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

The results reported here are from work conducted October 2017 to February 2018.

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*

This report focuses on Objective 1.

Description of Activities

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

In the summer of 2016, we investigated the interplay between vector feeding preference and transmission of *X. fastidiosa* from *PdR1* resistant and susceptible grapevine genotypes. We inoculated two *PdR1* resistant genotypes (labeled 094 and 102) and two susceptible genotypes (007 and 092) with *X. fastidiosa* STL strain. At 2, 5, 8, and 14 weeks post-inoculation, we introduced eight blue-green sharpshooters (BGSS, *Graphocephala atropunctata*) into a cage with one inoculated plant (from one of the four genotypes) and one *X. fastidiosa* -free test plant, of either susceptible genotype. We included eight replicates of each combination of week since inoculation and genotype, and each replicate were independent—using different plants and vectors in each trial. We recorded which plant the vectors were feeding on at regular intervals over a 4-day period, estimated *X. fastidiosa* populations in the source plants using culturing, assessed Pierce's disease symptoms in the source plants, and assessed transmission by culturing from *X. fastidiosa* -free test plants 3 months after the trials. We are in the process of estimating *X. fastidiosa* populations in vectors using qPCR.

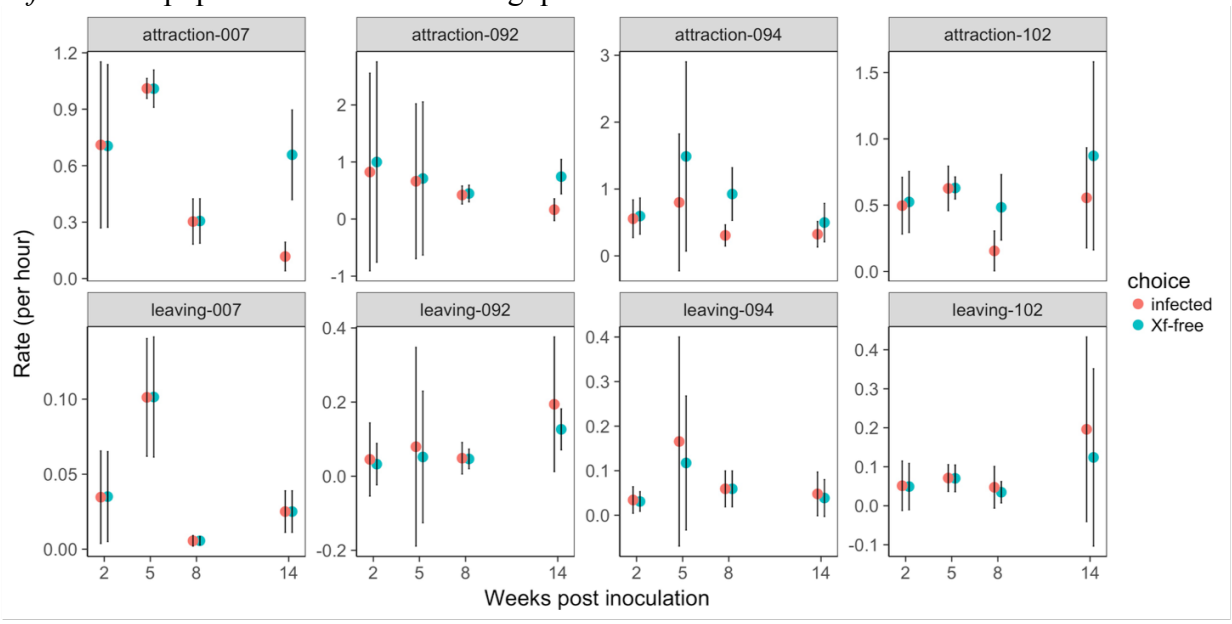

Figure 1. BGSS attraction rates (top four panels) varied significantly between infected and *Xylella*-free plant choices, particularly for susceptible genotypes (007 and 092) at 14 weeks post-inoculation. Leaving rates (bottom four panels) did not differ significantly. Error bars represent 95% confidence intervals.

We estimated attraction rates and leaving rates of the BGSS by fitting data collected on the number of insects on each plant to the Consumer Movement Model described in Zeilinger et al. (2014). We used general linear models with quasi-Poisson or Poisson link function to test for differences in genotypes and time since inoculation (2, 5, 8, and 14 weeks) in *X. fastidiosa* populations in source plants, *X. fastidiosa* populations in vectors, and in PD symptom severity. For PD symptom severity, we used the index described in Rashed et al. (2013). To test for differences in the percent of test plants infected with *X. fastidiosa*, we combined data for the resistant genotypes and the susceptible genotypes then fit these data to multiple linear and non-linear ecological models:

Linear $y = a + bx$

Ricker $y = axe^{-bx}$

Holling Type IV $y = \frac{ax^2}{b + cx + x^2}$

In these equations, y is the proportion of test plants infected, x is the weeks post-inoculation, and a, b, and c are model-specific parameters. The non-linear models were selected based on *a priori* hypotheses on the dynamics of infection in our experiment; see Bolker (2008) for further information.

BGSS showed significant preference for *X. fastidiosa*-free test plants compared to inoculated susceptible plants (007 and 092 genotypes) at 14 weeks post-inoculation. However, they showed no consistent preference in trials with inoculated resistant plants (094 and 102 genotypes) (Fig. 1).

Both of the susceptible genotypes exhibited deteriorating PD symptoms over time and were significantly worse than the resistant genotypes (Fig. 2A; week x genotype interaction: $F_{3, 102} = 9.83$, $P < 0.0001$). For the *X. fastidiosa* populations in the inoculated plants, the two susceptible genotypes had significantly greater populations than the resistant genotypes (Fig. 2B, $F_{3, 115} = 23.70$, $P < 0.0001$) and populations increased over time across genotypes ($F_{1, 115} = 4.92$, $P < 0.03$). The proportion of *X. fastidiosa*-free test plants exhibit clear non-linear dynamics over time post-inoculation (Fig. 2C). The best model for the resistant genotypes was the Holling Type IV whereas the best model for the susceptible genotypes was the Ricker model, suggesting significant differences in the transmission dynamics between the resistant and susceptible genotypes. These models suggest distinct biological processes underlying these dynamics, which we are exploring using additional modeling.

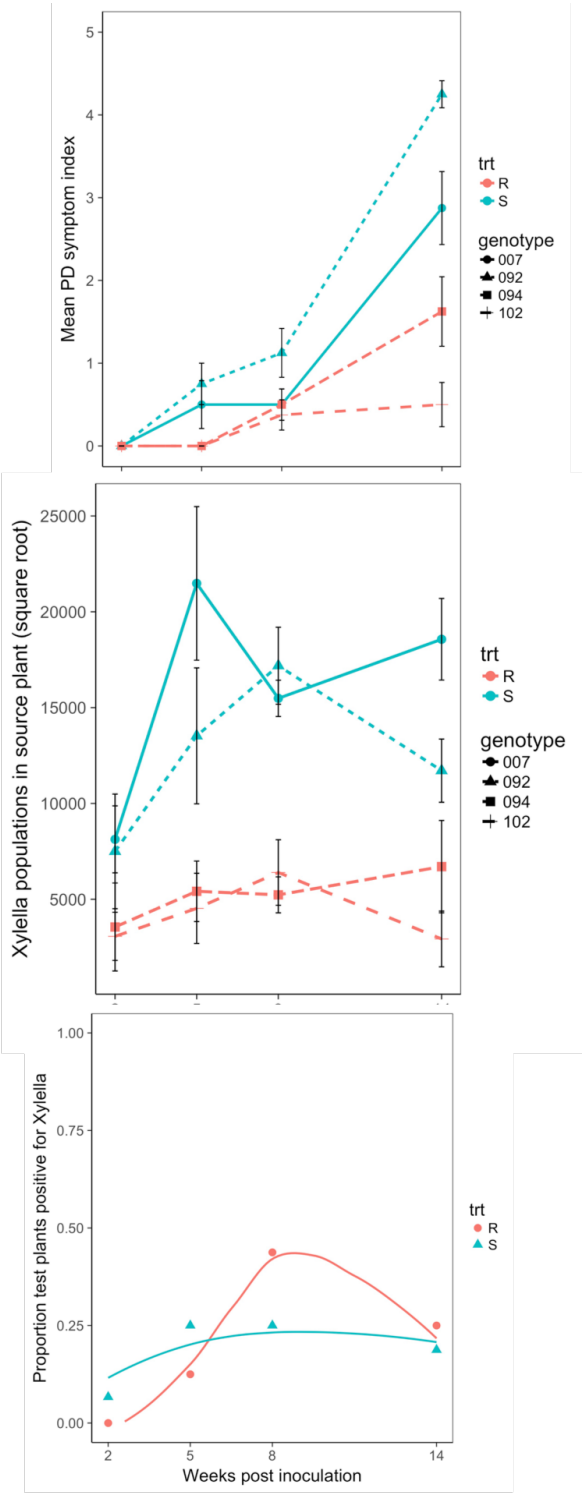

Figure 2. Mean PD symptom severity (A) and *X. fastidiosa* density in inoculated plants (B) were greater for susceptible genotypes than resistant genotypes. Proportion of test plants infected with *X. fastidiosa* were significantly different between resistant and susceptible genotypes, based on our model selection process. Error bars represent ± SE

Overall, our results confirm previous work in that *PdR1* resistant plants exhibit partial resistance to *X. fastidiosa*, resulting in reduced bacterial populations and reduced PD symptom severity. However, because *X. fastidiosa* is able to reach moderate population sizes in resistant plants, there is still significant vector transmission from these plants. Furthermore, because of reduced symptom severity, transmission dynamics are complex—transmission from resistant plants can be worse under some conditions (e.g., 8 weeks post-inoculation within our experiments).

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

Work on Objective 2 is beginning in winter/spring 2018, as described in proposal.

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 research confirms previous findings that *PdR1* grapevines are partially resistant to *X. fastidiosa* colonization. The partiality of resistance is a key finding—*X. fastidiosa* is able to grow in *PdR1* grapevines and insect vectors are able to transmit *X. fastidiosa* from them. 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. Our future work (as Objectives 2 and 3) will aid in identifying how growers can best manage Pierce's disease in *PdR1* vineyards.

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

Used as described in research proposal.

Status of intellectual property

None.

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