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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

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 nonmoulting 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.

Ma e a a d e d

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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
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(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 lar-

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.

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 ofs2.7(zero.)]TJ/F19deenef7IC

(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

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

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 = 0·9999; LRT weight at pupation ratio = $2{\cdot}44 \times 10^{-8}$, P = 0·9999). 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

Dwyer, G., Elkinton, J.S. & Buonaccorsi, J.P. (1997) Host heterogeneity in susceptibility and disease dynamics: tests of a mathematical model. American Naturalist, 150, 685–707.
Dwyer, G., Firestone, J. & Stevens, T.E. (2005) Should models of disease dynamics in herbivorous insects include the effects of variability in host-plant foliage quality? American Naturalist, 165, 16–31.
Dwyer, G., Dushoff, J., Elkinton, J.S. & Levin, S.A. (2000) Pathogen-driven outbreaks in forest defoliators revisited: building models from experimental data. American Naturalist, 156, 105–120.
Efron, B. & Tibshirani, R.J. (1998) An Introduction to the Bootstrap. Chapman and Hall, Boca Raton, FL.
Elderd, B.D., Dukic, V.M. & Dwyer, G. (2013) Population-level differences in disease transmission: a Bayesian analysis of multiple smallpox epidemics. Epidemics, 5, 146–156.
Elderd, B.D., Dushoff, J. & Dwyer, G. (2008) Host-pathogen dynamics, natural selection for disease resistance, and forest-defoliator outbreaks. American Naturalist, 172, 829–842.
Elderd, B.D., Haynes, K., Rehill, B. & Dwyer, G. (2013) Interactions between an induced plant defense and a pathogen drive outbreaks of a forest insect. Proceedings of the National Academy of Sciences United States of America, 110, 14978–14983.
Engelhard, E.K., Kammorgan, L.N.W., Washburn, J.O. & Volkman, L.E. (1994) The insect tracheal system – a conduit for the systemic spread of Autographa-californica-m nuclear polyhedrosis-virus. Proceedings of the National Academy of Sciences of the United States of America, 91, 3224–3227.
Faraway, J.J. (2006) Extending the Linear Model with R: Generalized Linear, Mixed Effects and Nonparametric Regressionic
Chapman

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Podgwaite, J.D., Reardon, R.C., Walton, G.S. & Witcosky, J. (1992) Efficacy of aerially-applied Gypchek against gypsy moth (Lepidoptera, Lymantriidae) in the Appalachian Highlands. Journal of Entomological Science, 27, 337–344.

Porter, K. (1982) Basking behavior in larvae of the butterfly Euphydryas aurinia. Oikos, 38, 308–312.

R Development Core Team (2011) R: A Language and Environment for Statistical Computing. R Foundation for Statistical Computing, Vienna, Austria. ISBN 3-900051-07-0. URL http://www.R-project.org/

Reeson, A.F., Wilson, K., Gunn, A., Hails, R.S. & Goulson, D. (1998) Baculovirus resistance in the noctuid Spodoptera exempta is phenotypically plastic and responds to population density. Proceedings of the Royal Society B-Biological Sciences, 265, 1787–1791.

Reeson, A.F., Wilson, K., Cory, J.S., Hankard, P., Weeks, J.M., Goulson, D. et al. (2000) Effects of phenotypic plasticity on pathogen transmission in the field in a Lepidoptera-NPV system. Oecologia, 124, 373–380.

Reeve, J.D., Cronin, J.T. & Strong, D.R. (1994) Parasitoid aggregation and the stabilization of a salt-marsh host-parasitoid system. Ecology, 75, 288–295.

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