Title

The epidemiology of novel *PdR1* resistant grapevines: epidemic and vector movement models to support integrated disease management

Authors

Rodrigo P. P. Almeida
Dept. Environmental Science, Policy, and Management
UC Berkeley
Phone: (510) 642-1603
Email: rodrigoalmeida@berkeley.edu
Principal Investigator

M. Andrew Walker
Dept. Viticulture and Enology
UC Davis
Email: awalker@ucdavis.edu
Cooperator

Matt Daugherty
Dept. Entomology
UC Riverside
Email: mattd@ucr.edu
Cooperator

Perry de Valpine
Dept. Environmental Science, Policy, and Management
UC Berkeley
Email: pdevalpine@berkeley.edu
Cooperator

Adam Kleczkowski
Dept. Computing Science and Mathematics
University of Stirling
Stirling, Scotland
Email: ak@cs.stir.ac.uk
Cooperator

Reporting Period

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Abstract

Resistant cultivars of agricultural crops are integral to sustainable integrated disease management strategies. Our previous work indicated that grapevines that express the *PdR1* gene exhibit resistance against *Xylella fastidiosa*, and are likely to slow the spread of *X. fastidiosa* among vineyards. In the current project, we are testing the generality of our previous results, by testing multiple *PdR1* resistant and susceptible genotypes into our vector transmission experiments and integrating greater biological detail into our epidemic modeling work. While *PdR1* resistant grapevines provide promising resistance, it remains unclear how growers may incorporate these hybrid plants into their production. Growers may be able to benefit from *PdR1* resistant cultivars without planting all of their acreage to them. We will explore the implications for *X. fastidiosa* spread and Pierce's Disease severity from planting adjacent blocks of *PdR1* resistant and susceptible grapevines through bio-economic modeling. Finally, our modeling efforts rely on assumptions on insect vector dispersal within and among vineyards; yet our knowledge of sharpshooter dispersal has been limited by the difficulty of experimentally measuring dispersal. We will use large spatio-temporal data sets of vector abundance—for both *Graphocephala atropunctata* and *Homalodisca vitripennis*—and hierarchical statistical models to estimate dispersal directly from field data. Taken together, our project will provide clearer recommendations for disease management strategies using *PdR1* and related resistant grapevines.

Introduction

Resistance against pathogens in agricultural crops is one of the more successful strategies to effectively manage agricultural diseases (Mundt 2002). This includes vector-borne pathogens. Though insecticide suppression of vectors is a common practice, previous research has called into question the efficacy of insecticides and highlighted the risks of evolved resistance against them (Perring et al. 2001; Erlanger et al. 2008).

However, while plant resistance traits are often effective at suppressing pathogen spread, this is certainly not the case with tolerance traits. Where resistance traits alleviate disease symptoms by reducing pathogen burden, tolerance traits alleviate symptoms with negligible effects on pathogen burden (Roy and Kirchner 2000). For vector-borne pathogens, the influence of resistance traits on pathogen spread and disease prevalence can differ dramatically from tolerance traits (Zeilinger and Daugherty 2014; Cronin et al. 2014). Introducing resistance traits into a host population will generally reduce pathogen spread, whereas tolerance traits can have the opposite effect. Specifically, when vectors of a pathogen avoid feeding on diseased (i.e., symptomatic) hosts, introducing tolerant hosts will enhance pathogen spread (Zeilinger and Daugherty 2014). Because the primary sharpshooter vectors of *X. fastidiosa* in California—BGSS and GWSS—preferentially avoid feeding on PD-symptomatic plants (Daugherty et al. 2011), tolerance traits in grapevines could increase the risk of *X. fastidiosa* spread within and among vineyards.

On-going efforts to identify resistance to *X. fastidiosa* in native *Vitis* spp. has resulted in hybrid plants that express the *PdR1* locus (Walker and Tenscher 2016). These hybrid vines do not suffer from PD symptoms to the same extent of susceptible lines (Krivanek and Walker 2005; Krivanek et al. 2006). Furthermore, from our previous results, *PdR1* resistant grapevines appear to reduce insect vector transmission rates. As such, they are likely to reduce spread of *X. fastidiosa* within and among vineyards.

Objectives

The overall goal of this project is to assess the epidemiological consequences of managing Pierce's Disease (PD) with resistant grapevines expressing the *PdR1* locus (Walker and Tenscher 2016). Specifically, we ask, under what conditions and spatial arrangements will the use of *PdR1* vines reduce *X. fastidiosa* spread and maximize economic benefits to growers? The research consists of three objectives:

1. *Test the effects of* PdR1 *resistant plants on vector feeding preference and transmission of* X. fastidiosa
2. *Model the optimal mixture of* PdR1 *and susceptible grapevines to reduce* X. fastidiosa *spread and maximize economic return*
3. *Estimate dispersal of insect vectors from field population data*

Description of Activities

1. Test the effects of PdR1 *resistant plants on vector feeding preference and transmission of* X. fastidiosa

We are currently collecting data, using quantitative PCR, on *X. fastidiosa* populations acquired by *G. atropunctata* vectors in our vector feeding preference and transmission experiment. We are using new PCR primers developed in our lab and we have verified their improved efficiency over previously published primers. Our data on *X. fastidiosa* populations in

vectors will provide further insights into the differences in transmission biology between *PdR1* resistant grapevines and susceptible grapevines.

2. Model the optimal mixture of PdR1 *and susceptible grapevines to reduce* X. fastidiosa *spread and maximize economic return*

We have built a preliminary economic extension to our vector-SI epidemic model, described in our proposal. We have included Box 1 from our proposal, which describes the epidemic model that we previously developed.

We consider a scenario where two vineyards are grown adjacent to each other—one composed of a grape cultivar susceptible to Pierce's Disease, Patch 1, and another composed of *PdR1* resistant grapevines, Patch 2. Then we can define the state variables in Box 1 for each patch, such that S_j, E_j, $H_{C,j}$, and $H_{I,j}$, where $j = 1, 2$, to represent hosts in either Patch 1 or Patch 2, respectively.

For the preliminary economic model, we followed the framework of Macpherson et al. (2017) and assumed that yield is proportional to the density of healthy or asymptomatic hosts at harvest time ($t = \tau$). In our epidemic model (Box 1), hosts in the compartments S_j, E_j, and $H_{C,j}$ are healthy, whereas hosts in $H_{I,j}$ are diseased. Then total yield, Y, is defined as:

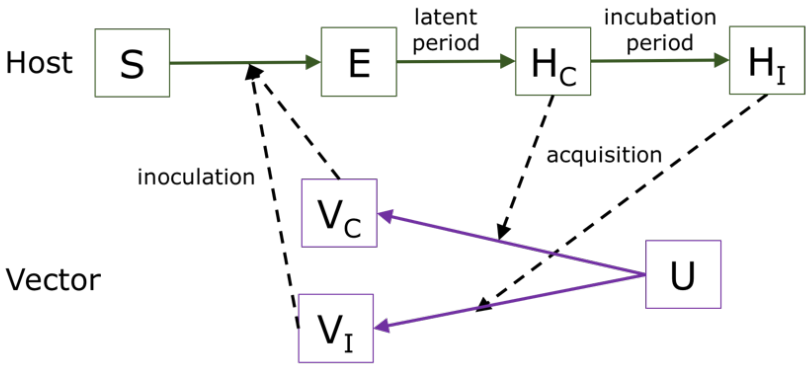
$$Y = c_1 M_1(t = \tau) + + c_2 M_2(t = \tau)$$

where $M_j = S_j + E_j + H_{C,j}$ and represents the total density of healthy hosts. The parameters c_j modulate the relative value of the two cultivars. For instance, if the resistant cultivar has a lower value per unit of harvested grapes, then we set $c_2 < c_1$. We set $\tau = 500$ to ensure that the epidemic model dynamics reach equilibrium. In addition, as a first approximation, we assume that all healthy hosts produce the same yield and all diseased hosts produce no yield.

We first explored the sensitivity of our bioeconomic model to variation in economic value of resistant grapevines and the area planted to resistant grapevines. We varied the value of the c_2 parameter between 0.01 and 10, while setting $c_1 = 1$ constant. For the epidemic model parameters, we used the mean values from our 2016 experimental results, as described in Box 1. Given our epidemic model parameters, the value of grapes from the resistant cultivar has a strong effect on total yield, indicating that yield from the susceptible patch is relatively poor and unimportant (Fig. 1). Unsurprisingly then, there is much higher yield when the resistant patch is larger.

In the initial simulation, we used epidemic parameter estimates from our experimental results. We also sought to explore the effects of uncertainty in the parameter estimates. Again, increasing the area planted to *PdR1* resistant grapevines increases the total expected yield (Fig. 2). At the same time, we see a large amount of uncertainty in the results as well, with a slight increasing in the 95% confidence intervals with increasing area planted to resistant grapevines.

Overall, our preliminary economic analysis suggests that planting *PdR1* resistant grapevines at high densities would be the most economically efficient strategy, under epidemiological conditions measured in our 2016 experiments. Our next steps will be simulate economic outcomes from our 2017 experimental results, which appear more robust than our 2016 experiment. We also explore more economically nuanced model structures and the effects of varying other parameters, such as dispersal rates of vectors between vineyard patches.



$$\frac{dS}{dt} = \eta(E + H_C + H_I) - \beta_c - \beta_I$$

$$\frac{dE}{dt} = \beta_C + \beta_I - \delta E - \eta E$$

$$\frac{dH_C}{dt} = \delta E - \gamma H_C - \eta H_C$$

$$\frac{dH_I}{dt} = \gamma H_C - \eta H_I$$

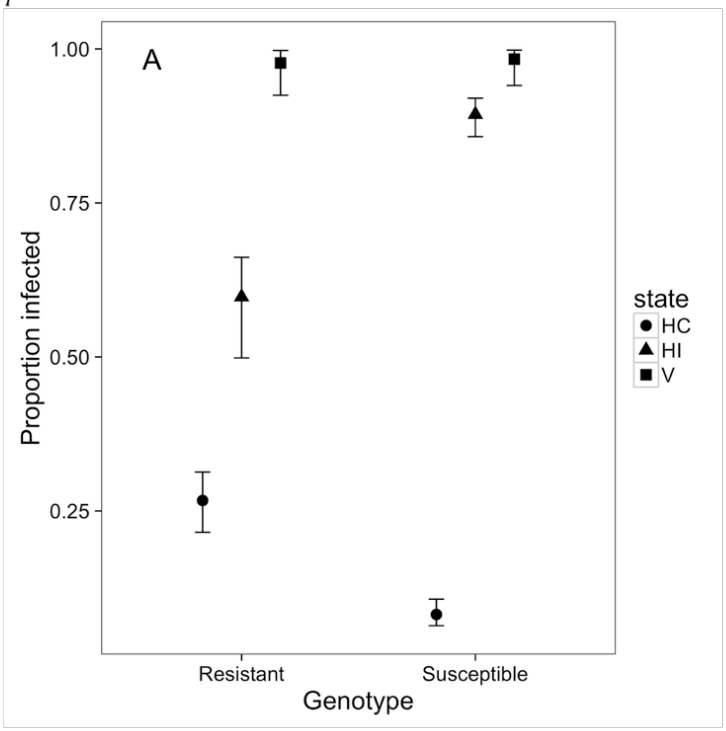
$$\frac{dU}{dt} = \lambda V_C + \lambda V_I - \alpha_C - \alpha_I$$

$$\frac{dV_C}{dt} = \alpha_C - \lambda V_C$$

$$\frac{dV_I}{dt} = \alpha_I - \lambda V_I$$

$$\beta_i = \frac{p_i(1 - e^{-b_i T})V_i S}{N}$$

$$\alpha_i = \frac{\mu_i(1 - e^{-a_i T})H_i U}{N}$$



Box 1. We modeled the spread of *X. fastidiosa* through simulated *PdR1* Resistant and Susceptible vineyards using a continuous-time SI-vector compartmental model. The model included compartments for non-infected hosts and vectors (S and U), exposed hosts (E), asymptomatic infected "Carrier" hosts (H_C), diseased infected hosts (H_I), and infectious vectors that acquired infection from either the H_C or H_I compartments (V_C and V_I). Inoculation and acquisition rates, β_i and α_i where i = C, I, were adapted from Madden et al. (2000). We used experimental data to estimate values for vector attraction rate (p_i), vector leaving rate (μ_i), inoculation probability (b_i), infectious period (δ^{-1}), incubation period (γ^{-1}), and host recover (η). Vector acquisition probability (a_i) was set as proportional to inoculation probability, pending data collection of *X. fastidiosa* populations in vectors. Vector recovery (λ) was set at 0.083. Time spent feeding (T) was calculated from Almeida and Backus (2004). $N = S + E + H_C + H_I$. Based on experimental results, estimates for i = C parameters were taken from 3-week trials while estimates for i = I parameters were taken from 12-week trials. We calculated standard errors for each experimentally-derived parameter and used Monte Carlo simulations (n = 5,000) to estimate mean and 95% confidence intervals for densities of infected hosts, H_C and H_I (filled circles and triangles, Panel A), and vectors, $V_C+V_I = V$ (filled squares), for *PdR1* Resistant and Susceptible vineyard scenarios. More detail and R code can be found at https://github.com/arzeilinger/pdr1_preference.

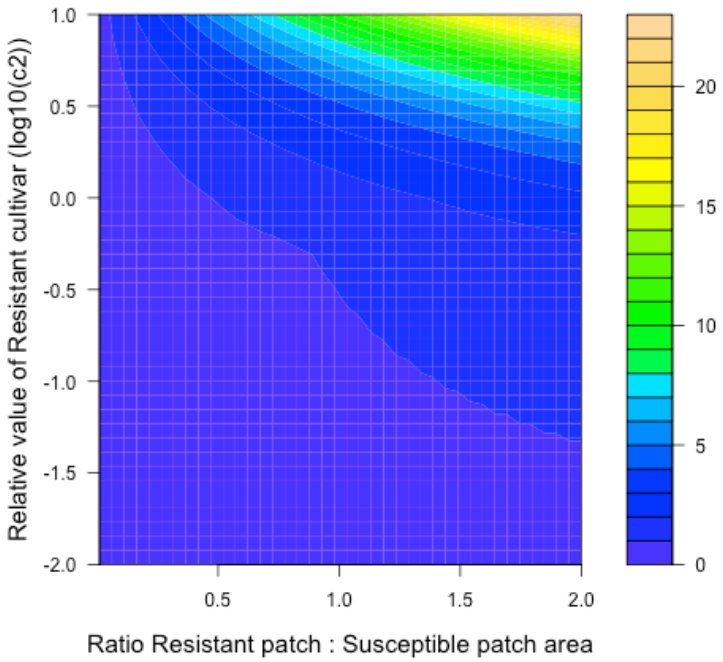


Figure 1. Contour plot showing the expected total yield from varying the area of the Resistant patch (relative to the susceptible patch area), and the relative value of grapes from the Resistant cultivar (c_2). Note that we varied c_2 from 0.01 to 10 and log10 transformed the y-axis. The colors indicate total yield, *Y*.

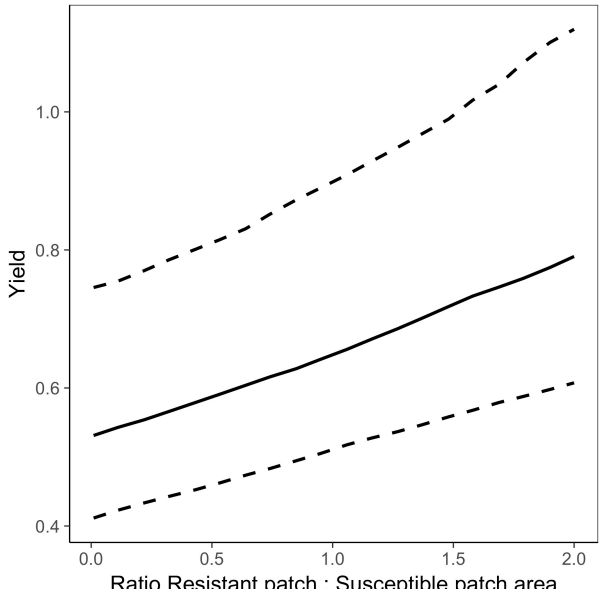


Figure 2. Median expected total yield (solid line) increases with increasing area planted to Resistant grapevines, but so too does uncertainty increase (95% confidence intervals, dashed lines). Confidence intervals were calculated from 600 Monte Carlo simulations of epidemic parameter values derived from our *PdR1* transmission experimental results. For these simulations, $c_1 = 1$, $c_2 = 0.1$.

3. Estimate dispersal of insect vectors from field population data

Work on Objective 3 will begin in fall 2018, informed by results in Objectives 1 and 2.

Publications

We are currently writing our results from the transmission experiments (Objective 1) for publication.

Research Relevance

Our preliminary analysis suggests that planting *PdR1* resistant cultivars is an economically efficient strategy for growers in areas with high Pierce's disease pressure. Moreover, improved return on investment are fairly robust against low value of *PdR1* grapes relative to susceptible cultivars. Under the conditions investigated so far, we see no trade-off between resistance and yield from *PdR1* grapevines. Further modeling efforts will aid in determining conditions under which trade-offs may exist.

Lay Summary

Sustainable management of Pierce's disease (PD) will rely on developing grape cultivars that are resistant to *Xylella fastidiosa*. Our research confirms previous findings that *PdR1* grapevines are partially resistant to *X. fastidiosa* colonization. While deployment of *PdR1* traits represent a promising management strategy, they will have to be deployed as part of an integrated management strategy, involving additional actions to slow the spread of *X. fastidiosa* within and among vineyards. We will integrate vector transmission and movement information to predict *X. fastidiosa* spread through *PdR1* and susceptible cultivars using mathematical models.

Status of funds

Funds are being used as originally proposed.

Status of intellectual property

IP not expected from this research.

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