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Plant defense against a pathogen drives nonlinear transmission dynamics through both vector preference and acquisition

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Abstract. Host defense against vector-borne plant pathogens is a critical component of integrated disease management. However, theory predicts that traits that confer tolerance or partial resistance can, under certain ecological conditions, enhance the spread of pathogens and spillover to more susceptible populations or cultivars. A key component driving such epidemic risk appears to be variation in host-selection behavior of vectors based on infection status of the host. While recent theory has further emphasized the importance of infection-induced host-selection behavior by insect vectors for plant disease epidemiology, experimental tests on the relationship between vector host-selection preference and transmission are lacking. We test how host plant defense —conferred by the *PdR1* gene complex—mediates vector host-selection preference and transmission of the pathogenic bacterium *Xylella fastidiosa* among grapevine cultivars. We confirmed that *PdR1* confers resistance against *X. fastidiosa* by reducing both pathogen population size and disease severity. We found that vector transmission rates to new hosts exhibited unimodal dynamics over the course of infection when both susceptible and resistant were infected and acted as sources of the pathogen. Transmission from susceptible plants initially increased and then declined as insect vectors avoided severely diseased plants. While transmission from *PdR1*-resistant plants also initially increased and then declined as well, this was not due to avoidance by vectors, although the exact mechanism remains unclear. We show that (1) vector preference changes over the course of disease progression, (2) vector preference is clearly important but a poor predictor of transmission, and (3) the post-latent incubation period—in which plant hosts are infectious but asymptomatic—is likely a key period for vector transmission of *X. fastidiosa*. Our results suggest that, consistent with theory, defensive traits lengthen the duration of the incubation period, increasing *X. fastidiosa* transmission. However, defensive traits may over the long-term ultimately reduce spread possibly through induced resistance. Vector host-selection preference, host resistance, and transmission are clearly dynamic, changing over the course of disease progression. Understanding these dynamics is critical for broader insights into the epidemiology of vector-borne plant pathogens, theory development, and deploying disease-resistant cultivars in an effective and sustainable manner.

Key words: condition-dependent movement; *Graphocephala atropunctata*; host manipulation hypothesis; host resistance; *Vitis arizonica*; *Vitis vinifera*.

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Introduction

Recent emergence events of vector-borne plant diseases have caused widespread socio-ecological harm. Fungal pathogens spread by wood-boring beetles have devastated forests from Hawaiian *Ohi'a lehua* to North American conifer forests, while viruses and bacteria spread by Hemipteran vectors—such as huanglongbing disease of citrus—exacerbate vulnerability and threaten biodiversity of agricultural systems worldwide (Anderson et al. 2004, Cheatham et al. 2009, Hulcr and Dunn 2011, Savary et al. 2012).

Host defense against pathogens in agricultural crops is one of the most successful and durable strategies to manage agricultural diseases (Gilligan 2008). The particular form of host defense—whether resistance or tolerance—can also influence pathogen spread broadly. Resistance alleviates the fitness costs of infection by reducing the pathogen population in host tissues, whereas tolerance alleviates the fitness costs by ameliorating disease symptoms with little effect on pathogen population size (Cooper and Jones 1983). Variation among hosts in tolerance and resistance can be maintained over evolutionary timescales through ecological feedbacks (Best et al. 2008, Brown and Tellier 2011) and has important epidemiological consequences (Dwyer et al. 1997, Laine et al. 2011, Borer et al. 2016). Generally, tolerance traits are predicted to increase the prevalence of a pathogen within a host population, though the mechanism depends on the precise form of tolerance (Roy and Kirchner 2000, Miller et al. 2006, Best et al. 2008). While variation in host defense may play an important role in vector-borne pathogen spread, this question has received relatively little attention in the literature compared with directly transmitted pathogens. Nonetheless, the relationship between defense and transmission for vector-borne pathogens may be substantially more nuanced because of the complexity of host-selection behavior by vectors (Zeilinger and Daugherty 2014).

Many insect vectors of plant pathogens—including viral, bacterial, and fungal pathogens—exhibit preference for feeding on hosts based on the infection status of the hosts and of the vectors themselves (reviewed in Eigenbrode et al. 2018). A recent flourishing of theory has highlighted how different components of host-selection behavior—namely attraction/orientation vs. leaving/departure behaviors—change based on infection status and, in turn, influence pathogen spread (Sisterson 2008, Roosien et al. 2013, Shaw et al. 2017, Donnelly et al. 2019). However, while infection-induced host-selection preference by vectors appears to be common and is predicted to be epidemiologically important, few empirical studies have attempted to test the influence of preference on vector transmission of plant pathogens.

A central challenge in testing the relationship between vector preference and transmission is inducing or measuring variation in vector host choice and transmission concurrently. Some studies have shown that host selection by vectors varies dynamically as disease progresses (Blua and Perring 1992, Werner et al. 2009, Legarrea et al. 2015, Lu et al. 2016, Daugherty et al. 2017), varies positively with host pathogen burden (Legarrea et al. 2015, Lu et al. 2016), or coincides with vector acquisition of the pathogen (Legarrea et al. 2015). However, the only study to our knowledge that showed an empirical relationship between vector preference and transmission to uninfected hosts was conducted by Daugherty et al. (2017), who showed that increasing vector avoidance of *Xylella fastidiosa*-infected grapevines as disease worsened coincided with reduced transmission to healthy host plants across different temperature regimes.

Comparing vector preference and transmission among plant species or genotypes that vary in defense could also offer an opportunity to test for preference–transmission relationships (Legarrea et al. 2015). Tolerant hosts decouple pathogen burden from the phenotypic responses to infection—which are often used by vectors in host selection (Eigenbrode et al. 2018)—and therefore allow for a decoupling of the influences of preference and pathogen population size on transmission. Furthermore, as hypothesized by Zeilinger and Daugherty (2014), the interactions between different forms of defense and vector preference can have important epidemiological implications. Traits conferring resistance against a pathogen should broadly reduce pathogen spread regardless of the particularities of vector preference. On the other hand, pathogen spread can be greatly enhanced when hosts are tolerant

to infection and when vectors avoid diseased hosts. Understanding the precise interplay between host defense and vector transmission can improve disease management because of their influence on the risk of disease spillover from partially resistant or tolerant hosts into nearby susceptible host populations (Sisterson and Stenger 2018).

Xylella fastidiosa (family Xanthomonadaceae) is a xylem-limited bacterium associated with over 500 plant taxa and causes diseases in many crop species, including olive quick decline syndrome, citrus-variegated chlorosis, Pierce's disease of grapevines, and leaf scorch of almond, oleander, and coffee (European Food Safety Authority [EFSA] 2018). The bacterium is transmitted in a propagative persistent but non-circulative manner by xylem-sap-feeding insect vectors in the family Cicadellidae and superfamily Cercopoidea, with all xylem-sap-feeding Hemiptera regarded as potential vectors (Sicard et al. 2018). Cicadellid vector species consistently avoid *X. fastidiosa*-infected symptomatic plant hosts but show no discrimination between infected asymptomatic hosts and uninfected hosts (Marucci et al. 2005, Daugherty et al. 2011, De Miranda et al. 2013).

Pierce's disease costs California viticulture ~ $100 million per year in yield losses and insecticide use to manage vectors of *X. fastidiosa* (Tumber et al. 2014). Such insecticide use potentially harms biodiversity and the provisioning of agroecosystem services (Cheatham et al. 2009, Chagnon et al. 2015). Alternatively, grapevine cultivars have been developed from hybridizing domesticated grape *Vitis vinifera* with a North American native congener *V. arizonica* (Krivanek et al. 2006). Putative resistance is conferred by the *PdR1* quantitative trait locus, and while the precise mechanism of resistance is still unresolved, backcrossed lines harbor much lower populations of *X. fastidiosa* and exhibit negligible disease symptoms (Krivanek and Walker 2005).

While much reduced, population sizes of *X. fastidiosa* in *PdR1* vines can still exceed that required for vector transmission (Hill and Purcell 1997, Krivanek and Walker 2005). Given that symptom development is slow (Krivanek et al. 2005) and insect vectors prefer asymptomatic or healthy hosts, *PdR1* grapevines could act as reservoir hosts of *X. fastidiosa*, enhancing vector transmission among vines and spillover to more susceptible vineyards. We hypothesize that transmission from susceptible grapevines will exhibit a unimodal curve—initially increasing as pathogen burden in infected plants increases, then decreasing as disease symptoms become increasingly severe—leading to vector avoidance and reduced acquisition (Fig. 1). At this later stage, vector transmission from tolerant or partially resistant hosts could exceed that from susceptible hosts.

To test our hypothesis on the epidemic risk from *PdR1* grapevines, we experimentally assessed host-selection preference and transmission dynamics by the efficient cicadellid vector *Graphocephala atropunctata* from inoculated *PdR1*-resistant grapevines and closely related susceptible vines. We compared transmission biology between *PdR1* resistant and susceptible vines over multiple time points and employed structural equation modeling to test which components of the vector transmission process best explained variation in transmission. From this, we show that host susceptibility mediates transmission most clearly through differences in pathogen population size and that host-selection by vector appears to play an important but more minor role.

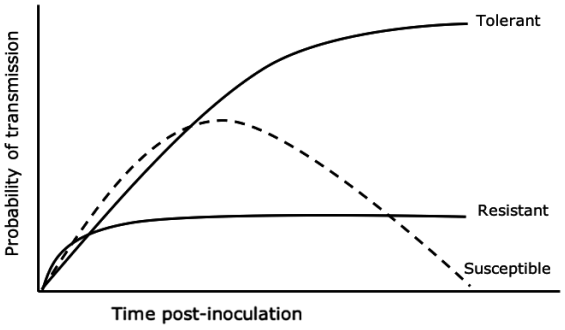

Fig. 1. Qualitative theoretical predictions on vector transmission dynamics in the context of plant host defense (or lack thereof) and vector avoidance of symptomatic hosts. Here, we assume resistance is partial, meaning that the pathogen can colonize the host but population growth is limited. The decline in transmission from susceptible hosts is predicted to correspond to increasing vector avoidance of symptomatic plants.

Materials and Methods

Plants and insects

All resistant and susceptible grapevine genotypes were segregants of a cross between *V. vinifera* cv. Airen and a hybrid of *V. rupestris* × *V. arizonica* (b40-14 background), with resistance conferred by the *PdR1c* gene (Krivanek et al. 2006, Walker and Tenscher 2016). We repeated the transmission experiment over two years: In 2016, we used one susceptible genotype (or line) and one resistant genotype; in 2017, we used two susceptible and two resistant genotypes, with one of the susceptible genotypes being the same in both years. See Appendix S1 for more information on experimental methods.

In both years, transmission trials were conducted with green cuttings grown at the Oxford Tract greenhouses at University of California Berkeley according to the methods described in Appendix S1. Colonies of *G. atropunctata* (Signoret; blue-green sharpshooter, Hemiptera: Cicadellidae) were started from wild-collected individuals and all insects were pre-screened for *X. fastidiosa* infection prior to transmission experiments.

To establish *X. fastidiosa*-infected source plants, we mechanically inoculated 3-month-old resistant and susceptible vines near the base of the main stem with 10 μL of a turbid suspension ($OD_{600} > 1$) of *X. fastidiosa* subsp. *fastidiosa* culture (STL isolate, American Type Culture Collection 700963) in succinate–citrate–phosphate (SCP) buffer as described in Hill and Purcell (1995). Test plants were mock-inoculated with 10 μL of SCP buffer only.

Transmission experiment

In both years, we paired one *X. fastidiosa*-free test plant of a susceptible genotype and one inoculated source plant—either *PdR1* resistant or susceptible—in a tent-style BugDorm cage (61 cm^3, BioQuip Products, Rancho Dominguez, California, USA). At the beginning of the trials, eight *G. atropunctata* adults were introduced into the middle of the cage, equidistant from the two plants. In 2016, we repeated the trials at 3, 8, and 12 weeks post-inoculation; in 2017, we repeated the trials at 2, 5, 8, and 14 weeks post-inoculation. New plants were used in each trial, making each trial independent. Each combination of genotype and weeks post-inoculation was replicated eight times in each year.

Once the *G. atropunctata* vectors were introduced into the cage, we recorded the number of insects on each plant an in neutral space (cage walls and pots) repeatedly at pre-determined times from the start of the trials. In 2016, these times were as follows: 1 min, 5 min, 10 min, 15 min, 30 min, 45 min, 1 h, 2 h, 4 h, 6 h, 24 h, 30 h, 48 h, 3 d, 4 d, 5 d, 6 d, 7 d, and 8 d. In 2017, we used the same observation times but shortened the total duration of the trials to 4 d, because the 2016 data showed that the *G. atropunctata* reached equilibrium in their movements by then (Appendix S2: Figs. S1, S2).

At the end of the trials, we removed all insects and noted the severity of Pierce's disease symptoms on the source plants using the 0–5 symptom severity scoring index developed by Guilhabert and Kirkpatrick (2005). We then estimated population sizes of live *X. fastidiosa* in the source plants by serial dilution culturing (Hill and Purcell 1995). To estimate vector acquisition of *X. fastidiosa*, we assayed the infection status of all *G. atropunctata* in trials using qPCR (Appendix S1). To estimate transmission to uninfected test plants, we assayed the infection status of all test plants by culturing 12 weeks after the end of the trials.

Statistical analysis

We first tested for differences in Pierce's disease symptom severity in source plants with a partial odds ordinal logistic regression model, because the response variable—PD symptom severity index—represented an ordinal categorical variable. We analyzed variation in population size of *X. fastidiosa* in inoculated source plants using a generalized linear model with quasi-Poisson distributed error to correct for over-dispersion. While the quasi-Poisson model showed modest nonconstant error variance, it performed much better than Poisson or negative binomial regression models (results not shown). Both tests included genotypes, weeks post-inoculation, and their interaction as explanatory variables.

We tested for preference of *G. atropunctata* vectors for *X. fastidiosa*-infected source plants or uninfected test plants by estimating attraction and leaving rates from each plant. We fit our repeated-measures count data on *G. atropunctata*

location to the consumer movement model developed by Zeilinger et al. (2014) and generalized to multiple consumers per preference trial by Gray et al. (2020) for each combination of plant genotype and weeks post-inoculation. We fit four variants to our data: (1) the fixed model, in which a single attraction rate and a single leaving rate were estimated for both choices, representing a null hypothesis of no preference between choices; (2) the free attraction model, in which the attraction rates to each plant were free to vary but the leaving rates from each were set equal to each other; (3) the free leaving model, in which attraction rates were fixed but leaving rates were free to vary; and (4) the free choice model, in which both the attraction and leaving rates were free to vary. We compared model variants using Akaike's information criterion corrected for small sample size (AIC_c) and calculated variances for each parameter using the quadratic approximation method (Bolker 2008). We calculated model-averaged parameter estimates and variances using the AIC_c weights of all models with values of $\Delta AIC_c \leq 7$ (Burnham et al. 2011) and calculated 95% confidence intervals (CI) from these model-averaged variances (Zeilinger et al. 2014). We inferred vector preference between host choices (infected source plant vs. test plant) using both model selection and 95% CI.

We analyzed the change in vector acquisition and transmission of *X. fastidiosa* over the weeks post-inoculation by fitting a series of nonlinear models to our data: two unimodal models—Holling type IV and Ricker models—and two saturating models—Holling type II and logistic growth models (Appendix S3: Table S1). We also fit data to a linear model as a null hypothesis to test against our nonlinear model set. We selected the best model using AIC_c as described in Appendix S3. Based on the best fitting model, we compared 95% CI of parameter estimates to make inferences on differences in the dynamics between resistant and susceptible genotypes.

Finally, we investigated which set of transmission-related parameters best explained variation in vector transmission, that is, infection status of test plants, using piecewise structural equation modeling (SEM). We were primarily interested in the paths through which the *PdR1* resistance trait influences transmission and in the relative importance of different components of host selection by vectors on transmission. We constructed a series of (generalized) linear models based on our hypothesized causal linkages or paths among the explanatory variables leading to infection of test plants. We evaluated the importance of the paths with model selection and standardized coefficient estimates (Appendix S4).

In discussing results of statistical tests below, we prefer the language of clarity over the use of significance, following the arguments of Dushoff et al. (2019). Also note that we rely more on 95% CI and AIC values rather than *P*-values in making inferences (Nakagawa and Cuthill 2007, Dushoff et al. 2019); nonetheless, we report *P*-values as well due to their prominence in traditional statistical reporting.

All analyses were performed in R 3.5.3 (R Core Team 2019). We used the rms package 5.1-3 to perform partial odds ordinal logistic regression (Harrell 2019). To fit consumer movement models, we used the optimx package version 7.10 with the spectral gradient optimization algorithm (Nash and Varadhan 2011); to fit nonlinear transmission models, we used the bbmle package 1.0.20 (Bolker and R Core Team 2017). For structural equation modeling, we used the piecewiseSEM package 2.0.2 (Lefcheck 2016). All data have been archived (Zeilinger et al. 2021), and all R code for analyses have been archived at https://doi.org/10.5281/zenodo.4547775.

Results

Pierce's disease severity and X. fastidiosa *population dynamics*

In both years, inoculated susceptible grapevines exhibited Pierce's disease symptoms earlier and more severely than inoculated resistant grapevines (Fig. 2A, B). In 2016, we could not detect a significant effect of week or difference in genotypes (weeks post-inoculation estimate ± standard error [SE] = 2.692 ± 6.885, Wald $Z = 0.39$, $P = 0.696$; genotype estimate ± SE = 1.677 ± 75.024, Wald $Z = 0.02$, $P = 0.982$; week-by-genotype interaction estimate ± SE = 1.137 ± 8.074, $Z = 0.14$, $P = 0.888$). In 2017, while we did not detect any clear difference in the genotype main effect, there was a clear trend of increasing symptom severity over time and a

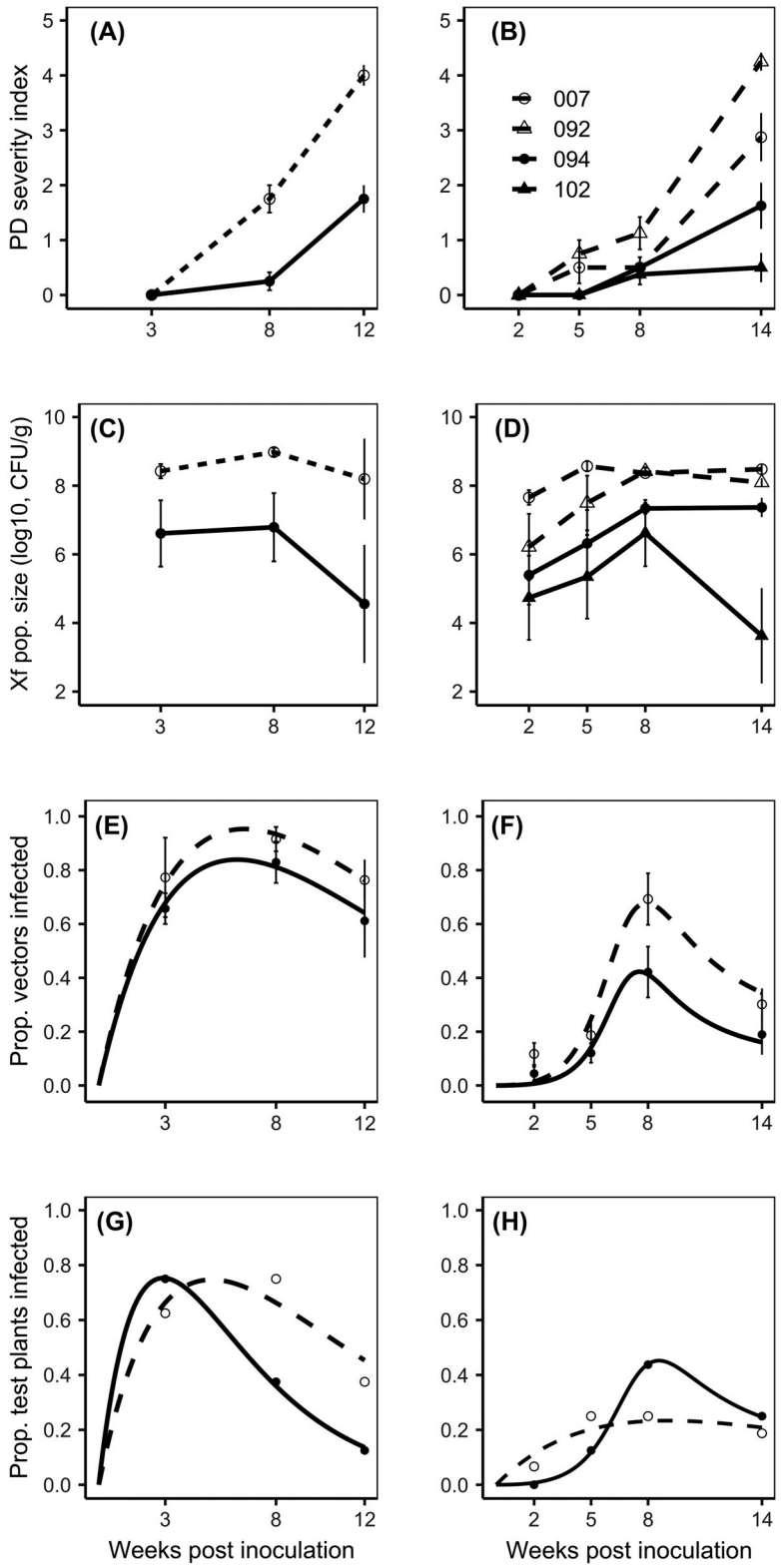

Fig. 2. Mean ± standard error (SE) Pierce's disease symptom severity index (A, B), mean ± SE population

(Fig. 2. *Continued*)

sizes of *Xylella fastidiosa* in inoculated source plants (C, D), vector acquisition (E, F), and transmission of *X. fastidiosa* to test plants (G, H). Panels in the left column show results from 2016 (A, C, E, G); panels in the right column are from 2017 (B, D, F, H). Susceptible genotypes are represented by open symbols and dashed lines; *PdR1*-resistant genotypes are in closed symbols and solid lines. Lines in panels of vector acquisition and transmission (E–H) represent predictions from best nonlinear model based on Akaike's information criterion corrected for small sample size. The legend in (B) indicates the different genotypes used in 2017; points in (F, H) represent data combined over genotypes. Population sizes are expressed as colony-forming units (CFU) g^{-1} of fresh plant tissue and $\log_{10}$ transformed, though untransformed data were used for statistical analysis (see *Statistical analysis* section). Error bars represent ± SE.

clear interaction between week and genotype (week estimate ± SE = 0.706 ± 0.141, $Z = 5.00$, $P < 0.0001$; genotype estimate ± SE = 1.526 ± 1.873, $Z = 0.81$, $P = 0.415$; week-by-genotype interaction estimate ± SE = −0.463 ± 0.183, $Z = -2.53$, $P = 0.012$).

Both susceptible and resistant grapevines harbored substantial populations of *X. fastidiosa* throughout our experiments, though populations in resistant lines were consistently lower (Fig. 2C, D). In 2016, *X. fastidiosa* populations increased over time and were clearly greater in susceptible compared with resistant lines (week post-inoculation coefficient estimate [95% CI] = 0.321 [0.0914, 0.660], $t = 2.32$, $P = 0.025$; genotype estimate [95% CI] = 2.96 [0.340, 6.94], $t = 1.85$, $P = 0.072$). There was no clear interaction between weeks and genotype (estimate [95% CI] = −0.122 [−0.471, 0.128], $t = -0.835$, $P = 0.41$). In 2017, we found no clear increase in *X. fastidiosa* populations over time (weeks post-inoculation estimate [95% CI] = 0.0382 [−0.0262, 0.102], $t = 1.18$, $P = 0.24$). However, *X. fastidiosa* populations were substantially lower in the resistant genotypes than the susceptible genotype 007, which was used as a baseline for statistical comparison (resistant line 094 estimate [95% CI] = −1.91 [−3.71, −0.448], $t = -2.34$, $P = 0.021$; resistant line 102 estimate [95% CI] = −1.86 [−3.83, −0.293], $t = -2.11$, $P = 0.037$).

Host selection by vectors

In the 2016 experiment, the fixed model had the lowest ΔAIC_c for each genotype at 3 and 8 weeks post-inoculation as well as for trials with the resistant genotype at 12 weeks (Table 1). The fixed model was clearly the best for most of these trials (i.e., the ΔAIC_c values for the remaining models were >2), indicating that the *G. atropunctata* showed no preference between inoculated source plants and uninfected test plants. The sole exception was in trials with susceptible source plants at 12 weeks post-inoculation; here, the free leaving model fits the data best and clearly much better than the fixed model (Table 1), indicating that *G. atropunctata* showed a preference between the host plant choices. However, the 95% CI for the attraction and leaving rates were quite large (Fig. 3A), muddling any apparent differences.

In the 2017 experiment, patterns of preference were more complicated than in 2016 though the differences were also clearer (Table 1). At two weeks post-inoculation, the fixed model fits the data best overall. While the fixed model performed slightly worse than the free attraction and free leaving models for trials with genotype 092 susceptible source plants, the difference was negligible ($\Delta AIC_c < 1$).

At five weeks post-inoculation, the fixed model fits the data best for trials with genotypes 007 susceptible and 102 resistant; once again, the fixed model performed worse than the free attraction and free leaving models for genotype 092 susceptible but now the difference in performance was stronger ($\Delta AIC_c > 2$). Interestingly, for genotype 094 resistant, the fixed model performed much worse than all other models, with the free attraction model performing best (Table 1).

At eight weeks post-inoculation, the fixed model performed the best for both susceptible genotypes (007 and 092), whereas the free attraction model performed the best for both resistant genotypes (094 and 102; Table 1).

Table 1. Model selection tables for consumer movement model variants for each combination of week post-inoculation and genotype for the 2016 and 2017 transmission experiments.

Genotype by no. weeks post-inoculation	Model	AIC_c	ΔAIC_c	df
2016				
3 weeks				
Susceptible	Fixed	171.90	0	2
	Free leaving	176.47	4.57	3
	Free attraction	177.46	5.56	3
	Free choice	183.90	12.00	4
Resistant	Fixed	146.47	0	2
	Free leaving	152.01	5.54	3
	Free attraction	152.06	5.59	3
	Free choice	161.34	14.87	4
8 weeks				
Susceptible	Fixed	153.10	0	2
	Free leaving	158.61	5.51	3
	Free attraction	158.63	5.53	3
	Free choice	167.94	14.85	4
Resistant	Fixed	132.33	0	2
	Free leaving	137.22	4.89	3
	Free attraction	137.84	5.51	3
	Free choice	146.54	14.22	4
12 weeks				
Susceptible	Free leaving	158.39	0	3
	Free attraction	159.81	1.41	3
	Fixed	161.58	3.19	2
	Free choice	167.52	9.13	4
Resistant	Fixed	145.83	0	2
	Free attraction	148.75	2.92	3
	Free leaving	149.49	3.66	3
	Free choice	157.95	12.12	4
2017				
2 weeks				
007 Susceptible	Fixed	138.32	0	2
	Free leaving	143.53	5.21	3
	Free attraction	143.59	5.27	3
	Free choice	152.85	14.52	4
092 Susceptible	Free leaving	127.55	0	3
	Free attraction	127.83	0.28	3
	Fixed	128.43	0.88	2
	Free choice	136.37	8.82	4
094 Resistant	Fixed	137.87	0	2
	Free leaving	140.58	2.71	3
	Free attraction	140.60	2.72	3
	Free choice	149.65	11.78	4
102 Resistant	Fixed	134.87	0	2
	Free attraction	137.72	2.84	3
	Free leaving	138.44	3.57	3
	Free choice	147.05	12.17	4
5 weeks				
007 Susceptible	Fixed	141.89	0	2
	Free leaving	147.42	5.53	3
	Free attraction	147.47	5.57	3
	Free choice	156.70	14.80	4

(Table 1. Continued.)

Genotype by no. weeks post-inoculation	Model	AIC_c	ΔAIC_c	df
092 Susceptible	Free leaving	151.41	0	3
	Free attraction	154.30	2.90	3
	Fixed	155.02	3.62	2
	Free choice	160.37	8.96	4
094 Resistant	Free attraction	147.77	0	3
	Free leaving	149.23	1.46	3
	Free choice	156.34	8.57	4
	Fixed	169.29	21.51	2
102 Resistant	Fixed	161.38	0	2
	Free leaving	166.20	4.82	3
	Free attraction	166.67	5.30	3
	Free choice	174.68	13.3	4
8 weeks				
007 Susceptible	Fixed	129.41	0	2
	Free attraction	134.83	5.41	3
	Free leaving	135.01	5.60	3
	Free choice	144.11	14.7	4
092 Susceptible	Fixed	148.86	0	2
	Free attraction	151.50	2.64	3
	Free leaving	152.37	3.50	3
	Free choice	160.73	11.87	4
094 Resistant	Free attraction	170.06	0	3
	Free choice	178.47	8.41	4
	Free leaving	181.82	11.76	3
	Fixed	184.15	14.09	2
102 Resistant	Free attraction	135.96	0	3
	Free leaving	139.16	3.20	3
	Free choice	144.13	8.17	4
	Fixed	154.92	18.96	2
14 weeks				
007 Susceptible	Free attraction	128.15	0	3
	Free choice	137.46	9.31	4
	Free leaving	138.13	9.98	3
	Fixed	144.90	16.74	2
092 Susceptible	Free attraction	149.60	0	3
	Free leaving	152.69	3.09	3
	Free choice	156.75	7.15	4
	Fixed	190.25	40.65	2
094 Resistant	Free attraction	136.05	0	3
	Free leaving	137.68	1.63	3
	Fixed	140.87	4.82	2
	Free choice	145.33	9.27	4
102 Resistant	Free attraction	149.07	0	3
	Free leaving	149.25	0.18	3
	Free choice	157.51	8.44	4
	Fixed	168.32	19.25	2

Notes: Separate sub-tables are sorted by ΔAIC_c in ascending order. Abbreviations are as follows: R, resistant genotype; S, susceptible genotype; AIC_c, Akaike's information criterion corrected for small sample size; ΔAIC_c, difference between lowest AIC_c score and a given score; df, degrees of freedom, that is, number of model parameters.

At 14 weeks post-inoculation, the free attraction model performed the best for all genotypes. At the same time, the performance of the free attraction model as the best model was much clearer for the susceptible genotypes than for the resistant genotypes (Table 1).

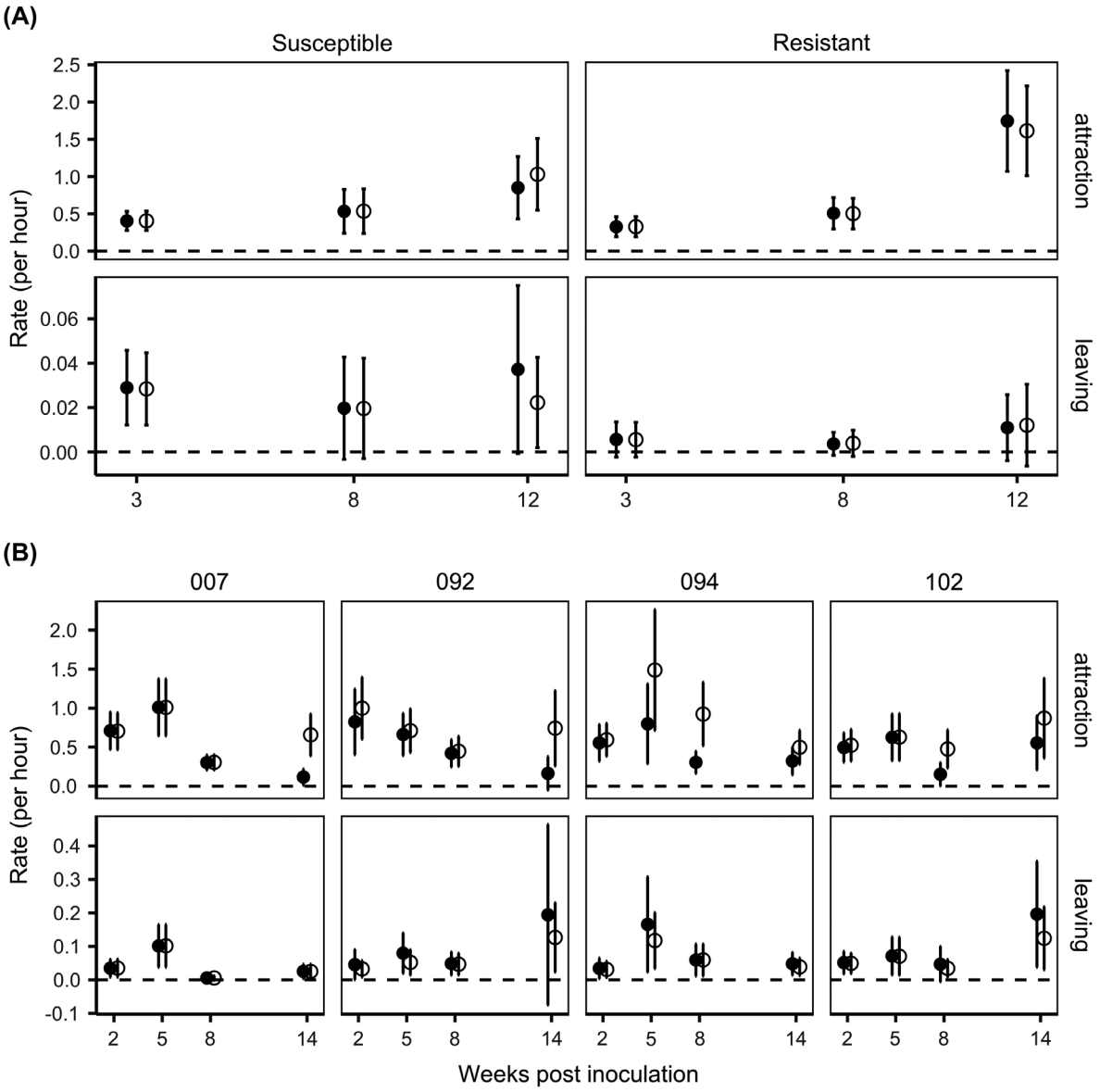

Fig. 3. Results of *Graphocephala atropunctata* vector feeding preference for inoculated source grapevines (closed circles) and uninfected test plants (open circles) over weeks post-inoculation for experiments in (A) 2016 and (B) 2017. Top sub-panels show attraction rate estimates; bottom sub-panels show leaving rate estimates. Genotypes are shown at the top of each set of sub-panels. In 2017, genotypes 007 and 092 are susceptible lines; genotypes 094 and 102 are resistant lines. Maximum-likelihood estimates (points) and variances were model-averaged using Akaike's information criterion corrected for small sample size weights. Error bars represent 95% confidence intervals.

Comparing across years, overall movement rates were not clearly greater in one year over the other. Attraction rates were on average greater in 2016 (2016 mean attraction rate = 0.713 h^{-1}; 2017 mean attraction rate = 0.633 h^{-1}) while leaving rates were much greater in 2017 (2016 mean leaving rate = 0.0165 h^{-1}; 2017 mean leaving rate = 0.0673 h^{-1}).

Vector acquisition and transmission

For the vector acquisition analysis, unimodal models performed the best in both years. In 2016, the Ricker model fits the data best for both the susceptible and resistant genotypes (Table 2). While vector acquisition was consistently greater from susceptible grapevines, the parameter estimates did not differ based on 95% CI (Table 3, Fig. 1E). In 2017, the Holling type IV model fits the acquisition data best for both susceptible and resistant genotypes (Table 2). In this case, 95% CI did not overlap for two out of three of the parameters estimated; CI could not be estimated for the third parameter (Table 3). Acquisition rates were clearly greater from susceptible plants than resistant genotypes (Fig. 1F).

Vector transmission (i.e., the proportion of test plants infected with *X. fastidiosa*) in 2016 was overall slightly greater from susceptible plants (total proportion of test plants infected = 0.538) than from resistant plants (0.435). AIC_c values among models were relatively similar (Table 3); three models fit the data from the resistant genotype equally well: the logistic growth, Ricker, and linear models. However, from the parameter estimates, the fits of the logistic growth and linear models resulted in very high transmission rates at the *y*-intercept, that is, immediately at the time of inoculation, which would be biologically implausible. We selected the Ricker model as it predicts a transmission rate near zero at the *y*-intercept. Likewise, the logistic growth, Ricker, and linear models fit the data from the susceptible genotype in 2016 similarly, though the Ricker model was slightly better in this case and again more biologically plausible. The initial increase in transmission rate was similar between the two genotypes—as seen in similar estimates for parameter a (Table 3)—but the timing of peak transmission differed slightly—with 95% CI of the parameter b only slightly overlapping (Table 3); transmission from resistant genotypes peaked much earlier than from susceptible genotypes (Fig. 2G). Both curves suggest a similar probability of transmission at the peak (~0.75).

In 2017, transmission was overall slightly greater from resistant plants (total proportion of test plants infected = 0.234) than from

Table 2. Results of model selection among nonlinear models for vector acquisition and transmission in 2016 and 2017.

Genotype by year and response	Model	AIC_c	ΔAIC_c	df
Acquisition				
2016				
Resistant	Ricker	15.49	0	2
	Holling type IV	17.71	2.21	3
	Logistic growth	18.87	3.38	2
	Linear	19.09	3.59	2
	Holling type II	20.07	4.58	2
Susceptible	Ricker	15.41	0	2
	Holling type IV	17.63	2.21	3
	Holling type II	22.95	7.54	2
	Logistic growth	23.70	8.29	2
	Linear	23.71	8.29	2
2017				
Resistant	Holling type IV	32.13	0	3
	Ricker	56.70	24.58	2
	Holling type II	62.26	30.13	2
	Linear	71.56	39.44	2
	Logistic growth	74.55	42.43	2
Susceptible	Holling type IV	59.73	0	3
	Ricker	85.56	25.83	2
	Holling type II	94.61	34.88	2
	Linear	109.31	49.58	2
	Logistic growth	112.50	52.77	2
Transmission				
2016				
Resistant	Logistic growth	13.14	0	2
	Ricker	13.15	0.01	2
	Linear	13.16	0.01	2
	Holling type II	14.21	1.07	2
	Holling type IV	18.74	5.59	3
Susceptible	Ricker	14.34	0	2
	Logistic growth	15.35	1.01	2
	Linear	15.4	1.06	2
	Holling type II	16	1.66	2
	Holling type IV	19.4	5.06	3
2017				
Resistant	Holling type IV	16.95	0	3
	Linear	19.87	2.92	2
	Ricker	19.88	2.93	2
	Holling type II	20.06	3.11	2
	Logistic growth	22.55	5.6	2
Susceptible	Ricker	16.37	0	2
	Holling type II	16.97	0.6	2
	Linear	17.93	1.56	2
	Logistic growth	18.05	1.68	2
	Holling type IV	18.7	2.33	3

Notes: Separate sub-tables are sorted by ΔAIC_c in ascending order. Abbreviations as in Table 1.

Table 3. Parameter estimates and 95% confidence intervals (CI) for best nonlinear model for vector acquisition and transmission in 2016 and 2017.

Genotype by year and response	Model	Parameter	Estimate [2.5%, 97.5% CI]
Acquisition			
2016			
Resistant	Ricker	a	0.366 [0.238, 0.508]
		b	0.16 [0.139, 0.2]
Susceptible	Ricker	a	0.394 [0.315, 0.466]
2017		b	0.152 [0.145, 0.175]
Resistant	Holling type IV	a	0.049 [0.04, 0.057]
		b	50.364 [ND, ND]
		c	−13.35 [−13.353, −13.348]
Susceptible	Holling type IV	a	0.111 [0.099, 0.123]
		b	52.354 [ND, ND]
		c	−13.234 [−13.234, −13.234]
Transmission			
2016			
Resistant	Ricker	a	0.704 [0.291, 1.032]
		b	0.344 [0.27, 0.524]
Susceptible	Ricker	a	0.399 [0.172, 0.575]
		b	0.196 [0.151, 0.294]
2017			
Resistant	Holling type IV	a	0.069 [ND, ND]
		b	62.221 [ND, ND]
		c	−14.517 [ND, ND]
Susceptible	Ricker	a	0.07 [0.021, 0.189]
		b	0.111 [0.011, 0.238]

Note: ND, not determined; CI could not be calculated by inverting the Hessian matrix.

susceptible plants (0.203), though transmission from resistant plants was much more dynamic (Fig. 2H). The Ricker model fit the data from susceptible genotypes the best, though the Holling type II provided a similar fit (Table 3). In contrast, the Holling type IV model fits the data from resistant genotypes the best. That different models were selected indicates qualitatively different dynamics for transmission from the resistant and susceptible genotypes. Comparing transmission between years, the peak transmission in 2017 was later and lower than in 2016 for both genotypes (Fig. 2G, H). Model selection results and parameter estimates are discussed in more detail in Appendix S3.

Finally, we synthesized all our data from 2017 on the vector transmission process into a structural equation model (as pointed out in the *Materials and Methods*, we lacked sufficient sample size in 2016). The best structural equation model —based on AIC_c values—included all variables except Pierce's disease symptom severity index, although the model excluding leaving rates as well was nearly indistinguishable (Appendix S4: Table S1). The absolute fit of the best model—based on Fisher's *C* statistic—was appropriate given the data (Fisher's $C = 32.83$, $df = 32$, $P = 0.43$). Variation in transmission rate was best explained by variation in the vector acquisition rate, which in turn was best explained by *X. fastidiosa* population size in source plants (Fig. 4). Population size itself was best explained by the presence or absence of the *PdR1* resistance trait. All components of vector preference remained in the best model, with leaving rates more strongly influencing vector transmission than attraction rates.

Discussion

Vector-borne plant pathogens threaten agricultural sustainability, and development of disease-resistant cultivars for agricultural species provides an important management alternative to

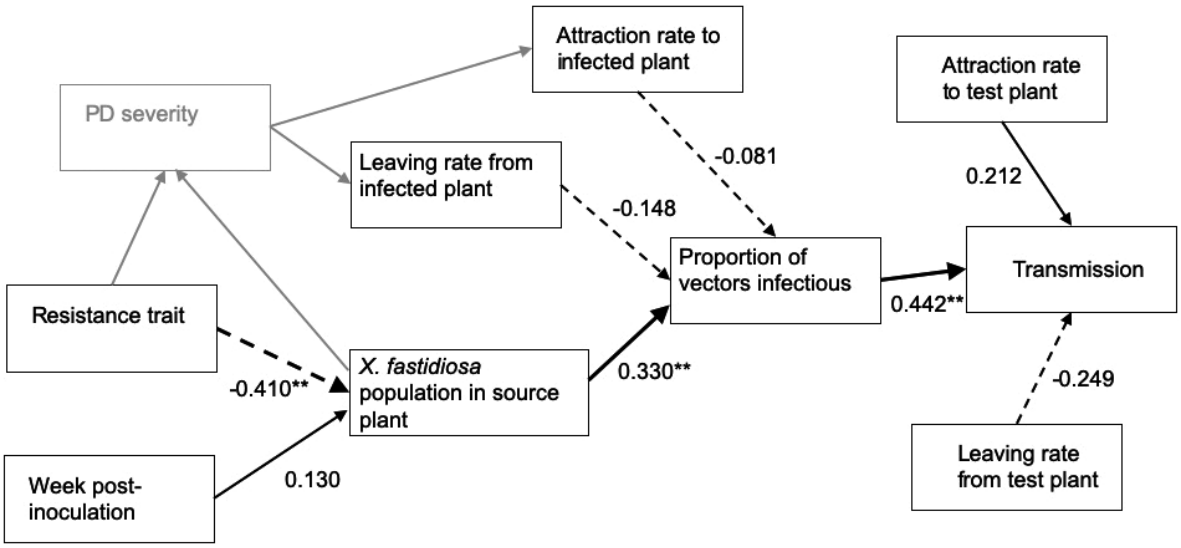

Fig. 4. Standardized coefficient estimates from piecewise structural equation modeling on the relationship between explanatory variables and transmission (i.e., infection status of test plant) in 2017. The resistance trait variable indicates a binomial variable where trials with *PdR1*-resistant sources plants are coded as 1, indicating presence of resistance, and trials with susceptible source plants are coded as 0. Attraction rates and leaving rates are estimated for each trial using the consumer movement model described in the full text. Variables and paths in gray were dropped from the best model. Solid paths represent positive relationships; dashed paths represent negative relationships; statistically significant ($P < 0.001$) relationships are indicated by ** and larger arrows.

insecticides. At the same time, partially resistant or tolerant cultivars could—according to theoretical predictions—increase the risk of disease spread and spillover to more susceptible hosts (Zeilinger and Daugherty 2014, Sisterson and Stenger 2018). In the context of such epidemic risks, we tested the impacts of the *PdR1* resistance trait in grapevines on transmission of the bacterial pathogen *X. fastidiosa* by the insect vector *G. atropunctata*. Our results confirmed previous findings that *PdR1* grapevines exhibit reduced disease severity and harbor lower *X. fastidiosa* population sizes than susceptible genotypes (Krivanek et al. 2005, Krivanek and Walker 2005, Fritschi et al. 2007).

Host defense against pathogens—whether resistance or tolerance—is predicted to have widely divergent epidemic consequences depending on how host-selection behavior of vectors is influenced by host infection: Resistance should generally reduce pathogen spread whereas tolerance traits combined with avoidance of diseased hosts should enhance pathogen spread (Zeilinger and Daugherty 2014). In our 2017 experiments, we found evidence of avoidance by *G. atropunctata* of infected host plants only when disease symptoms were severe—at late stages of infection (14 weeks post-inoculation) in susceptible plants. Avoidance of severely diseased susceptible plants of the 007 genotype was clear from both model selection by AIC_c and model-averaged 95% CI around attraction rate estimates (Table 1, Fig. 3B). For the 092 susceptible genotype, the 95% CI overlapped while model selection results strongly suggested preference, making evidence of avoidance less clear. In 2016, the results of our model selection process suggest avoidance of diseased susceptible plants; however, the wide CI make inferences still more difficult (Table 1, Fig. 3A). Previous work also found that cicadellid vectors of *X. fastidiosa*—including our study species—consistently avoid symptomatic infected host plants (Marucci et al. 2005, Daugherty et al. 2011, 2017, De Miranda et al. 2013). Furthermore, when avoidance was clear in our study, it was realized through differences in attraction rates, corroborating previous research that cicadellid vectors orient toward

host plants using visual cues (Rashed et al. 2011). Unexpectedly, we also found evidence of preference toward infected resistant plants at intermediate stages of infection (8 weeks post-inoculation). As no previous studies report similar findings, it remains unclear what may be driving this preference.

Vector acquisition rates—measured as the proportion of vectors that became infectious—were clearly nonlinear over the course of disease progression, following unimodal dynamics. Furthermore, vector transmission rates—measured as the proportion of healthy test plants that became infected—exhibited similar unimodal dynamics. Both acquisition and transmission rates were consistently greater in 2016 than 2017, likely because we ran trials for twice as long in 2016.

Contrary to predictions from theory (Fig. 1; Daugherty et al. 2017), we saw similar dynamics in transmission from both susceptible and *PdR1*-resistant grapevines. For transmission from susceptible vines, our results broadly conform to our predictions of unimodal transmission dynamics: transmission increases early after inoculation coinciding with increasing population size of *X. fastidiosa*, then declines with increasing symptom severity and concomitant avoidance by vectors. For trials with resistant vines however, because of a lack of symptom development, the unimodal dynamics appear to be more strongly tied to *X. fastidiosa* population size; specifically, transmission appears to decline in later stages of infection because of substantial declines in *X. fastidiosa* population size in two of the three resistant genotypes that we tested. Thus, the available evidence suggests distinct mechanisms underlying these similar patterns. In 2017, different models fit our transmission data from the different genotypes—with the Ricker model for susceptible plants and the Holling type IV model for resistant plants—suggesting qualitatively different dynamics and corroborating our interpretation. Previous studies of *PdR1* resistance report similar findings. Fritschi et al. (2007) found unimodal *X. fastidiosa* population dynamics in a range of resistant *Vitis* spp., including an accession of *V. arizonica* related to the parental background of the grapevines used in our study; they suggested that such unimodal dynamics could be caused by induced resistance. This was echoed by Riaz et al. (2018) who hypothesized that *PdR1* could confer a broad set of constitutive and inducible defensive traits against pathogens.

Beginning with the seminal work of McElhany et al. (1995), theory on the epidemiology of vector-borne plant diseases has increasingly emphasized the importance infection-induced host-selection behavior by vectors. Meanwhile, experimental work has succeeded in documenting a wide variety of infection-induced host-selection behaviors in vectors (reviewed in Eigenbrode et al. 2018). However, the theory remains disconnected from the empirical work because the vast majority of the empirical studies fail to test whether vector preference has any effect on transmission of pathogens to new hosts. While a handful of studies have suggested a positive or neutral association between infection-induced vector preference and transmission (Jennersten 1988, Daugherty et al. 2011, 2017, Del Cid et al. 2018), no study has yet to quantitatively test for such an empirical relationship.

We were able to efficiently determine the relationships between vector preference and transmission by decomposing preference into attraction and leaving rates using the consumer movement model of Gray et al. (2020) and incorporating these estimates into a structural equation model (SEM) of the vector transmission process. We found that while host selection by vectors is important for determining transmission, these components were far less important than vector acquisition rate, which was mostly driven by population size of *X. fastidiosa* in the infected plants, which in turn was determined by the presence or absence of the *PdR1* resistance trait. Furthermore, leaving rates from infected as well as healthy plants tended to be stronger predictors of transmission than their respective attraction rates.

Our results provide only partial support to the existing theory. Attraction rates and leaving rates of *G. atropunctata* undoubtedly influenced transmission dynamics of *X. fastidiosa* to some degree; we observed reduced attraction rates toward susceptible plants at later stages of disease that clearly coincided with—and provide the only plausible explanation for—a decline in transmission. However, our SEM analysis contradicts the results of Sisterson (2008) and Shaw et al. (2017) who predict a much stronger influence of

attraction rates over leaving rates in driving pathogen spread. At the same time, the apparent importance of the leaving rate from healthy hosts supports the theory of Madden et al. (2000), who predicted that transmission of persistent pathogens was sensitive to tenure time on healthy hosts (where tenure time = leaving rate^{-1}).

Much of the theory on vector-borne plant diseases was developed in the context of the host manipulation hypothesis and as such assumes that infection-induced vector preference is adaptive for the pathogen (Mauck et al. 2018). In our system, vector avoidance of diseased plants is unlikely to be adaptive for *X. fastidiosa*, an important distinction from the systems motivating much of the theory. What's more, the effect size of preference in our study was relatively modest and inconsistent. Nonetheless, the theory should still apply to all cases of infection-induced vector preference; the relationship between preference and spread has largely been modeled using monotonic relationships, implying that the relationship should remain regardless of the direction and magnitude of preference (McElhany et al. 1995, Sisterson 2008, Zeilinger and Daugherty 2014, Shaw et al. 2017). For example, Shaw et al. (2017) predict that avoidance of infected hosts—such as what we find in our system—should broadly reduce spread (δ and ε in their model). However, we failed to find such a clear relationship. At the same time, the over-riding importance of vector acquisition in our results provides some indirect support for the prediction of Shaw et al (2017) that abundance of infectious vectors is more influential than vector behavior.

What could lead to a disconnect between host-selection behavior of vectors and pathogen transmission? If a vector prefers feeding on an infected plant, why would this not inevitably lead to greater transmission or, inversely, why would avoidance not inevitably lead to reduced transmission? One possibility is that herbivorous insects suffer from well-documented neurological limitations in processing sensory inputs, leading them to not always make the apparently best host selections (Bernays 2001). These neural limitations are hypothesized to be greater for generalist herbivores. Thus, with more complex environmental stimuli, we might expect the relationship between infection-induced vector preference and transmission to break down further, for example, when going from greenhouse experiments to field conditions. Such hypotheses could be incorporated into the theory with stochastic models, though only Donnelly et al. (2019) have so far considered stochasticity in their modeling. Clearly, further empirical work that test the relationship between vector preference (or manipulation) and transmission are needed to properly assess the generality of current theory across plant disease systems.

While the use of resistant cultivars is a key component to integrated disease management in agriculture, the risks of disease spillover or epidemics from such cultivars should be assessed prior to release. Generally, the greatest risk of transmission of vector-borne plant pathogens should occur within a window of disease progression when both pathogen burden and attractiveness for the vector are high, all else being equal (De Moraes et al. 2014). These two processes are clearly dynamic and may be governed by different drivers. For disease systems where vectors avoid symptomatic hosts, such as *X. fastidiosa*-associated diseases, the post-latent incubation period—in which hosts are infectious but asymptomatic—should be the period of greatest vector acquisition and thus most critical for pathogen spread (Daugherty et al. 2017, Kyrkou et al. 2018, Bragard et al. 2019).

Our results indicate that *PdR1* hybrid grapevines can produce transmission rates greater than those from susceptible vines, potentially posing a risk of enhanced spread of *X. fastidiosa*. However, our results also suggest that these higher transmission rates are transient, followed by transmission rates similar to or lower than those from susceptible plants. Importantly, our structural equation modeling indicates that the *PdR1* trait influences vector transmission much more strongly through *X. fastidiosa* population size than through the progression of disease symptoms. This suggests that the *PdR1* trait operates more like resistance than tolerance. Further work is needed to examine additional environmental factors that could contribute to shortening or broadening the incubation period, such as temperature and water stress (Daugherty et al. 2017, Del Cid et al. 2018). Validating the dynamics explored here under field conditions will also be critical. We might expect transmission dynamics in the field to proceed over a longer time scale with larger

more established vines—potentially over multiple seasons—and operate with greater stochasticity due to greater environmental variation.

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Supporting Information

Additional Supporting Information may be found online at: http://onlinelibrary.wiley.com/doi/10.1002/ecs2.3505/full