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A Bioeconomic Model of Plant Disease Management under Spatial-Dynamic Externalities:

Grapevine Leafroll Disease

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Abstract

Grapevine leafroll disease (GLRD) presently threatens the grape and wine industry worldwide. We develop a cellular automata model of GLRD diffusion and control in two ecologicallyconnected, independently-managed vineyards. One vineyard produces high-value wine grapes whereas the other produces low-value wine grapes. Disease management is modeled as a twoagent bargaining game. We show that under nooncooperative disease management, it is optimal for neither vineyard manager to control the disease. We consider the case of cooperative disease management and compute the benefits accruing from cooperation. We find it optimal for the manager of the high-value vineyard to pay the low-value vineyard manager to exit production. We determine the size of a Pareto-efficient side payment that can remedy the negative spatial externality emerging from noncooperative disease control.

Key Words: Bargaining games, Bioeconomic Models, Cellular Automata, Computational Methods, Externality, Disease Control, Grapevine Leafroll Disease, Side payment, Spatial-Dynamic Processes. *JEL Codes*: C63, C71, C72

Grapevine leafroll disease (GLRD) presently threatens grape harvests in the United States (Fuchs et al. 2009; Golino et al. 2008; Martin et al. 2005) and around the world (Cabaleiro et al. 2008; Charles et al. 2009; Martelli and Boudon-Padieu 2006). This viral disease hinders berry development and growth, delays budbreak, flowering, and berry ripening, and causes color change, sugar content reduction, and acidity increase in grape juice and wine (Goheen and Cook 1959; Martinson et al. 2008). These physiological changes cause a reduction in the yield (30-50%) and quality of grapes and wines. GLRD economic impact was recently estimated at \$25,000- \$40,000 per hectare over a 25 year-period in New York State vineyards if the disease is left uncontrolled (Atallah et al. 2012). The American Vineyard Foundation (2012) reports that GLRD is a serious threat to industry sustainability and its control is therefore a top research priority for growers (AVF 2012). GLRD is primarily transmitted via vegetative propagation. Once introduced through infected planting material, the disease is transmitted to healthy vines by several species of mealybugs (Hemiptera: Pseudococcidae) and soft-scale insects (Hemiptera: Coccidae) (Martelli and Boudon-Padieu 2006; Pietersen 2006; Tsai et al. 2010). Mealybugs can transmit GLRD within and across vineyards in at least three ways (Grasswitz and James 2008; Charles et al. 2009). Insect crawling on wires and fruiting canes can cause disease transmission to neighboring vines. Vineyard management activities can facilitate mealybug dispersal to farther neighboring vines within the same vineyard. Finally, disease spread between neighboring blocks or vineyards can take place through aerial dispersal of mealybugs (Le Maguet et al. 2013).

Vineyard managers are currently advised to avoid introducing GLRD into their vineyards by planting certified vines derived from virus-tested mother plants (Almeida et al. 2013; Fuchs 2007; Golino et al. 2002; Rayapati et al. 2008). However, when GLRD is already present,

disease management consists mainly of minimizing the source of inoculum by roguing symptomatic vines after harvest, especially the young ones, and replacing them with virus-tested vines (Maree et al. 2013; Rayapati et al. 2008; Walton et al. 2009). Vector management is recommended to reduce disease transmission (Skinsis et al. 2009). However, although insecticide sprays can reduce mealybug densities, they have not been effective at controlling GLRD spread, mainly because of the exceptionally low insect density needed for disease transmission (Cabaleiro and Segura 2006, 2007; Golino et al., 2002, 2008; Almeida et al. 2013).

Most GLRD research has focused on studying the pathogens with less work done on disease ecology and disease management (Almeida et al 2013). Nonetheless, recent research has evaluated nonspatial and spatial GLRD control strategies in a virtual, isolated vineyard (Atallah et al. 2012). Using computational experiments, the authors show that spatial strategies improve the expected net present value of a vineyard by around 20% over the strategy of no disease control. In these strategies, young symptomatic vines are rogued and replaced, and their nonsymptomatic immediate neighbors are virus-tested, then rogued and replaced if the test is positive. It is not immediately clear, however, whether these spatial disease control strategies remain profit-maximizing in the case where disease diffusion is characterized by an "edge effect". Charles et al. (2009) refer to an "edge effect" when the number of infected grapevines is decreasing with the distance from the edge of the vineyard. In the case of GLRD, the edge is an entry point for vectors carried by wind from a neighboring vineyard serving as a GLRD reservoir (Grasswitz and James 2008). Once mealybugs enter a vineyard through aerial dispersal, they are able to move between adjacent plants. In the presence of aerial dispersal from a neighboring vineyard, roguing and replanting strategies are likely to be less effective and additional control measures might be necessary (Rayapati et al. 2008). Some studies examining spatial dimensions

of GLRD report clusters of infected vines at the borders of a vineyard. These studies measure inter-vineyard GLRD spread as a gradient of diminishing disease incidence from the border towards the center of the vineyard (Cabaleiro and Segura 1997, 2008). In most cases, the diffusion gradient increases in the opposite direction of an adjacent older, infected vineyard (Klaassen et al. 2011). The major environmental factor affecting long-distance mealybug aerial dispersal is wind, which has been shown to disperse mealybugs across distances greater than 50 km (Barrass, Jerie, and Ward 1994).

Inter-vineyard GLRD transmission can be depicted as a problem of transboundary renewable resource management in ecologically-connected and independently-managed systems (Munro 1979; Bhat and Huffaker 2007). In the case of agricultural diseases, differences in the value of the product affects disease control strategies employed by growers. Using plot-level panel data, Lybbert et al. (2010) find that high-value winegrape growers make greater efforts to control powdery mildew treatment strategies in response to disease forecast information more than their low-value winegrape growers counterparts. Fuller (2011) finds that the optimal strategy for winegrape growers is to abandon the blocks infected by Pierce's Disease if prices paid for grapes were below a certain threshold. In ecologically-connected vineyards, such value or price differential might cause conflicts in disease management strategies whereby the low-value vineyard acts as a vector reservoir of GLRD for the high-value one. When vineyards are independently managed, negative spatial externalities can arise due to the failure of one manager to internalize the negative externality of her disease management decisions on her neighbor's expected revenues.

Traditionally, the literature on the economics of controlling insect-borne plant diseases has examined the temporal dimensions of disease control strategies (e.g. Hall and Norgaard.

1973; Regev et al. 1976). In the last decade, however, studies have concentrated on the spatial dimensions of disease control strategies. Brown, Lynch and Zilberman (2002) show that vineyard managers can maximize profits by planting barriers or by removing the source of Pierce's Disease. Their model, however, is static and largely ignores certain critical production costs such as replanting. More importantly, the authors fail to consider adjacent vineyards and incentives for cooperation. More recently, Fuller et al. (2011) address the limitations of Brown, Lynch and Zilberman (2002) and show that cooperative management of Pierce's Disease can reduce grapevine losses and land abandonment. Nevertheless, their study does not develop explicit cooperation mechanisms among vineyard managers.

We contribute to the disease control bioeconomic literature by extending previous work on disease management in vineyards where inter-vineyard transmission was not considered (Atallah et al. 2013). We also contribute to the literature that models the inter-plot disease transmission by developing a plant-level, spatial-dynamic disease dispersal function as opposed to using a farm-level function as in Keeling et al. (2001) or a fixed diffusion as in Fuller et al. (2011). We embed the disease diffusion model in a two-agent bargaining game to generate distributions of payoffs under possible combinations of disease management strategies. To do this, we build on the literature on transboundary resource management that has used gametheoretic frameworks to characterize bargaining games between adjacent resource owners or managers (Munro 1979; Sumaila 1997; Bhat and Huffaker 2004).

Our results suggest that, under no cooperation strategies, both growers optimally choose not to control the disease. However, if the managers agree to cooperate, it is optimal for the highvalue manager to pay the low-value manager to exit production.

A Bioeconomic Model of Disease Diffusion under Spatial Externalities

We consider two ecologically-connected, independently-managed vineyard plots. Vineyard 1 is represented by grid G_1 that is the set of I * J cells denoted by their row and column position (i, j)and representing grapevines. Similarly, Vineyard 2 is represented by grid G_2 that consists of M * N cells denoted by their row and column position (m, n). The problems faced by the managers of vineyards 1 and 2 differ in their initial conditions and bioeconomic parameters. We therefore restrict the model description to plot G_1 . Vineyard 1 produces high-value winegrapes whereas Vineyard 2 produces low-value winegrapes. The disease is introduced through infected plant material to Vineyard 2, from which it spreads to Vineyard 1. Each grapevine is modeled as a cellular automaton. A vine's infection state transitions are governed by a Markov Chain model. We first expose the growers' private profit maximization problem. Later, we expose the Nash bargaining maximization problem under cooperative disease management.

Bioeconomic model

The objective of Grower 1 is to maximize the expected net present value $(ENPV_1)$ from vineyard plot G_1 by choosing an optimal disease control strategy from a set of alternatives. Based on this strategy, the grower decides, for each vine in cell (i, j) in each period t of T discrete periods of time, whether or not to rogue and replant $(u_{w_{i,j,t}} = 1 \text{ if roguing takes place, 0 otherwise})$, test for the virus $(v_{w_{i,j,t}} = 1 \text{ if virus testing takes place, 0 otherwise})$, or rogue without replanting $(z_{w_{i,j,t}} = 1, \text{ if roguing without replanting take place, 0 otherwise})$.¹ The grower's disease control decisions are based on a vine's composite age-infection state $W_{i,j,t}$. This composite state $W_{i,j,t}$ is composed of a vine's age $A_{i,j,t}$ and its infection $S_{i,j,t}$ states. $A_{i,j,t}$ is a 600 * 1 vector holding a 1 for a vine's age in months and zeros for the other possible ages. A vine can be in one of 5

¹ 'Roguing and replanting' and 'roguing without replanting' are mutually exclusive strategies.

infection states: *Healthy* (*H*), *Exposed-undetectable* (E_d), *Exposed-undetectable* (E_u), *Infective-moderate* (I_m) and *Infective-high* (I_h). $S_{i,j}^t$ is the age-infection state vector at time *t* of dimension 5*1. The vector holds a 1 for the state that describes a vine's infection status and zeros for the remaining four states. The optimal sequence of control variables { $u_{w_{i,j,t}}, v_{w_{i,j,t}}, z_{w_{i,j,t}}$ } for each vine is the one that allocates disease control effort over space and time so as to yield the maximum vineyard ENPV improvement over the strategy of no disease control. Revenues from a vine $r_{w_{i,j,t}}$, depend on its age-infection state $W_{i,j,t}$, which is determined by the stochastic disease diffusion process. Letting *E* be the expectation operator over the random variable $r_{w_{i,j,t}}, \rho^t$ the discount factor 2 at time *t* (*t*>0), *t* \in *T* time in months, where $T=\{0,1,2,...,T_{max}\}$, the objective of a vineyard manager is to maximize the ENPV as follows:³

$$(1) \max_{\{u_{w_{i,j,t}}, v_{w_{i,j,t}}, z_{w_{i,j,t}}\}} E \sum_{t} \rho^{t} \sum_{(i,j)} \sum_{w_{i,j}} \begin{cases} r_{w_{i,j,t}} * \left(1 - \sum_{\tau=0}^{\tau_{max}} u_{w_{i,j,t-\tau}}\right) * \left(1 - z_{w_{i,j,t}}\right) \\ - \sum_{\tau=0}^{\tau_{max}} \left(u_{w_{i,j,t-\tau}} * c_{u_{i,j}}\right) - \left(v_{w_{i,j,t}} * c_{v_{i,j}}\right) - \left(z_{w_{i,j,t}} * c_{z_{w_{i,j,t}}}\right) - C_{i,j,t} \end{cases}$$

The first expression in the curly brackets of equation 1 represents the revenue of a vine in location (i, j) and age-infection state $W_{i,j,t}$ at time t. If a manager decides to rogue and replant a vine in the last τ_{max} periods, $u_{w_{i,j,t-\tau}}$ is equal to 1 and the revenue is multiplied by zero for a period of τ_{max} months until the replant bears fruit, where $\tau \in \{1, 2, ..., \tau_{max}\}$ and $\sum_{\tau=0}^{\tau_{max}} u_{w_{i,j,t-\tau}} \leq 1.^4$ If a manager decides to rogue a vine without replanting it $(z_{w_{i,j,t}} = 1)$,

 $\overline{\rho}^{t} = \frac{1}{(1+r)^{t}}$, where r is the discount rate.

³ Please refer to table 1 for variable and parameter notation and meaning.

⁴ This condition says that roguing and replanting in cell (i, j) cannot occur more than once in τ_{max} periods. It implies that a replant is never rogued before it bears fruit.

the revenue from the cell corresponding to this vine equals zero from t to T. The second expression in the curly brackets of equation 1 represents the costs of roguing-and-replanting $(c_{u_{i,j}})$, the cost of testing $(c_{v_{i,j}})$, and the cost of roguing-without-replanting $(c_{z_{w_{i,j,t}}})$ premultiplied by their corresponding binary choice variables, in addition to other costs of production $(C_{i,j,t})$.⁵

A vine's infection and age states map into a third dynamic state variable, its economic value, or per-vine revenue, $r_{w_{i,j,t}}$ defined as follows:

$$(2) \quad r_{w_{i,j,t}} = 0 \text{ if } A_{i,j,t} < \tau_{max}$$

$$= r_{S_{i,j,t}} \left(y_{S_{i,j,t=H}}, \widetilde{y_{S_{i,j,t=H}}}, \widetilde{p_{S_{i,j,t=H}}} \right) \text{ if } A_{i,j,t} \ge \tau_{max}$$

$$(3) \quad r_{S_{i,j,t}} \left(y_{S_{i,j,t=H}}, \widetilde{y_{S_{i,j,t=H}}}, \widetilde{p_{S_{i,j,t=H}}} \right) = y_{S_{i,j,t=H}} * \left(1 - \widetilde{y_{S_{i,j,t=H}}} \right) * p_{S_{i,j,t=H}} * \left(1 - \widetilde{p_{S_{i,j,t}}} \right)$$

Per-vine revenue equals zero if the vine's age $A_{i,j}^t$ is below τ_{max} (equation 2). Beyond that age, $r_{w_{i,j,t}}$ is known to the vineyard manager at time t. Nevertheless, the per-vine revenue is random for periods prior to t because it depends on the vine's infection state $S_{i,j}^t$. This is because GLRD causes a yield reduction of $\widetilde{y_{S_{i,j,t}}}$ compared to the yield of a healthy vine $(y_{S_{i,j,t=H}})$. In addition, grapes from GLRD-affected vines are subject to a price penalty $\widetilde{p_{S_{i,j,t}}}$ (equation 3) imposed on the price paid for grapes harvested from healthy vines $(p_{S_{i,j,t=H}})$. ^{6,7}

In each period, the infection state transition of a vine (i, j) is given by an infection-state transition equation:

⁵ We only include production costs that are different for managers 1 and 2, namely cultural costs (mostly canopy management).

⁶ The same description applies to cells (m, n) in grid G_2 . See table 2 for the notational differences between plots 1 and 2.

⁷ Per-vine yield $(y_{S_{i,j,t=H}})$ is obtained by dividing per-acre yield (y_{G_1}) over planting density (d_{G_1}) : $y_{S_{i,j,t=H}} = \frac{y_{G_1}}{d_{G_2}}$

(4)
$$\boldsymbol{S}_{i,j,t} = \boldsymbol{P}^t \, \boldsymbol{S}_{i,j,0}$$

Given each vine's initial state $S_{i,j}^0$, and an infection state transition matrix P, its state at time t ($S_{i,j,t}$) is computed according to equation 4. Disease diffusion is spatially constrained by the vineyard's horizontal (eq. 5a) and vertical boundaries (eq. 5b).⁸

$$(5a) \quad (i-1) \in \{1, \dots, I-1\}; (i+1) \in \{2, \dots, I\}.$$

(5b)
$$(j-1) \in \{1, \dots, J-1\}; (j+1) \in \{1, \dots, J-1\};$$

We describe in what follows how the infection state transition probability matrix P governs the disease diffusion. Vines in state H are susceptible to infection. Once they are exposed to the virus, they enter a latency period during which they are nonsymptomatic, and noninfective. This transition is governed by vector B. If the virus population in the vine is below levels that can be detected by virus tests, the disease is undetectable and the vine is in state E_u . The vine will transition later to state E_d according to parameters a, b, and m. In state E_d , the disease can be detected using virus tests. The transition from E_d to I_m is governed by vector C and marks the end of the latency period and the beginning of the infectivity period as well as the onset of visual symptoms. Symptoms, which consist of reddening and downward rolling of the leaves, are at the moderate severity level first (I_m), and transition to a state of high level severity (I_h) according to parameter ϕ . Mathematically, P can be expressed as follows:⁹

(6)
$$\boldsymbol{P} = \begin{pmatrix} (\mathbf{1} - \boldsymbol{B})^T & \boldsymbol{B}^T & 0 & 0 & 0 \\ 0 & 1 - \frac{(x-a)^2}{(b-a)(m-a)} & \frac{(x-a)^2}{(b-a)(m-a)} & 0 & 0 \\ 0 & 0 & (\mathbf{1} - \boldsymbol{C})^T & \boldsymbol{C}^T & 0 \\ 0 & 0 & 0 & e^{-\varphi} & (1 - e^{-\varphi}) \\ 0 & 0 & 0 & 0 & 1 \end{pmatrix}$$

⁸ These spatial constraints are formulated by defining the set of indices that vine (i, j)'s within-column (eq. 5a) and the across-column neighbors (eq. 5b) can have. They ensure that the disease does not spread beyond the vines situated at the borders of the vineyard.

⁹*P* reads from row (states H, E_u, E_d, I_m, I_h at time t) to column (states H, E_u, E_d, I_m, I_h at time t+1).

B is the *Healthy* to *Exposed-undetectable* vector of transition probabilities conditional on

previous own and neighborhood infection states:

$$(7) \qquad B = \begin{pmatrix} \Pr(S_{i,j}^{t+1} = E \mid S_{i,j}^{t} = H, S_{N_{i,j,k}}^{t} = S_{N_{i,j,1}}^{t}) \\ \Pr(S_{i,j}^{t+1} = E \mid S_{i,j}^{t} = H, S_{N_{i,j,k}}^{t} = S_{N_{i,j,2}}^{t}) \\ \Pr(S_{i,j}^{t+1} = E \mid S_{i,j}^{t} = H, S_{N_{i,j,k}}^{t} = S_{N_{i,j,3}}^{t}) \\ \Pr(S_{i,j}^{t+1} = E \mid S_{i,j}^{t} = H, S_{N_{i,j,k}}^{t} = S_{N_{i,j,3}}^{t}) \\ \Pr(S_{i,j}^{t+1} = E \mid S_{i,j}^{t} = H, S_{N_{i,j,k}}^{t} = S_{N_{i,j,3}}^{t}) \\ \Pr(S_{i,j}^{t+1} = E \mid S_{i,j}^{t} = H, S_{N_{i,j,k}}^{t} = S_{N_{i,j,3}}^{t}) \\ \Pr(S_{i,j}^{t+1} = E \mid S_{i,j}^{t} = H, S_{N_{i,j,k}}^{t} = S_{N_{i,j,3}}^{t}) \\ \Pr(S_{i,j}^{t+1} = E \mid S_{i,j}^{t} = H, S_{N_{i,j,k}}^{t} = S_{N_{i,j,3}}^{t}) \\ \Pr(S_{i,j}^{t+1} = E \mid S_{i,j}^{t} = H, S_{N_{i,j,k}}^{t} = S_{N_{i,j,14}}^{t}) \\ \Pr(S_{i,j}^{t+1} = E \mid S_{i,j}^{t} = H, S_{N_{i,j,k}}^{t} = S_{N_{i,j,15}}^{t}) \\ \Pr(S_{i,j}^{t+1} = E \mid S_{i,j}^{t} = H, S_{N_{i,j,k}}^{t} = S_{N_{i,j,16}}^{t}) \\ \Pr(S_{i,j}^{t+1} = E \mid S_{i,j}^{t} = H, S_{N_{i,j,k}}^{t} = S_{N_{i,j,16}}^{t}) \\ \Pr(S_{i,j}^{t+1} = E \mid S_{i,j}^{t} = H, S_{N_{i,j,k}}^{t} = S_{N_{i,j,16}}^{t}) \\ \Pr(S_{i,j}^{t+1} = E \mid S_{i,j}^{t} = H, S_{N_{i,j,k}}^{t} = S_{N_{i,j,16}}^{t}) \\ \Pr(S_{i,j}^{t+1} = E \mid S_{i,j}^{t} = H, S_{N_{i,j,k}}^{t} = S_{N_{i,j,16}}^{t}) \\ \Pr(S_{i,j}^{t+1} = E \mid S_{i,j}^{t} = H, S_{N_{i,j,k}}^{t} = S_{N_{i,j,16}}^{t}) \\ \frac{1 - e^{-(\alpha + \gamma_{2,1})}}{1 - e^{-(\alpha + \gamma_{2,1})}} \\ \frac{1 - e^{-(\alpha + \gamma_{2,1})}}{1 - e^{-(\alpha + \gamma_{2,1})}} \\ \frac{1 - e^{-(\alpha + \gamma_{2,1})}}{1 - e^{-(\alpha + \gamma_{2,1})}} \\ \frac{1 - e^{-(\alpha + \gamma_{2,1})}}}{1 - e^{-(\beta + \gamma_{2,1})}} \\ \frac{1 - e^{-(\beta + \gamma_{2,1})}}}{1 - e^{-(\beta + \gamma_{2,1})}}} \\ \frac{1 - e^{-(\beta + \gamma_{2,1})}}}{1 - e^{-(\beta + \gamma_{2,1})}}} \\ \frac{1 - e^{-(\beta + \gamma_{2,1})}}}{1 - e^{-(\beta + \gamma_{2,1})}}} \\ \frac{1 - e^{-(\beta + \gamma_{2,1})}}}{1 - e^{-(\beta + \gamma_{2,1})}}} \\ \frac{1 - e^{-(\beta + \gamma_{2,1})}}}{1 - e^{-(\beta + \gamma_{2,1})}}} \\ \frac{1 - e^{-(\beta + \gamma_{2,1})}}}{1 - e^{-(\beta + \gamma_{2,1})}}} \\ \frac{1 - e^{-(\beta + \gamma_{2,1})}}}{1 - e^{-(\beta + \gamma_{2,1})}}} \\ \frac{1 - e^{-(\beta + \gamma_{2,1})}}}{1 - e^{-(\beta + \gamma_{2,1})}}} \\ \frac{1 - e^{-(\beta + \gamma_{2,1})}}}{1 - e^{-(\beta + \gamma_{2,1})}}} \\ \frac{1 - e^{-(\beta + \gamma_{2,1})}}}{1 - e^{-(\beta + \gamma_{2,1})}$$

In equation (7), $S_{N_{i,j,k}}^{t}$ is the infectivity of a vine's von Neumann neighborhood.¹⁰ For example, $S_{N_{i,j,2}}^{t} = (I, I, I, NI)$ is a neighborhood composed of two within-column infective (I) neighbors, one across-column infective neighbor and one across-column noninfective (NI) neighbor. Given that each of the four neighbors can be in one of two infectivity states (I or NI), $S_{N_{i,j,k}}^{t}$ can be in one of 2⁴ states¹¹, where $k \in \{1, ..., 16\}$ denotes 1 of the 16 possible neighborhood infectivity states in equation 7. α and β are the within- and across-column transmission rates with $0 < \beta < \alpha$, suggesting that infective vines transmit the disease to their von-Neumann neighbors within the same grid column at a higher rate than they transmit it to their von-Neumann neighbors situated in the adjacent grid column. We choose this neighborhood-based infection state transition to

¹¹
$$S_{N_{i,j,k}}^t \in \{S_{N_{i,j,1}}^t = (I, I, I, I), S_{N_{i,j,2}}^t = (I, I, I, NI), S_{N_{i,j,3}}^t = (I, I, NI, NI), ..., S_{N_{i,j,16}}^t = (NI, NI, NI, NI)\}$$

¹⁰ This type of neighborhood, where a cell is in contact with its four neighbors in the four cardinal directions, represents the most common vertical trellis system where each vine is in contact with its four surrounding vines (Striegler and Jones 2012). In contrast, a horizontal trellis system favors contact with up to eight neighbors (Cabaleiro and Segura 2006) and would be more accurately represented by a Moore neighborhood.

reflect patterns of GLRD diffusion observed in spatial analyses where the disease is shown to spread preferentially along grid columns (Habili et al. 1995; Le Maguet et al. 2012). We assume that within- and across-column infections occur independently in Poisson processes with rate parameters α and β with $\theta < \beta < \alpha$. That is, random variables T_t and T_2 , independently distributed with p.d.f.'s $\alpha e^{-\alpha t}$ and $\beta e^{-\beta t}$, govern the within-column and the across-column state transitions, respectively. The within- and across-column *Healthy* to *Exposed-undetectable* state transition probabilities are triggered in each time step by a random variable u_t . Where u_t is a random draw from $U \sim (0, 1)$, the disease is transmitted from one infective vine to another healthy vine in the same column at time t+1 if $u_t < 1 - e^{-\alpha}$. Conversely, the disease is not transmitted if $u_t \ge 1 - e^{-\alpha}$. Similarly, the disease is transmitted from one infective vine to another healthy vine in an adjacent column at time t+1 if $u_t < 1 - e^{-\beta}$ and is not transmitted if $u_t \ge 1 - e^{-\beta}$. Longdistance dispersal of vectors from G_2 to G_1 is governed by a random variable T_3 , independently distributed with p.d.f. $\gamma_{2,1} e^{\gamma_{2,1} t}$. The parameter $\gamma_{2,1}$ is specified by the following spatialdynamic dispersal function.

(8)
$$\gamma_{2,1} = j^{-\gamma} * \sum_{n \in \mathbb{N}} \sum_{m \in \mathbb{M}} \frac{(V_{m,n} | S_{m,n}^t = Infective) * n}{M * (N - n + 1)}, \quad \gamma > 0$$

For a vine (i, j), $\gamma_{2,1}$ is inversely proportional to the distance from the border (column *J*) that serves as a point of entry of the disease vectors in vineyard G_2 . This distance is given by the column position *j* of the vine.¹² We choose a power-law dispersal specification because it can model the GLRD inter-vineyard transmission characteristic whereby new infection foci of

¹² For the power-law dispersal parameter γ , we use the estimated slope of the disease gradient obtained by regressing the natural logarithm of GLRD incidence on the natural logarithm of the distance (column position) in the Sisan vineyard plot in Cabaleiro and Segura (1997) (table 1).

infection emerge beyond the disease front (Gibson 1997; Reynolds 2011).¹³ $\gamma_{2,1}$ is also proportional to the total number of *Infective* vines in G_2 ($V_{m,n} | S_{m,n}^t = Infective$), weighted by their column position *n* (numerator in equation 8).¹⁴ The denominator in equation 8 allows the multiplier of the power- law expression (the term in the double summation) to vary between 0 and 1 as the number of *Infective* vines in G_2 varies between 0 and M * N. When more than one type of transmission can be realized, such as when a vine has one *Infective* within-column neighbor, one *Infective* within-row neighbor, and is situated on the border of the vineyard, the realized type of transmission is determined by the smaller of T_1 , T_2 and T_3 (Cox 1959).

The transition from *Exposed-undetectable* (E_u) to *Exposed-detectable* (E_d) is governed by a period during which the disease cannot be detected in the vine. Cabaleiro and Segura (2007) and Constable et al. (2012) report minimum, maximum and most common values for the period in which a vine is infected but undetectable. With no further knowledge on the distribution of this period, we model it as a random variable drawn from a triangular distribution with parameters *a* (minimum), *b* (maximum), and *m* (mode). Then, if T_4 is the period it takes a vine to transition from E_u to E_d , the probability that the transition happens in less than *x* time periods, or $\Pr(T_3 < x)$, is $\frac{(x-a)^2}{(b-a)(m-a)}$ for $a \le x \le m$. The probability is equal to 0 for x < a, $(1 - \frac{(b-x)^2}{(b-a)(m-a)})$ for $m \le x < b$, and 1 for x > b (Kotz and Rene van Dorp 2004).

The *Exposed-detectable* to *Infective* state transition probabilities conditional on age category are given by vector C as follows:

¹³ Alternatively, an exponential specification produces a homogeneous expanding front without new distant infection foci.

¹⁴ Weighting each *Infective* vine by its column position n allows vines in bordering columns to contribute more to the increase in $\gamma_{2,1}$ than vines situated farther from the border.

(9)
$$C = \begin{pmatrix} \Pr\left(S_{i,j}^{t+1} = I \mid S_{i,j}^{t} = E, A_{i,j}^{t} = Young\right) \\ \Pr\left(S_{i,j}^{t+1} = I \mid S_{i,j}^{t} = E, A_{i,j}^{t} = Mature\right) \\ \Pr\left(S_{i,j}^{t+1} = I \mid S_{i,j}^{t} = E, A_{i,j}^{t} = Old\right) \end{pmatrix} = \begin{pmatrix} 1 - e^{-\lambda_{y}} \\ 1 - e^{-\lambda_{m}} \\ 1 - e^{-\lambda_{o}} \end{pmatrix}$$

These probabilities are conditional on own previous infection state and age category. Younger vines have shorter latency periods (Pietersen 2006). Therefore, the rate of transition out of latency decreases with age implying that $\lambda_y > \lambda_m > \lambda_o$, where the subscripts *y*, *m* and *o* denote young (0-5 years), mature (5-20 years) and old (>20 years) vines, respectively. Conversely, where *L* is the latency period, $L_y < L_m < L_o$.

Finally, once a vine becomes *Infective* and has moderate symptoms, symptom severity increases over time and reaches a high level after a period *Inf*, which is exponentially distributed with fixed rate parameter φ . Thus, the probability that a vine transitions from *Infective moderate* (I_m) to *Infective-high* (I_h) is Pr $(S_{i,j}^{t+1} = I_h | S_{i,j}^t = I_m) = 1 - e^{-\varphi}$. Symbols, definitions, values, and references for the disease diffusion parameters are presented in table 1.

[Table 1 here]

Model initialization

Grapevines are initialized *Healthy* and of age equal zero ($S_{i,j}^t = H$ and $A_{i,j}^t = 0$) in both vineyard plots G_1 and G_2 (high- and low-value vineyard, respectively). At t = 1, one percent of the grapevines in G_2 are chosen at random from a uniform spatial distribution U (0, M * N) to transition from *Healthy* to *Exposed*.¹⁵ Subsequently, GLRD spreads within G_2 according to the infection-state transition Markov Chain process in equation 4. Subsequently, the *Infective* vines act as a primary source of infection for the healthy vines in G_1 . The disease spreads from G_2 to G_1 according to the distance-and-density-dependent dispersal function $\gamma_{2,1}$ (equation 8). We

¹⁵ Recall that M * N is the total number of grapevines in G_2 where M and N are the number of rows and columns, respectively.

assume that inter-vineyard disease diffusion is unidirectional, that is, *Infective* vines in G_1 do not act as a source of re-infection for *Healthy* vines in G_2 ($\gamma_{12}=0$).¹⁶

The transmission from G_2 to G_1 is caused by the private disease management decisions taken by Grower 2 who faces bioeconomic parameters that makes it optimal not to control the disease. Although Grower 2 has a similar objective function to Grower 1 (equation 1), her production system consists of higher per-vine yields $(y_{S_{m,n,t=H}} > y_{S_{i,j,t=H}})$ and lower per-ton prices for wine grapes $(p_{S_{m,n,t=H}} < p_{S_{i,j,t=H}})$. The disease yield impact is similar in both plots $(\widetilde{y_{w_{i,j,t}}} = \widetilde{y_{w_{m,n,t}}})$ but the quality penalty is higher for the higher value winegrapes $(\widetilde{p_{S_{i,j,t}}} > \widetilde{p_{S_{m,n,t}}})$. Both vineyard managers face the same disease management unit costs $(c_{z_{i,j}} = c_{z_{n,m}}, c_{u_{i,j}} = c_{u_{m,n}}, c_{v_{i,j}} = c_{v_{m,n}})$. However, Grower 1 undertakes vineyard management practices that are more labor-, and machinery-intensive, which translate into higher cultural costs per vine $(C_{i,j,t} > C_{m,n,t})$. Per-vine costs are independent of the level of roguing. Symbols, definitions, values, and references for the economic parameters are presented in table 2.

[Table 2 here]

Experimental Design

We design and implement Monte Carlo experiments to evaluate various disease control strategies by comparing their economic outcomes to those resulting from a *no disease control* strategy. Each experiment consists of a set of 1,000 simulation runs, for each vineyard plot, over 600 months. Experiments differ in the disease control strategies employ. Outcome realizations for a run within an experiment differ due to random spatial initialization in G_2 , and random spatial

¹⁶ Wilen (1997) refers to this as a [unidirectional] corridor invasion where one landowner is in advance of the invading front.

disease diffusion within each vineyard and from G_2 to G_1 . Data collected over simulation runs are the probability density functions of the ENPVs under each strategy.

Disease control strategies

We formulate and evaluate three sets of disease control strategies. The first set has *nonspatial strategies* that consist of roguing vines and replacing them based on their symptomatic infection state and/or their age. The second set has *spatial strategies* that consist of roguing-and-replanting symptomatic vines, but also testing their nonsymptomatic neighbors, and roguing-and-replanting them if they test positive. The third set has *fire-break strategies* that consist in roguing (without replanting) vines in the border columns of a vineyard in order to create 'fire breaks' that would reduce disease diffusion from to G_2 to G_1 . All strategies are available to both growers. For conciseness, we describe them below using the notation of G_1 only.

In the set of *nonspatial strategies*, the grower decides whether to rogue and replace $(u_{w_{i,j,t}} = 1)$ symptomatic vines based on their states $W_{i,j,t}$, specifically their infection severity (*Infective-moderate; Infective-high*) and/or their age category (*Young:* 0-5; *Mature:* 6-19; *Old:* 20 and above). The infection-age control strategies are the following: (1) no disease control (provides a baseline for comparison); (2) roguing and replacing vines that are *Infective-moderate* and *Young* (strategy $I_m Y$); (3) roguing and replacing vines that are *Infective-moderate* and *Mature* (strategy $I_m M$); (4) roguing and replacing vines that are *Infective-moderate* and *Old* (strategy $I_m O$); (5) roguing and replacing vines that are *Infective-high* and *Mature* (strategy $I_h M$); (6) roguing and replacing vines that are *Infective-high* and *Old* (strategy $I_h O$); ¹⁷ (7) roguing and replacing all vines that are *Infective-high*; and (9) roguing and replacing all vines that are *Infective*. Strategies (7), (8), and

¹⁷ We exclude the strategy of roguing and replacing *Infective-high* and *Young* (I_hY) because this age-infection combination cannot be reached; it takes a vine more than 5 years to transition to the *Infective-high* state.

(9) are intended to examine the impact of structuring control strategies by age and infection states on ENPV improvement over the baseline of no disease control, compared to their counterparts that are not structured by either age or infection state

In the set of *spatial strategies*, the vineyard manager decides whether to rogue and replant symptomatic vines ($u_{w_{i,j,t}} = 1$), test their neighbors ($v_{w_{i,j,t}} = 1$) and rogue-and-replace them ($u_{w_{i,j,t}} = 1$) if they test positive. The *spatial strategies* are the following: (1) roguing and replacing symptomatic vines in addition to testing their two within-column neighbors and roguing them if they test positive (strategy I_mNS); (2) roguing and replacing symptomatic vines in addition to testing their symptomatic vines in addition to testing the von-Neumann neighbors (two across-column neighbors and two-within column neighbors) and roguing them if they test positive (strategy I_mNSEW); (3) roguing and replacing symptomatic vines in addition to testing their four within-column neighbors and two across-column neighbors and roguing them if they test positive (strategy I_mNSEW); (4) roguing and replacing symptomatic vines in addition to testing their four within-column and four within-row neighbors and roguing those that test positive (strategy I_mNS2EW).

In the set of fire-break strategies, vines in bordering columns are rogued without replanting ($z_{w_{i,j,t}} = 1$) at t = 24, which corresponds to the moment when initially infected vines in G_2 develop visual leafroll symptoms. The strategies consist of creating a fire-break barrier of increasing width up to roguing all the vines in a vineyard. For example strategy1*Col* consists of roguing without replanting one bordering column and strategy *16Col* consists of roguing without replanting columns.

Noncooperative and cooperative games with side payments

In order to find the optimal noncooperative disease control strategy for each vineyard manager, we employ the objective function (equation 1) to rank the vineyard expected net present values

under the alternative strategies. The objective function takes into account the total amount of control realized under each strategy but also the timing, intensity and location of that control. Given that inter-vineyard transmission is unidirectional ($\gamma_{2,1} > 0$, $\gamma_{2,1} = 0$), in the noncooperative game we first solve for the optimal strategies for G_2 . Then, given G_2 's optimal strategy, G_1 solves for her optimal strategy. The expected payoffs of noncooperative strategies constitute the players' threat points (Nash 1953). We assume that each grower can observe disease diffusion and control in her neighbor's vineyard.

If the two vineyards are cooperatively managed, G_1 and G_2 solve the Nash bargaining game to find payoffs that ensure the existence of a mutually beneficial agreement. The Nash bargaining solution (NBS) is the unique pair of cooperative payoffs ($ENPV_1^C$, $ENPV_2^C$) that solves the following maximization problem (Nash 1953):

(10)
$$\max_{\{ENPV_1, ENPV_2\}} (ENPV_1^C - ENPV_1^{NC}) (ENPV_2^C - ENPV_2^{NC})$$

where the maximand, known as the Nash product, is the product of the differences between the cooperative and noncooperative payoffs for G_1 and G_2 and $ENPV^C \ge ENPV^{NC}$. Under the standard axiomatic bargaining theory, Eq. (10) has a unique solution (Muthoo 1999).¹⁸

(11)
$$ENPV_{1}^{C} = ENPV_{1}^{NC} + \frac{1}{2} \left[(ENPV_{1}^{C} + ENPV_{2}^{C}) - (ENPV_{1}^{NC} + ENPV_{2}^{NC}) \right]$$

(12)
$$ENPV_2^C = ENPV_2^{NC} + \frac{1}{2} \left[(ENPV_1^C + ENPV_2^C) - (ENPV_1^{NC} + ENPV_2^{NC}) \right]$$

The solution can be interpreted as follows: the growers agree that each first gets her expected noncooperative payoff (the payoff of the threat point, or the disagreement point) and then they equally split the expected cooperative surplus. The latter is defined as the difference between the aggregate expected cooperative $(ENPV_1^C + ENPV_2^C)$ and the aggregate expected noncooperative

¹⁸ The axioms are individual rationality, invariance to equivalent utility representations, symmetry, and independence of irrelevant alternatives.

payoffs $(ENPV_1^{NC} + ENPV_2^{NC})$. The equal allocation of the expected cooperative surplus is implemented through a side-payment that consists of compensation up to the threat point (i.e., the difference between the noncooperative and cooperative expected payoffs) plus half of the expected benefits to cooperation.^{19 20}

Results and Discussion

We find that "no disease control" is optimal among all noncooperative disease management strategies for both growers. However, a cooperative disease management strategy achieves an overall economic improvement of 25% over the baseline of no control. We show that a Pareto-efficient lump sum side payment from the manager of the high-value, healthy vineyard (G_1) to the manager of the low-value, diseased vineyard (G_2) can achieve this cooperative outcome.

Noncooperative disease management

Our simulation results indicate that the vineyard ENPV is highest (\$37,000/acre) for the lowervalue vineyard G_2 when the manager does not to control the disease, compared to the strategies I_mY , I_mNS , and I_mNSEW (table 3).^{21 22} Given G_2 's decision of not controlling the disease, G_1 's

¹⁹ We also solve a game that features alternating offers through an infinite time horizon (Rubenstein 1982) and obtain the same solution as in the Nash bargaining game. Both bargaining games have the same optimal solution because our players have the same discount rate. In situations where players have different discount rates, the cooperative surplus is shared proportionally according to those rates: more 'patient' players get a higher share of the surplus. Our result is also a special case of the solution to the generalized (or asymmetric) Nash bargaining game where players have the same 'bargaining power' (Muthoo 1999, p. 35). Muthoo (1999, p. 52) also shows that the bargaining outcome generated by the Nash bargaining game is identical to the limiting outcome generated by the basic alternating-offers model (Rubenstein 1982) when bargaining costs are small.

²⁰ An alternative way to solve the problem is the proportional solution to the surplus-sharing model (Moulin 1988). According to this solution, the surplus is shared proportionally to a player's relative opportunity cost (noncooperative payoff).

²¹ Recall that these strategies consist of roguing-and-replanting the moderately infected and young vines ($I_m Y$); roguing-and-replanting the symptomatic vines, testing their two nonsymptomatic neighbors and roguing-and-replanting them if they test positive ($I_m NS$); and roguing-and-replanting the symptomatic vines, testing their four nonsymptomatic neighbors and roguing-and-replanting them if they test positive ($I_m NS$).

optimal response is not to control the disease either. This is because G_1 's ENPV is highest under no disease control (\$214,000/acre), which is greater than the ENPV corresponding to the I_mY , I_mNS , and I_mNSEW strategies. As a result, the Nash equilibrium is for neither grower to control the disease. The strategy of *no control* is therefore a credible threat point for G_2 in a cooperative game. The optimal noncooperative expected payoffs for growers G_1 and G_2 are \$214,000 and \$37,000, respectively.

[Table 3 here]

Table 3 shows that the noncooperative winning strategy (*no control*, *no control*) does not yield the highest aggregate possible expected payoff. In fact, if the growers were to cooperate and agree that G_2 rogues all her vines according to the spatial strategy I_mNS^{23} , the aggregate payoff (ENPV) would be \$297,000, which is an improvement of 18% over the noncooperative aggregate payoffs. Moreover, we find that if G_1 pays G_2 to exit production (strategy *I6col*) the aggregate payoff (ENPV) would be \$313,000, or an improvement of 25% over the noncooperative aggregate payoffs. These benefits to cooperation are consistent with previous studies on cooperative harvesting in fisheries (Sumaila 1997) and nuisance wildlife species (Bhat and Huffaker 2007). However, strategy I_mNS and strategy *I6col* make the low-value grower G_2 worse off relative to the strategy of no disease control. In this case, the two growers could enter into a self-enforcing cooperative agreement that includes side payments negotiated ex-ante and threats by each manager to revert to noncooperative behavior if a party breaches the agreement.

²² We only mention here the three strategies that yield the highest aggregate ENPV. Note that they include two spatial strategies (I_mNS , and I_mNSEW) and one nonspatial, age-structured strategy (I_mY), and no nonage-structured strategies.

²³ Recall that this strategy consists of roguing and replacing symptomatic vines (I_m) then testing their nonsymptomatic immediate neighbors (NS) and roguing them if they test positive.

Cooperative disease management

A cooperative disease management game unfolds as follows. At the beginning of the game, the growers negotiate a Pareto-efficient disease management agreement that includes a single lumpsum side payment made by the high-value grower to the low-value grower.²⁴ The growers monitor the evolution of the game, ensuring that cooperative disease management is in place and that payments are made on schedule. If growers do not violate the agreement then they would continue cooperating. If either G_1 fails to pay or G_2 fails to implement the cooperative disease management strategy, the other party reverts to her credible threat strategy of no disease control. In order for the cooperative disease management strategy to be Pareto-efficient, G_1 compensates G_2 for the difference between the noncooperative and cooperative expected payoffs of the latter. In addition, the managers get equal share of the total benefits resulting from cooperation.

We show the results from the cooperative games involving spatial strategies in table 4. Among the cooperative spatial strategies, the one that yields the highest payoff consists of G_2 employing the spatial strategy I_mNS and G_1 paying her \$36,000 at the onset of the game. This self-enforcing cooperative disease management strategy achieves benefits to cooperation equal to \$46,000, an overall economic improvement of 18% over the noncooperative strategy. This improvement is statistically significant at the 1% level. The solution is Pareto-efficient and satisfies the individual rationality constraint for both growers. Second-best cooperative strategies all involve G_1 paying G_2 to employ the spatial disease control strategy I_mNS with G_1 either employing the same spatial disease control strategy I_mNS or the age-structured strategy I_mY .

[Table 4 here]

²⁴ The single lump sum payment can be converted to a fixed annual payment using the annuity formula (see Bhat and Huffaker 2007).

This game assumes that enforcement costs are negligible for both growers. However, monitoring costs might be high and thus cause the optimal roguing-and-replanting strategy to yield lower aggregate expected payoffs than alternative strategies. Monitoring would involve G_1 observing whether G_2 is identifying symptomatic vines, roguing-and-replacing them, and testingand-roguing their immediate nonsymptomatic neighbors. An alternative set of cooperative strategies is to establish fire-breaks in the lower-value vineyard G_2 , which are likely to have low monitoring costs for G_1 . These strategies do not require the monitoring needed in the cooperative strategies discussed above (visual identification of GLRD symptoms, testing, replanting). Among the fire-break strategies, the winning cooperative strategy involves G_1 paying G_2 to rogue all her vines without replanting, in which case G_1 adopts a strategy of not controlling the disease (no control, 16Col strategy in table 5). The aggregate expected payoff to this strategy is \$319,000, which is 25% higher than the noncooperative outcome. This improvement is statistically significant at the 1% level, and is higher than the 18% improvement under the best spatial strategy in table 4 (no control, I_mNS). We conclude that it is optimal for G_1 to pay G_2 to exit production and compensate her through a fixed transfer payment of \$74,000 (table 5).

[Table 5 here]

Concluding Remarks and Next Steps

There is growing interest in research on the economics of integrated spatial-dynamic processes in general, and those involving multiple growers with spatial externalities in particular. This paper presents a two-agent bargaining game in the context of a spatial-dynamic model of disease diffusion and control. We apply this model to the GLRD spread and generate distributions of vineyard ENPVs to solve the game. We measure the willingness-to-pay of the high-value grower to control the diffusion externality originating from a neighboring infested vineyard. We find it

optimal for the high-value grower to pay the low-value manager to exit production. This result depends on the difference in the value of production of G_1 and G_2 . We expect that the smaller the price differential between the growers, the smaller the willingness-to-pay of G_1 . In such cases, strategy (*no control*, 16col) would not be optimal.

This model can be adapted to crop diseases characterized by spatial-dynamic processes by adjusting the spatial configuration and input data. In particular, it can be employed to inform profit-maximizing disease management in high-value horticultural crops that are subject to negative, spatial-dynamic externalities. However, it has certain limitations that suggest further areas of research.

Our model has the side payments determined ex-ante and does not accommodate changing circumstances that can shift the bargaining power of the players over the lifecycle of the vineyard. Future research should formulate a dynamic contractual mechanism with renegotiation-proof variable side payments. This is especially relevant given the high level of price variability observed in the winegrape industry and the impact it can have on incentives to cooperate as well as on the size of the payments.

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Table 1	Disease	Diffusion	Parameters
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Parameter	Description	Value	Unit	Sources
α	Within-column H to E_u transition rate	4.200^{*}	month ⁻¹	Model calibration to data in
β	Across-column H to E_u transition rate	0.014*	month ⁻¹	Charles et al (2009) with validation using data in Cabaleiro and Segura (2006) and Cabaleiro et al (2008)
γ	Inter-plot transmission power-law exponent	2.5-3	unitless	Cabaleiro and Segura (1997), table 1 (vineyard Sisan)
L_y	Latency period for young vines	24	months	Age-specific latency periods
L_m	Latency period for mature vines	48	months	constructed based on latency
L_o^m	Latency period for old vines	72	months	period in Jooste, Pietersen, and Burger (2011)
а	Minimum of undetectability period	4	months	Cabaleiro and Segura (2007);
b	Maximum of undetectability period	18	months	Constable et al. (2012)
т	Mode of virus undetectability period	12	months	
Inf	Period spent in state I_m before a vine transitions to state I_h	36	months	M. Fuchs, personal communication, April 9, 2012
$ au_{max}$ T_{max} , A_{max}	Period from planting until productivity Maximum model time, max. vine age	36 600	months months	White (2008) White (2008)

*Transition rates are constant for a particular location over the 50 year period of study. This excludes for instance situations where new insect vector species are introduced and contribute to an increase in transmission rates.

		Plot G₁ ²⁵	1	Plot G₂ ²⁶		
Vineyard layout						
Grid dimensions (rows*columns)	I * J	68*23=1,564	M * N	49*16=784		
Grid row (vine) spacing (ft.)		4		5		
Grid column spacing (ft.)		7		11		
Revenues and revenue parameters						
Per-vine revenue	$r_{w_{i,j,t}}$	Random (eq. 2)	$r_{w_{m,n,t}}$	Random (eq. 2)		
Grapes price (\$/ton)	$p_{S_{i,j,t=H}}$	4,455 2,683 911	$p_{S_{m,n,t=H}}$	480		
Price penalty (%)	$\widetilde{p_{s_{\iota,l,t}}}$	70	$\widetilde{p_{s_{m,n,t}}}$	0		
Yield (tons/acre)	\mathcal{Y}_{G_1}	4.5	y_{G_2}	10		
Yield (tons/acre/month)	J 0 ₁	0.375	<i>J</i> 0 ₂	0.834		
Planting density (vines/acre)	d_{G_1}	1,564	d_{G_2}	784		
Yield (tons/vine/year)	$y_{S_{i,j,t=H}}$	0.0029	$y_{S_{m,n,t=H}}$	0.0128		
Yield (tons/vine/month)	𝔊 𝔅 𝔅,],t=H	0.0002	<i>y sm</i> , <i>n</i> , <i>t</i> = <i>H</i>	0.0011		
Yield reduction (%)	$\widetilde{\mathcal{Y}_{S_{l,l},t}}$	Depends on $S_{i,j,t}$	$\widetilde{\mathcal{Y}_{S_{m,n,t}}}$	Depends on $S_{i,j,t}$		
$S_{i,j}^t = Exposed$	$\widetilde{Y_{S_{l,J,t=E}}}$	30	$\widetilde{y_{S_{m,n,t=E}}}$	30		
$S_{i,j}^t = Infectious, moderate$	$\widetilde{y_{S_{i,j,t=I_m}}}$	50	$\widetilde{\mathcal{Y}_{S_{m,n,t}=I_m}}$	50		
$S_{i,j}^t = Infectious, high$	$y_{\widetilde{S_{i,j,t=I_h}}}$	75	$y_{S_{m,n,t=I_h}}$	75		
Cost parameters						
Roguing and replanting ²⁷ (\$/vine)	$C_{u_{i,j}}$	14.6	$c_{u_{m,n}}$	14.6		
Roguing ²⁸ (\$/vine)	$C_{Z_{i,j}}$	8	$C_{Z_{m,n}}$	8		
Testing (\$/vine)	$C_{v_{i,j}}$	2.6	$C_{v_{m,n}}$	2.6		
Cultural and harvest costs			·			
\$/vine/year	$C_{i,j,t}$	3.6	$C_{m,n,t}$	2.8		
\$/vine/month		0.30		0.23		
Discount factor (month ⁻¹) ²⁹	ρ	0.9959	ρ	0.9959		

Table 2. Economic Parameters Faced by Managers of Plots G_1 and G_2

 ²⁵ Cooper M., K. M. Klonsky, and R. L. De Moura. "Sample Costs to Establish a Vineyard and Produce Winegrapes: Cabernet Sauvignon in Napa County." North Coast Region. University of California Cooperative Extension GR-NC-10 (2012): 1-19.
 ²⁶ Verdegaal, P. S., K. M. Klonsky, and R. L. De Moura. "Sample Costs to Establish a Vineyard and Produce Winegrapes: Cabernet Sauvignon in San Joaquin County." San Joaquin Valley North! University of California Cooperative Extension GR-NC-10 (2012): 1-21.

²⁷ Klonsky, Karen, and Pete Livingston. 2009. Cabernet Sauvignon Vine Loss Calculator. Davis, CA: University of California

²⁸ Klonsky, Karen, and Pete Livingston. 2009. Cabernet Sauvignon Vine Loss Calculator. Davis, CA: University of California

²⁹ Equivalent to an annual discount rate of 5%

	G_1 given G_2									
Strategies	no control				$I_m Y$			$I_m NS$		
				Ex	pected Pa	uyoffs ^a				
G_2	G_1	G_2	Aggregate	G_I	G_2	Aggregate	G_{I}	G_2	Aggregate	
no control	$214(5)^{b}$	37 (1)	251(6)	194 (7)	37 (1)	231 (7)***	125 (6)	37 (1)	163 (6)***	
$I_m Y$	247 (3)	19 (2)	266 (5)***	247 (4)	19 (2)	266 (5)***	184 (4)	19 (2)	203 (6)***	
$I_m NS$	273 (4)	25 (5)	297 (8)***	267(5)	25 (5)	292 (9)***	266 (4)	25 (5)	291 (8)***	
<i>I_mNSEW</i>	269 (4)	-18 (5)	252 (9)***	264 (5)	-18 (5)	246 (10)***	259 (4)	-18 (5)	241 (9)***	

Table 3. Expected Payoffs for Grower 1 (G_1) and Grower 2 (G_2) under Noncooperative Disease Management Strategies (\$1,000 per acre over 50 years)

 ^a Expectations are obtained from 1,000 simulations.
 ^b Standard deviations in parentheses.
 ^{***} Difference with the expected aggregate payoff of the "no control, no control" strategy is significant at the 1% level.

	Expected Payoffs ^a (\$1,000)							
	Fixed							
				Aggregate	Transfer	Cooperative	Cooperative	
	Payoff	Payoff	Aggregate	Benefits to	Payment	Payoff to G_1	Payoff to G_2	
Strategies (G ₁ , G ₂)	to G_1	to G_2	payoff	Cooperation	to G_2^{b}			
Noncooperative								
no control, no control	214 (5) ^c	37 (1)	251 (6)	n/a	n/a	n/a	n/a	
Cooperative								
no control, I_mNS	273 (4)	25 (5)	297 (8)	46 (5)***	36	237	60	
$I_m Y, I_m NS$	267 (5)	25 (5)	292 (9)	41 (5)***	33	234	58	
I_mNS, I_mNS	266 (4)	25 (5)	291 (8)	40 (5)***	33	234	57	

Table 4. Benefits of Cooperative Disease Management for Growers 1 (G_I) and 2 (G_2) (\$1,000 per acre over 50 years): the Case of Rogue-and-Replant Strategies

n/a is not applicable.

^a Expectations are obtained from 1,000 simulations.

^b FTP to G_2 =Expected Payoff to G_2 (Noncooperative)–Expected Payoff to G_2 (Cooperative)+50%*Expected Aggregate Benefits to Cooperation

^c Standard deviations in parentheses.

*** Significantly different from zero at the 1% level.

Strategies									
	Expected Payoffs ^a (\$1,000)								
	Fixed								
				Aggregate	Transfer	Cooperative	Cooperative		
	Payoff	Payoff	Aggregate	Benefits to	Payment	Payoff to G_1	Payoff to G_2		
Strategies (G_1, G_2)	to G_1	to G_2	payoff	Cooperation	to G_2^{b}				
Noncooperative									
no control, no control	$214(5)^{c}$	37 (1)	251(6)	n/a	n/a	n/a	n/a		
Cooperative									
no control, 1Col	218 (5)	35 (1)	253 (6)	1 (3)***	3	215	38		
no control, 2Col	222 (5)	32 (1)	254 (6)	3 (3)***	7	215	39		
no control, 3Col	226 (5)	29 (1)	255 (6)	$4(3)^{***}$	10	216	40		
no control, 4Col	231 (5)	27 (1)	258 (6)	6 (3) ^{***}	14	217	41		
no control, 8Col	251 (5)	16(1)	267 (6)	16 (3)***	29	222	46		
no control, 10Col	264 (5)	11 (1)	275 (6)	24 (3)***	38	226	49		
no control, 12Col	279 (5)	6(1)	285 (6)	33 (3)***	48	230	54		
no control, 14Col	298 (4)	0(1)	298 (6)	47 (3)***	61	237	61		
no control, 16Col	319 (1)	- 6 (0)	313 (6)	62 (5) ^{***}	74	243	69		

Table 5. Benefits of Cooperative Disease Management (\$ per acre over 50 years): the Case of Fire-Break

 Strategies

n/a is not applicable.

^a Expectations are obtained from 1,000 simulations.

^b *FTP* to G_2 =Expected Payoff to G_2 (Noncooperative)–Expected Payoff to G_2 (Cooperative)+50%*Expected Aggregate Benefits to Cooperation.

Significantly different from zero at the 1% level.