factor $\left[\frac{S(t)}{S(0)}\right]^{K^2}$, which starts at one and declines as the number of susceptibles decreases during the epizootic from time 0 to time t. As K increases, the population becomes more heterogeneous. This results in transmission between individuals declining more rapidly when compared to smaller values of K. Due to this decline in transmission, the greater the population's heterogeneity in risk to the virus the lower the final fraction of infected individuals (Fig. 1). Conversely, as heterogeneity decreases, $K \to 0$ and eqn 2 becomes eqn 1.

Due to the fact that the field experiments take place in an enclosed environment, there is no change in the density of the virus or the host. Given this, we set $V = V_0$ and solve eqn 2:

$$\frac{S(T)}{S(0)} = \left(1 + K^2 \bar{\beta} V_0 T\right)^{-1/K^2} \qquad \text{eqn 3}$$

where T is the time at the end of the experiment, so that $S(T)/S(0)$ is the fraction of uninfected larvae and V_0 is the initial

virus-killed cadaver density. By fitting this equation to the data from field transmission experiments, we can estimate the mean transmission rate $\bar{\beta}$ and the variability in transmission K. If we assume that there is no heterogeneity in the population, the above equation becomes $S(T)/S(0) = \exp(-\beta V_0 T)$. This is the solution for eqn 1 when integrating from 0 to T. Using the experimental data to calculate the fraction infected, $1-S(T)/S(0)$, we can compare between models that assume no heterogeneity between individuals to models that assume heterogeneity exists. As we show, heterogeneity is important for describing disease transmission. Given that, we can then determine whether or not temperature changes the mean transmission rate $\bar{\beta}$, the coefficient of variation associated with the transmission rate K, or both.

THE ANALYSES

First, we calculated the effects of the OTCs on temperature and humidity using the iButton data. For each of the three experimental trials, we calculated the daily average of daytime and night-time temperatures as well as humidity. Humidity measurements were transformed using the empirical logit (Warton & Hui 2011). The effects of the OTC treatment on the daily and nightly averages were analysed using a mixed-effect repeated measures analysis of variance (rmANOVA), in which plot was treated as a random effect. All assumptions of the analysis were met (Pinheiro & Bates 2004). Each of the experimental trials were analysed separately, as was true for all analyses conducted.

To test for the effects of temperature on transmission, we fit a suite of candidate models (Table 1) to the data using eqn 3 and its linear counterpart. If there was virus mortality in the controls, we used Abbott's method to correct the data (Morgan 1992). We assumed a binomial error distribution (McCullagh & Nelder 1989) to calculate the likelihood of the data. To choose which model best fits the data, we used the small sample correction of the Akaike Information Criterion, AICc. AICc scores, in turn, were compared using ΔAICc and AIC weights. ΔAICc was defined as the difference between the AICc score and the lowest AICc score of the models being compared. Thus, the best-fit model had a ΔAICc of zero. ΔAICc scores were used to calculate the AICc weights associated with each model, which was defined as the weight of evidence for a particular model given all models considered (Burnham & Anderson 2002). By comparing across models, we tested whether increased temperature changed the mean transmission rate $\bar{\beta}$, the coefficient of variation K associated with transmission or both. Given the best-fit model for each experimental trial, we then bootstrapped estimates of $\bar{\beta}$ and K using 10 000 bootstrap samples (Efron & Tibshirani 1998). This allowed us to estimate 95% confidence intervals (CI) for the transmission parameters.

In the light of the best-fit models (Table 1), we examined the relationship between the model parameters and temperature across the three separate experiments we conducted. We regressed the log-transformed value of the five estimates of K and the untransformed estimates of $\bar{\beta}$ obtained from the experiments against either the daytime or night-time average temperatures. As the relationship between daytime temperature and K was significant, we calculated the 95% CI associated with the slope of the regression using the 10 000 bootstrapped estimates of K for the best-fit models and calculated the resulting slope. This as well as all other analyses was conducted in R (R Development Core Team 2011).

Table 1. Number of parameters (k), AICc scores, ΔAICc and AICc weights for each experiment. For the models considered, climate effect is due to OTCs raising temperatures in the experimental plots. CV > 0 assumes heterogeneity in disease risk. When CV = 0, no difference in risk is assumed such that every larva is equally at risk. $\bar{\beta}$ refers to estimates of mean transmission rate. Best-fit model is in bold. Note that CV is equivalent to K in eq. 2.

Model	k	AICc	ΔAICc	AICc wt
October 2010				
No climate effect with CV = 0	1	29·2	8·0	0·01
No climate effect with CV > 0	2	24·2	3·0	0·09
Climate effect, difference in $\bar{\beta}$ with both CVs = 0	2	28·0	6·8	0·01
Climate effect, difference in $\bar{\beta}$ and CV with both CVs > 0	4	23·7	2·5	0·12
Climate effect, difference in $\bar{\beta}$ and CV with control CV = 0	3	26·0	4·8	0·04
Climate effect, difference in $\bar{\beta}$ and CV with treatment CV = 0	3	25·9	4·7	0·04
Climate effect, difference in CV only	**3**	**21·2**	**0·0**	**0·42**
Climate effect in $\bar{\beta}$ only	3	22·1	0·9	0·27
July 2011				
No climate effect with CV = 0	1	43·1	4·8	0·04
No climate effect with CV > 0	**2**	**38·3**	**0·0**	**0·46**
Climate effect, difference in $\bar{\beta}$ with both CVs = 0	2	45·4	7·1	0·01
Climate effect, difference in $\bar{\beta}$ and CV with both CVs > 0	4	42·0	3·7	0·07
Climate effect, difference in $\bar{\beta}$ and CV with control CV = 0	3	47·0	8·7	0·01
Climate effect, difference in $\bar{\beta}$ and CV with treatment CV = 0	3	40·9	2·6	0·13
Climate effect, difference in CV only	3	40·6	2·3	0·15
Climate effect in $\bar{\beta}$ only	3	40·7	2·4	0·14
September 2011				
No Climate effect with CV = 0	1	57·8	10·2	0·00
No Climate effect with CV > 0	2	52·7	5·1	0·03
Climate effect, difference in $\bar{\beta}$ with both CVs = 0	2	52·5	4·9	0·04
Climate effect, difference in $\bar{\beta}$ and CV with both CVs > 0	4	50·1	2·5	0·12
Climate effect, difference in $\bar{\beta}$ and CV with control CV = 0	3	52·8	5·2	0·03
Climate effect, difference in $\bar{\beta}$ and CV with treatment CV = 0	3	50·3	2·7	0·11
Climate effect, difference in CV only	**3**	**47·6**	**0·0**	**0·42**
Climate effect in $\bar{\beta}$ only	3	48·6	1·0	0·25

THE LABORATORY EXPERIMENTS

Since the baculovirus must be ingested along with the leaf tissue on which it resides for an infection to occur, we also examined how leaf consumption rates changed under increased temperature. We chose two temperatures that correspond to the current July average temperature, 28·9 °C, and projected 2099 temperature, 33·5 °C, for Baton Rouge, LA (NOAA Geophysical Fluid Laboratory Climate Model 2.1, Scenario A2), which represents a 4·6 °C difference. We reared 37 larvae in total to the fourth instar and presented them with a pre-measured soybean leaf

(CI202 Leaf Area Meter, CID) from the Gasoy 17 variety. The larvae were placed in two growth chambers. One maintained the temperature at the current average and the other at the projected temperature. For this experiment, we were limited in the number of chambers available and only had access to two. During the experiment, larvae fed for 8 h at the treatment temperatures, a period of time sufficient to ensure that the larvae did not eat the entire soybean leaf. The leaf consumption rate was estimated as the difference between the post-feeding leaf area minus the pre-feeding leaf area divided by time. The data, which met all analysis assumptions (Faraway 2006), were analysed using a linear model with temperature as the response variable.

As development rate could also affect baculovirus transmission dynamics due to sloughing of the host's midgut cells prior to moult (Engelhard *et al.* 1994), we conducted a separate experiment on development rate. For this experiment, we reared up to 240 larvae from hatch to pupation under current and projected temperatures in four chambers with 60 larvae per chamber. Two chambers were maintained at the current average temperature, and two were maintained at the projected temperature. During rearing, the chambers were set to a 16-h day and 8-h night cycle. We recorded time to pupation, weight at pupation and sex of the pupa. Time to pupation was log-transformed prior to analysis. The data, which met all analysis assumptions (Pinheiro & Bates 2004), were analysed using a linear mixed-effects model with temperature as a fixed effect and growth chamber as a random effect. As we measured two response variables for each individual, we used a standard Bonferroni correction (Faraway 2006).

Results

The open-top chambers or OTCs significantly increased daytime temperatures (Fig. 2 and Table S1, Supporting information). During the night, the OTCs warmed the plots, but only during the September 2011 experiment was there a significant increase in night-time temperature. Average daytime temperatures in the OTCs increased by 2·1 °C, 1·6 °C and 4·4 °C in the October 2010, July 2011 and September 2011 experiments, respectively. In September 2011, night-time temperatures increased by 1·1 °C. There was little effect of the OTCs on humidity (see Table S2, Supporting information) with a only marginally significant increase in humidity during the daytime in September 2011 (Warmed mean: 51%; 95% CI: 50·5%, 54·2%; Control mean: 42%; 95% CI: 37·8%, 46·3%). In general, July 2011 had much lower temperatures than either October 2010 or September 2011 (Fig. 2). This was due to unusually cool and cloudy conditions during July 2011. Because temperatures were forecasted to increase up to 5 °C at the experimental site by 2099 (Karl, Melillo & Peterson 2009), the temperature increases across the experimental treatments were within realistic bounds of current and future projections.

Transmission, measured by the natural log of the fraction infected, increased under warmer temperatures. During October 2010 and September 2011, when daytime temperatures for control and warmed plots were much higher than July 2011, over 90% of the AICc weights were accounted for by models that had temperature as a factor (Table 1). The best-fit model assumed that the mean transmission rate between the control and warmed plots did not differ. Instead, the best-fit model showed that the differences between control and warmed plots were due to a decrease in heterogeneity. A decrease in heterogeneity resulted in individuals having more similar transmission rates in the warmed plots as compared to control plots. The models that included differences in heterogeneity represented 62% and 68% of the AICc weights for the October 2010 and September 2011 experiments, respectively. Thus, by decreasing heterogeneity and not mean transmission rates, infection increased under warmer temperatures (Fig. 3a and c). There was also support for temperature effects on mean transmission rate, $\bar{\beta}$ with ΔAICc values close to 1·0 (Burnham & Anderson 2002).

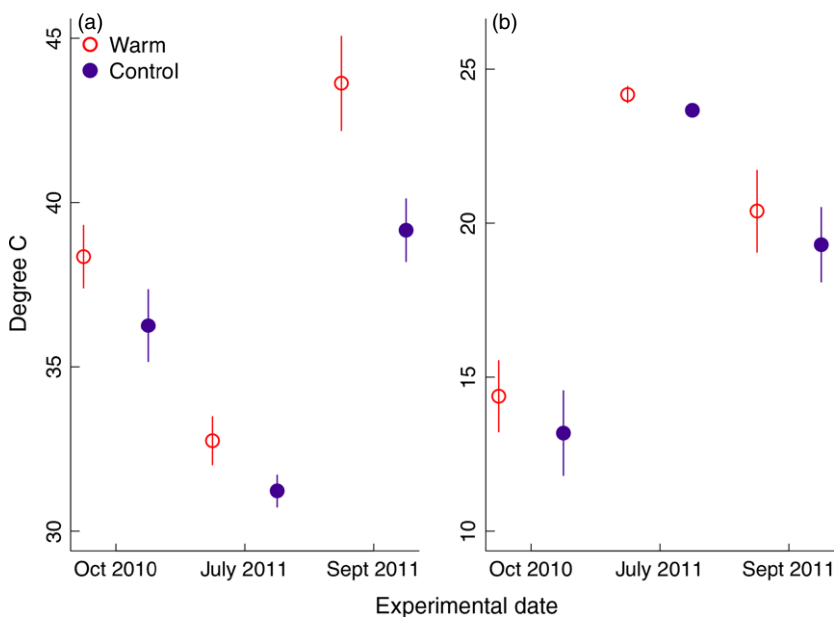

Fig. 2. Average temperatures (°C) in control (closed circles) and warmed (open circles) treatments during the (a) day (10 am–4 pm) and (b) night (10 pm–4 am).

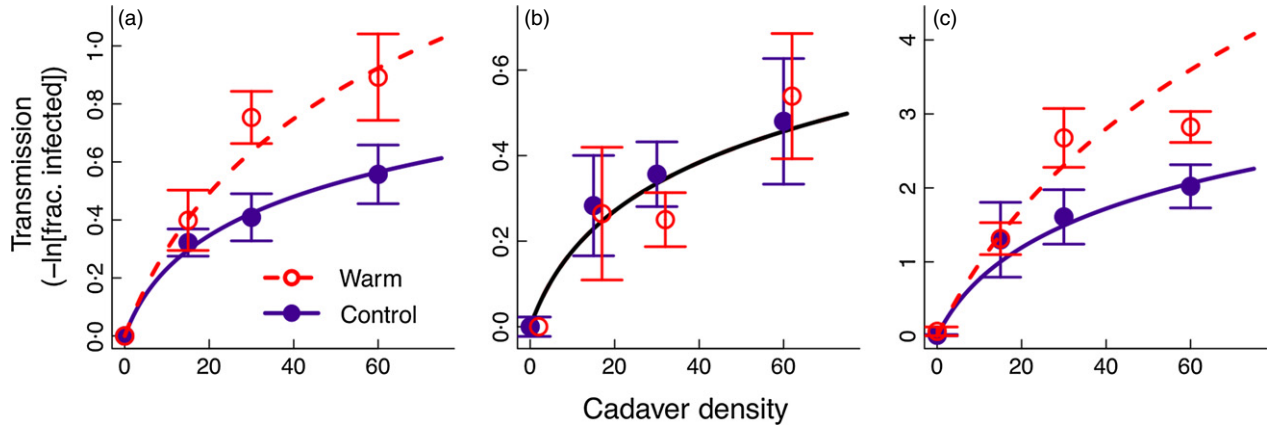

Fig. 3. Effects of the experimental manipulation of cadaver density (i.e. virus-killed first instar larvae) on the fraction infected in the control (closed circles) and warmed (open circles) treatments for (a) October 2010, (b) July 2011 and (c) September 2011. The lines represent the best-fit model in Table 1 for each date. For July 2011, the points are jittered for sake of clarity, and a single line represents the best-fit model of no difference between control and warmed treatments. For September 2011, five plots in the warmed treatment contained no survivors, which would result in a negative log of infinity. For plotting, but not analytical purposes, the per cent survival was adjusted (Collett 2003). Note differences in *Y*-axes.

During July 2011, when temperatures in general were cooler, the null models accounted for 50% of the AICc weights (Table 1 and Fig. 3b). Overall, when temperature differences were greater between control and warmed plots (i.e. October 2010 and September 2011), transmission increased in the warmed plots due to a decrease in heterogeneity of risk.

While there was a great deal of evidence that warming affected disease transmission (Fig. 3), whether the effect was due to changes in transmission rate $\bar{\beta}$, heterogeneity in transmission rate K or both was less clear given the direct comparison of the AIC values (Table 1). However, bootstrapped values of K and $\bar{\beta}$ associated with the best-fit model along with their degree of overlap provided additional evidence that heterogeneity in the transmission rate could be of greater importance. For both the October 2010 and September 2011 experiments, the parameter estimate for the coefficient of variation K decreased as temperatures rose, which had a large effect on transmission dynamics by increasing the fraction infected (Fig. 3a and c). Additionally, the difference in the 95% CI of the bootstrapped estimates of K between warmed and control treatments did not overlap with zero (October 2010 median: −0·75; 95% CI: −1·178, −0·357; September 2011 median: −0·42; 95% CI: −0·804, −0·104). The above results lent further support for experimental transmission differences arising from a decrease in heterogeneity of risk in warmed plots. Across experimental trials, the estimate for $\bar{\beta}$ was higher for September 2011 (0·057; 95% CI: 0·038, 0·172) than October 2010 (0·010; 95% CI: 0·006, 0·039) with some overlap in the 95% CI. Moreover, the disease transmission rate estimates were of the same magnitude, and the effects of temperature on heterogeneity estimates were much greater. In general, as temperatures rose, a larger fraction of individuals became infected driven by a decrease in the coefficient of variation K associated with heterogeneity of risk.

When examining the coefficient of variation across all experimental trials, increasing temperatures decreased the logarithm of the coefficient of variation K (Fig. 4a, $F_{1,3} = 18{\cdot}50$; $P = 0{\cdot}02312$; Fig. 4b, slope: −0·11; 95% CI: −0·216, −0·030). Therefore, as temperature increased, K decreased at an exponential rate. There was no effect of daytime temperature on $\bar{\beta}$ ($F_{1,3} = 2{\cdot}96$; $P = 0{\cdot}1839$). The

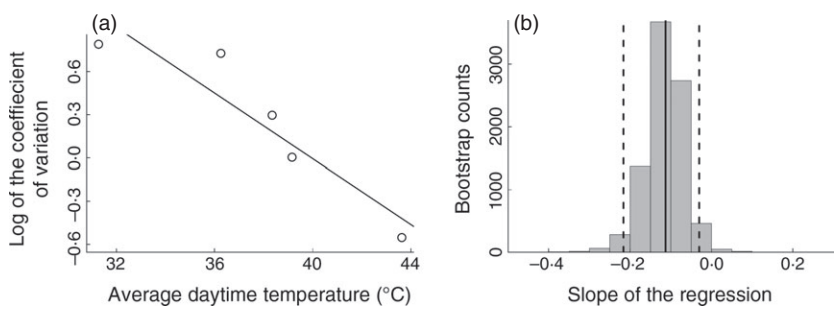

Fig. 4. (a) Effects of average daytime temperature on the log-transformed coefficient of variation K for the disease transmission rate and (b) histogram of the slope from bootstrapped estimates of K. The vertical solid line is the median, and the dashed vertical lines are the 95% confidence intervals.

effects of changes in the coefficient of variation could be best seen by examining how infection rates change as virus-killed cadaver density increases. Across experimental trials, as temperature rose, the fraction infected increased for a given pathogen-killed cadaver density (Fig. 5). In Fig. 5, any increase in transmission rate $\bar{\beta}$ across experimental trials resulted in a decrease in the lowest density at which an epizootic occurs. The small increase in $\bar{\beta}$ across trials would result in an epizootic starting with a smaller amount of virus in the system. The decline in the coefficient of variation or heterogeneity in transmission, on the other hand, causes the infection rate to increase more rapidly under increased temperatures. Together, these results show that as temperatures increased, outbreak intensity also increased by decreasing individual differences in disease risk.

In the laboratory experiments, feeding rates increased [mean (SE) for current: 0·37 (0·0412) $cm^2\ h^{-1}$; mean (SE) for projected: 0·53 (0·0481) $cm^2\ h^{-1}$; $F_{1,35} = 6·7837$, $P = 0·0134$; See Fig. S2, Supporting information] and developmental time decreased (mean (95% CI) for current: 11·6 days (11·55, 11·73); mean (95% CI) for projected: 9·1 days (9·01, 9·11); Table 2; See Fig. S3A, Supporting information) as temperature increased. Interestingly, the decrease in developmental time did not affect pupal weight (mean (95% CI) for current: 0·186 grams (0·1785, 0·1940); mean (95% CI) for projected: 0·188 grams (0·1828, 0·1939); Table 2, See Fig. S3B, Supporting information). This latter result ran counter to the temperature–size rule, whereby increased temperatures should result in decreased body mass (Angilletta 2009). For both developmental time and pupal weight, the effect of sex and the interaction between sex and temperature were not significant (Table 2). In general, increased temperatures affected both feeding behaviour and developmental times.

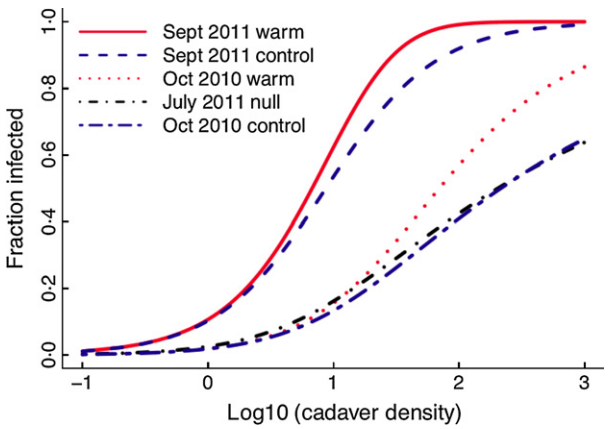

Fig. 5. The effects of the experimental treatments on fraction infected during an epizootic. The increase in fraction infected across dates and treatments is driven by a decrease in the transmission rate's coefficient of variation. To calculate the fraction infected, the best-fit model for each experiment was used. For example, we used the null model (i.e. no difference between treatments) to construct the July 2011 Null line.

Table 2. Effects of current and 2099 projected July average temperatures in Baton Rouge, LA, on time to pupation and weight at pupation

Response variable	Effect	d.f.	*F*-value	*P*-value
Time to pupation	Temperature	1,2	1105·8	0·0009
	Sex	1,209	1·5	0·2255
	Temperature*Sex	1,209	0·0303	0·8261
Weight at pupation	Temperature	1,2	0·182	0·7714
	Sex	1,209	2·070	0·1517
	Temperature*Sex	1,209	0·674	0·4125

Discussion

One reason why the effects of climate change on disease dynamics may be particularly hard to resolve is the lack of empirical data associated with disease transmission and climate change (Pascual & Bouma 2009). Using a host–pathogen system in which empirical data could be easily gathered, we showed that increasing temperatures increased disease transmission and outbreak intensity. While there was support for differences in transmission rate (Table 1), the change in intensity, such that a greater fraction of individuals were infected under warmer temperatures, most likely resulted from a decrease in the heterogeneity of disease risk. In general, as temperatures rose, the coefficient of variation in disease transmission declined, whereas the mean transmission rate remained relatively constant. This effect becomes especially distinct at high cadaver densities (Fig. 5). Thus, warmer temperatures by decreasing differences in disease risk among individual larvae largely contributed to an increase in outbreak intensity.

Increased leaf consumption under warmer temperatures (See Fig. S2, Supporting information) may also play a role in declining heterogeneity of risk. For most lepidopteran baculoviruses, the host consumes foliage on which the virus is residing (Miller 1997). As consumption increases, the larvae have a greater likelihood of encountering and consuming a lethal dose of virus. Perhaps, the individuals at the lowest risk in a population are those that eat less leaf tissue under cooler temperatures. Under warmer temperatures, their consumption rates increase leading to higher infection rates among the low-risk subgroup, thereby decreasing overall population heterogeneity. Yet, this assumes that larvae at the other end of the distribution (i.e. those that eat a great deal already) are at an upper limit to feeding rates. Given that we only used two temperatures, we did not test this assumption, but the data could be easily obtained. In general, changes in host activity in other pathogen-driven systems may result in similar dynamics.

For the feeding rate experiment, we only had two growth chambers available. Thus, the increase in feeding under warmer temperatures could have also resulted from differences between growth chambers. However, the measured random effects associated with the pupation

experiments, which were conducted in four growth chambers, were relatively small given the non-significance of the likelihood ratio test (LRT) between a model that contained a random effect for chamber and one that did not (LRT time to pupation ratio = $6{\cdot}65 \times 10^{-9}$, $P = 0{\cdot}9999$; LRT weight at pupation ratio = $2{\cdot}44 \times 10^{-8}$, $P = 0{\cdot}9999$). Thus, the potential effect of the growth chamber on our feeding rate results should also be relatively low.

Insect physiology may also be important for determining infection rate as it can affect the probability of becoming infected after consuming the pathogen. A higher rate at which midgut cells are sloughed decreases the probability of infection because sloughing eliminates the pathogen from the host (Engelhard *et al.* 1994). Temperature can increase sloughing rate by decreasing developmental time (See Fig. S3A, Supporting information) due to the fact that the midgut is sloughed prior to moulting to the next instar (Engelhard *et al.* 1994). Thus, changes in developmental rate also represent a potential mechanism that may affect disease transmission dynamics under warmer temperatures.

In general, both changes in feeding rate and the physiological response of the larvae can alter infection risk. Whether the change in risk based on feeding behaviour or insect physiology will result in differences in transmission rate or declines in heterogeneity of risk needs to be a subject for further study. Given that warmer temperatures will affect epizootic intensity and disease transmission, a natural progression is to more fully examine what mechanisms cause these changes. Investigating the mechanism or mechanisms responsible will help link individual physiology and development with population and community dynamics as the climate warms (O'Connor, Gilbert & Brown 2011).

Interestingly while developmental time decreased, final pupal weight did not decrease, which does not support the temperature–size rule (Angilletta 2009). However, there are a number of exceptions to this rule (Angilletta & Dunham 2003; Kingsolver 2009) with a great number occurring in the Lepidoptera (Atkinson 1994). However, the relationship that we found is inferred by two temperature points, which limit our ability to definitively say that the fall armyworm does not follow the temperature–size rule. Yet, given the trend in Lepidoptera, the fall armyworm may be another example where increased temperature does not result in decreased body size.

While the field experiments were subject to natural variability in temperature, the laboratory experiments were conducted using the current mean and 2099 projected mean temperatures. Others have shown the importance of considering temperature variability and its impact on disease transmission (Duncan, Fellous & Kaltz 2011; Yakob & Mumby 2011; Ben-Horin, Lenihan & Lafferty 2013). Future work, in this system and others, should examine how both changes in the mean and variation about the mean temperature affect not just the host but also the pathogen and the host–pathogen interaction. For example, baculovirus-infected tent caterpillars die more quickly at higher temperatures (Frid & Myers 2002), which can alter epizootic dynamics. Alternatively, increased temperatures could act similarly to physiological or behavioural fevers, which can increase recovery rates (Angilletta 2009). In general, investigating both changes in the mean temperature and the variability about the mean would provide additional insight into how temperatures may be affecting multiple facets of disease transmission.

A potential limitation of our results stems from how we measured temperature. In our experiments, the temperature recorded in each plot does not directly measure the temperature of the organism. Ideally, we would have directly measured internal temperature or placed thermistor wires next to the animal to get a closer estimate of the larva's internal temperature (Porter 1982; Frid & Myers 2002). Given that the transmission experiments were conducted in a sewn mesh bag, the logistics of using other methods for recording temperature proved difficult. However, the organism's temperature would be expected to track the ambient temperature except at temperature extremes near the organism's thermal maximum (Frid & Myers 2002). Instead of true measurements of the host's temperature, the iButtons used in the plots measured ambient temperature, which served as a measure of the relative difference in temperature between individual larva in the control and warmed plots.

We also have examined changes in disease transmission over the course of a single transmission event. Whether these changes in physiology or development lead to more frequent or less frequent outbreaks over the long term (i.e. multiple generations) will depend upon host, pathogen and host–pathogen responses to increased temperatures. For instance, host fecundity will most likely be affected by rising temperatures (Crozier & Dwyer 2006). Additionally, we have focused our efforts on understanding transmission dynamics from the host's perspective. Changes in temperature and environmental conditions can also affect the pathogen (Fuller, Elderd & Dwyer 2012; Paaijmans *et al.* 2012). Lastly, while we have focused on temperature, other abiotic factors affected by climate change such as precipitation (Karl, Melillo & Peterson 2009) may either amplify or depress the effects of temperature on disease transmission. Examining the effects of increased temperatures and other abiotic factors over longer temporal scales will add understanding to how climate change will affect both the short-term dynamics of disease transmission and the long-term dynamics of the host population.

It may also be important to consider an organism's phenotypic plasticity in response to climate change and how plasticity affects transmission dynamics. It is well known that phenotypic plasticity in lepidopteran larvae affects the susceptibility of the larvae to pathogen infection. For instance, *Spodoptera exempta*, the African armyworm, has distinct phenotypes that differ depending upon

whether the larvae are reared alone or in groups. The larvae reared in groups show increased melanin and phenoloxidase production (Wilson *et al.* 2001). The increase of these two compounds results in decreases in baculovirus infection and parasitic rates (Reeson *et al.* 1998, 2000; Wilson *et al.* 2001). Thus, as density increases, the larvae exhibit density-dependent prophylaxis by investing in immune responses that may inhibit disease transmission. However, the opposite may also occur as increased densities can result in increased transmission due to larval stress as seen with the gypsy moth (Reilly & Hajek 2008). Interestingly, larvae in the *Spodoptera* genus also have the ability to self-medicate by modifying their diet (Lee *et al.* 2006). In our experiments, an increase in temperature may have resulted in a decrease in the phenotypic plasticity of responses available to an individual organism as the organism is pushed towards the upper limit of its thermal tolerance. To determine whether the fall armyworm is near its upper limit requires additional experiments with an increase in the number of temperatures tested. To understand the extent to which phenotypic differences play a role in controlling the decline in transmission rate heterogeneity represents an area for future research.

For the host, changes in metabolic rate may also be important for determining transmission dynamics as metabolic rates depend upon the temperature of the environment (Gillooly *et al.* 2001; Angilletta 2009). In other systems, changes in metabolic rates result in changes in consumer–prey (O'Connor, Gilbert & Brown 2011) and host–macroparasite interactions (Molnar *et al.* 2013). For the fall armyworm, decreased developmental times most likely arose due to increased metabolic rates. How the pathogen responds to changes in developmental times and warmer temperatures needs to be examined as well (Rohr *et al.* 2011). In general, incorporating aspects of the metabolic theory of ecology (Gillooly *et al.* 2001; Brown *et al.* 2004; Rohr *et al.* 2011) in a host–pathogen framework represents a promising avenue from both an empirical (O'Connor, Gilbert & Brown 2011) and theoretical (Vasseur & McCann 2005; Molnar *et al.* 2013) perspective.

Clearly, climate change is having dramatic impacts on species demography and distributions (Crozier & Dwyer 2006; Adler & HilleRisLambers 2008; Doak & Morris 2010). Yet, little is known about how rising temperatures will affect disease transmission and intensity due to the limited amount of field data. Our results show that increasing temperatures increase disease transmission between host and pathogen. In our system, the best-supported proximate cause arises from changes in the coefficient of variation associated with host risk, which differs considerably from those usually assumed (e.g. changes in mean transmission rate in Moore *et al.* 2012). This can have important ecological and economic consequences for the short-term and, potentially, the long-term dynamics of disease outbreaks (Lafferty & Holt 2003).

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Supporting Information

Additional Supporting Information may be found in the online version of this article.

Fig. S1. Open-top chamber (OTC) used to raise temperatures in the field experiments.

Fig. S2. The effect of current and 2099 projected July average temperature on the mean feeding rate (±95% confidence intervals) of fourth instar larvae.

Fig. S3. Mean (±95% confidence intervals) of A) time to pupation and B) weight at pupation for fall armyworm larvae raised under current and 2099 projected July average temperatures.

Table S1. rmanova results for the effects of Open-Top Chambers (OTCs) on daytime and nighttime temperatures during the field transmission experiments showing the degrees of freedom (df), the *F*-statistics, and the associated *P*-value.

Table S2. rmanova results for the effects of Open-Top Chambers (OTCs) on daytime and nighttime humidity during the field transmission experiments showing the degrees of freedom (df), the *F*-statistics, and the associated *P*-value.