

A PLANT-LEVEL, SPATIAL, BIOECONOMIC MODEL OF PLANT DISEASE DIFFUSION AND CONTROL: GRAPEVINE LEAFROLL DISEASE

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Grapevine leafroll disease threatens the economic sustainability of the grape and wine industry in the United States and around the world. This viral disease reduces yield, delays fruit ripening, and affects wine quality. Although there is new information on the disease spatial-dynamic diffusion, little is known about profit-maximizing control strategies. Using cellular automata, we model the disease spatial-dynamic diffusion for individual plants in a vineyard, evaluate nonspatial and spatial control strategies, and rank them based on vineyard expected net present values. Nonspatial strategies consist of roguing and replacing symptomatic grapevines. In spatial strategies, symptomatic vines are rogued and replaced, and their nonsymptomatic neighbors are virus-tested, then rogued and replaced if the test is positive. Both nonspatial and spatial classes of strategies are formulated and examined with and without considering vine age. We find that spatial strategies targeting immediate neighbors of symptomatic vines dominate nonspatial strategies, increasing the vineyard expected net present value by 18% to 19% relative to the strategy of no disease control. We also find that age-structured disease control is preferred to non-age-structured control but only for nonspatial strategies. Sensitivity analyses show that disease eradication is possible if either the disease transmission rate or the virus undetectability period is substantially reduced.

Key words: bioeconomic models, cellular automata, computational methods, disease control, grapevine leafroll disease, spatial-dynamic processes.

JEL codes: C15, C63, D24.

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 reduces yield, delays fruit ripening, and negatively affects wine quality by lowering soluble solids and increasing fruit juice acidity (Goheen and Cook 1959; Martinson et al. 2008). Its economic impact was recently estimated at \$25,000 to \$40,000 per hectare if the disease is left uncontrolled, which represents more than 75% of a vineyard's net present value (Atallah et al. 2012). GLRD is primarily introduced to vineyards through infected planting material. Once introduced, the disease can be transmitted from vine to vine by several species of mealybugs and soft-scale insects (Martelli and Boudon-Padieu 2006; Pietersen 2006; Tsai et al. 2010). Mealybugs can transmit GLRD within and across vineyards in at least three ways (Charles et al. 2009; Grasswitz and James 2008). Insects 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.

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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). However, when GLRD is already present, disease management consists mainly of minimizing the source of infection by roguing symptomatic vines after harvest, especially the young ones and replacing them with virus-tested vines (Maree et al. 2013; Rayapati, O'Neil, and Walsh 2008; Walton et al. 2009). Vector management is recommended to reduce disease transmission (Skinkis et al. 2009). 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 (Almeida et al. 2013; Cabaleiro and Segura 2006, 2007; Golino et al., 2002, 2008).

Most GLRD research has focused on studying the pathogens, with less emphasis on disease ecology and disease management (Almeida et al. 2013). This article uses information available in the GLRD disease ecology literature to develop a computational, spatial bioeconomic model that can be used to identify profit-maximizing strategies for GLRD control. Using cellular automata, we model the disease at the plant level in a spatial-dynamic way. In the simulations, the disease is introduced to an artificial vineyard through infected plant material at the time of planting. Subsequently, its diffusion follows a Markov process that is affected by each vine's location, virus detectability, age, own infection state, and infection states of its neighbors. We then use a vineyard manager's profit maximization objective function to evaluate the cost-effectiveness of disease control strategies formulated based on these vine-level characteristics. Our model contributes to the literature that uses nonspatial, compartmental models when modeling diseases by relaxing the simplifying assumptions that individuals are homogeneous in their attributes and spatially perfectly mixed.¹

¹ The perfect-mixing assumption implies that any infective individual can transmit the infection to any healthy individual with equal probability (Brauer and Castillo-Chavez 2001).

We examine the impact of alternative disease control strategies on distributions of bioeconomic outcomes and rank them based on the vineyard expected net present values (ENPVs). The results highlight the potential of vine-level, spatial strategies in reducing the economic cost of GLRD. In addition, our model can be modified to address spatial-dynamic disease diffusion and control issues in other perennial crops. We are not aware of previous work in agricultural and resource economics that formulates a spatial, plant-level, model of plant disease diffusion and control.

Literature Review

The unique characteristics of certain insect-transmitted plant diseases restrict the choice of approaches to model disease diffusion and control. The first characteristic of such diseases is that they are simultaneously driven by integrated dynamic and spatial forces, rather than by dynamic processes alone. When diseased plants are heterogeneously distributed in space and the physical environment includes spatial constraints on disease diffusion, such as a vineyard's spatial configuration, the optimality of disease control is affected not only by its intensity but also by its location.

Second, in insect-transmitted plant diseases, pesticide applications can be ineffective. This is particularly true in the case of GLRD, where insect vectors can have a short infectivity retention period,² live in crevices and underneath the bark of the grapevine (Cabaleiro and Segura 2006, 2007; Daane et al. 2012), and spread disease rapidly even if their population is kept at a low density (Charles et al. 2009; Tsai et al. 2008; Walton and Pringle 1999). Instead, insect-transmitted disease control relies mostly on reducing the source of infection by roguing (removing) infected plants and replacing them with young, healthy ones (Chan and Jeger 1994). Thus, despite the attractive features of pest control models, such as the ability to account for product quality in estimating pest control effectiveness (Babcock, Lichtenberg, and Zilberman 1992) or incorporating pest randomness in pesticide application decision

² The insect infectivity period is the time in which insect vectors retain the virus and remain infective (Tsai et al. 2008).

rules (Saphores 2000), these models are not appropriate for vector-transmitted plant diseases such as GLRD.

Plant heterogeneity is the third characteristic of certain diseases. In the case of GLRD, individual vines that are infected but nonsymptomatic are heterogeneous in the time it takes for their virus population to be detectable by virus tests (Cabaleiro and Segura 2007; Constable et al. 2012). For some of these vines, the virus may not be detected and rogued before they transmit the disease to neighboring vines, causing disease control to lag behind disease diffusion and impeding eradication. Taken together, these three characteristics call for plant-level, spatial-dynamic models of disease diffusion and control.

Spatial Bioeconomic Models

Spatial-dynamic processes have only recently been studied by economists, and the bioeconomic literature on agricultural diseases and invasive species control is mostly non-spatial (see review in Wilen 2007). Sanchirico and Wilen (1999, 2005) show that ignoring spatial processes can lead to suboptimal managerial decisions. Space can be incorporated in bioeconomic disease models by introducing barriers to disease diffusion (e.g., Brown, Lynch, and Zilberman 2002), specifying location-dependent, state-transition probabilities (e.g., Rich and Winter-Nelson 2007), or using partial differential equations (e.g., Holmes et al. 1994). In such models, spatial heterogeneity is exogenous and fixed over time (see review in Smith, Sanchirico, and Wilen 2009). In some diseases, including GLRD, however, spatial heterogeneity such as the health status of a plant's neighborhood can be endogenously determined by the diffusion process, affect disease diffusion, and be affected by the implementation of control strategies. The challenge of incorporating such spatial feedbacks into state dynamics is a common thread in resource economics and not confined to disease dynamic models (Smith, Sanchirico, and Wilen 2009). Moreover, spatial bioeconomic models often make restrictive assumptions such as linear growth and control to achieve tractability or to focus on steady state analyses in simple landscapes (see review in Epanchin-Niell and Wilen 2012). Relaxing such assumptions precludes analytical solutions and calls for numerical methods in most applications (Smith, Sanchirico, and Wilen 2009; Wilen 2007).

Bioeconomic Models of Agricultural Diseases

Research on the economics of agricultural disease control has increasingly moved toward integrated epidemiological models that incorporate feedbacks between economic and disease diffusion components within the model (Beach, Poulos, and Pattanayak 2007; Fenichel and Horan 2007; Horan and Wolf 2005). These models typically aggregate individuals into disease-state (e.g., Horan et al. 2010) or age-state (e.g., Tahvonen 2009) compartments (they are thus called compartmental models) and use differential or difference equations to represent transitions between states. They assume that the population is spatially perfectly mixed and that the individuals are homogenous in their attributes within each compartment.

These assumptions are limiting in disease modeling, especially in the case of GLRD where (a) plants are heterogeneous in virus detectability and (b) disease diffusion follows imperfect mixing processes and is shaped by vineyard spatial configuration and location of vines (Constable et al. 2012; Pietersen 2006). The homogeneity assumption of aggregate models is particularly restrictive because it precludes the formulation and testing of disease control strategies targeting individuals based on their heterogeneous, spatial-dynamic attributes. Also, the perfect-mixing assumption has been shown to underestimate the rate of spread in the early stages of a disease and to overestimate it in the later stages (Cane and McNamee 1982). These assumptions can be relaxed in difference equation models to represent distinct groups where individuals are heterogeneous by increasing the number of subpopulations or dividing the subpopulations into smaller stocks (e.g., Medlock and Galvani 2009). Depending on the level of heterogeneity desired, however, this process can lead to a combinatorial explosion in the number of state variables, equations, parameters, and data requirements (Teose et al. 2011). Moreover, in aggregate bioeconomic models of diseases, transmission rates are imposed on individuals exogenously depending on membership in a specific subpopulation. In reality, however, these rates are determined in a spatial-dynamic fashion as a result of the spatial-dynamic feedbacks between disease diffusion and disease control.

Table 1. Overview of the Modeling Process

	Modeling Step	Tool
1.	<i>Formal model.</i> Define bioeconomic model.	None
2.	<i>Computational model.</i>	
2a.	<i>Model specification.</i> - Specify cellular automata model by defining: o Space and time o States and state transitions - Define model parameters and variables.	Java, AnyLogic
2b.	<i>Model verification.</i> - Conduct simulation and collect simulated data. - Debug to ensure consistency in model behavior between computational model and formal model.	Java, AnyLogic
2c.	<i>Model calibration</i> Define optimization experiment that aims to find the optimal transmission parameter values using field data from the literature.	OptQuest, AnyLogic
2d.	<i>Model validation</i> Validate calibrated model by testing that the expected time to 50% disease prevalence (expected half-life) and expected time to 100% disease prevalence measures fall within intervals reported in the literature.	Java, AnyLogic
3.	<i>Simulation experiments</i> Define and conduct Monte Carlo experiments: scenarios of “no disease,” “no disease control,” eight nonspatial and ten spatial disease control strategies.	Java, AnyLogic
4.	<i>Statistical analyses</i> Conduct statistical tests on the differences between expected net present values.	Stata
5.	<i>Sensitivity analyses</i> Repeat steps 3 and 4 for each parameter considered in the sensitivity analysis.	

Cellular Automata Models

With dramatic decreases in computational costs, cellular automata and agent-based models have emerged as a preferred methodological framework to study complex systems (Miller and Page 2007) such as diseases. Cellular automata are dynamic models that operate in discrete space and time on a uniform and regular lattice of cells. Each cell is in one of a finite number of states that get updated according to mathematical functions and algorithms that constitute state transition rules. At each time step, a cell computes its new state given its own old state and the old state of its neighborhood according to the transition rules (Tseftis and Judd 2006; Wolfram 1986). The spatial-dynamic structure is especially relevant when modeling processes that face physical constraints (Gilbert and Terna 2000) such as boundaries and geometry, as in the case of managed agricultural systems. In contrast with compartmental models, cellular

automata and agent-based models do not aggregate individuals in compartments, thus allowing each individual to be heterogeneous in any finite number of attributes (Rahman-dad and Serman 2008). Although cellular automata models have been extensively used to model spatial-dynamic processes (e.g., Sun et al. 2010; Yassemi, Dragičević, and Schmidt 2008), their use in the agricultural economics literature has been rare. The few examples include one application to foot-and-mouth disease control (Rich, Winter-Nelson, and Brozović 2005) and land use change studies (Balmann 1997; Kaye-Blake et al. 2009; Marshall and Homans 2004; Roth et al. 2009).

We contribute to the disease control bioeconomic literature by using cellular automata to offer a model that is inherently spatial and dynamic. We formally define the bioeconomic model, then build the computational model, verify its behavior, calibrate it, and validate it using GLRD disease ecology literature field data. Using simulation experiments, we generate distributions of bioeconomic

Table 2. Vine Revenue

Age ($a_{i,j,t}$) and Infection ($s_{i,j,t}$) States	Yield Reduction (%) ^a	Quality Penalty (%) ^b	Vine Revenue (\$/vine/month) ^c
$a_{i,j,t} < 36$ months	N/A	N/A	0.00
$a_{i,j,t} \geq 48$ months and H	0	0	0.43
$a_{i,j,t} \geq 48$ months and E_u or E_d	30	10	0.27
$a_{i,j,t} \geq 48$ months and I_m	50	10	0.19
$a_{i,j,t} \geq 48$ months and I_h	75	10	0.09

Note: N/A indicates not applicable (a vine is not productive before the age of 36 months).

^aGoheen and Cook (1959); Martinson et al. (2008)

^bAtallah et al. 2012.

^cVine revenue calculations are based on the Cabernet franc grape yield of 3.3 tons per acre, per year (White 2008), a planting density of 1,096 vines per acre (Wolf 2008), and a grape price of \$1,700/ton (White 2008).

outcomes for the scenario of no disease, the strategy of no disease control, and eighteen alternative nonspatial and spatial disease control strategies. We then conduct statistical analyses to rank the ENPVs generated in each experiment and find the optimal disease control strategy. Finally, we conduct sensitivity analyses to key bioeconomic model parameters. We synthesize our modeling process in table 1.

Bioeconomic Model

The spatial geometry of disease diffusion is represented by a two-dimensional grid G representing a vineyard plot. G is the set of $I \times J$ cells where I and J are the number of rows and columns, respectively. In our model, there are 5,720 cells $(i, j) \in G$, each holding one grapevine. Vineyard rows are oriented north to south with $I = 44$ vines per grid row and $J = 130$ vines per grid column resulting in a vineyard area of approximately 5.2 acres.³

Each cell (i, j) has an age state and an infection state. Time t progresses in discrete monthly steps up to 600 months. $\mathbf{a}_{i,j,t}$ is a $600 \times I$ vector holding a I for a vine’s age in months and zeros for the other possible ages. A vine can be *Infective* or *Noninfective*. *Infective* (I) and *Noninfective* (NI) vines differ by whether or not they exhibit symptoms and have the ability to transmit the infection to their neighbors. *Noninfective* vines, in turn, can be in the following infection states: *Healthy* (H), *Exposed-undetectable*

(E_u), and *Exposed-detectable* (E_d). Subdividing the *Noninfective* states allows us to separate healthy vines from those that have been exposed to the virus and have therefore lower grape yield and quality. The distinction between the states *Exposed-undetectable* (E_u) and *Exposed-detectable* (E_d) is important to separate the vines whose virus populations have not reached detectable levels (*Exposed-undetectable*), from those with virus populations high enough to be detectable through a virus test (*Exposed-detectable*). *Infective* vines, for their part, can exhibit two states, namely *Infective-moderate* (I_m) or *Infective-high* (I_h). Separating the two states allows us to model the decrease in vine economic value as GLRD symptoms severity increases over time from the moderate to the high level. $\mathbf{s}_{i,j,t}$ is the infection state vector at time t of dimension $5 \times I$. The vector holds a I for the state that describes a vine’s infection state and zeros for the remaining four states. $\mathbf{w}_{i,j,t}$ is an age-infection composite state defined as the combination of a vine’s age state $\mathbf{a}_{i,j,t}$ and its infection state $\mathbf{s}_{i,j,t}$.

A vine’s infection and age states map into a third dynamic state variable, its economic value, or per-vine revenue $r(\mathbf{w}_{i,j,t})$. Per-vine revenue equals zero if the vine’s age $\mathbf{a}_{i,j,t}$ is below τ_{max} . Beyond that age, $r(\mathbf{w}_{i,j,t})$ is a function of the vine infection state. Grapes from GLRD-affected vines are subject to a penalty imposed on the price paid for grapes harvested from healthy vines. Furthermore, GLRD reduces grapevine yield by 30%, 50%, and 75% for vines in states *Exposed* (both *Exposed-undetectable* and *Exposed-detectable*), *Infective-moderate*, and *Infective-high*, respectively (table 2). A vine’s revenue is known to a vineyard manager at time t . Nevertheless, the per-vine revenue

³ This configuration is considered representative of a typical vineyard in the Northeastern United States (Wolf 2008). The represented vineyard dimensions are 350’ x 650’ with an area of 227,500 ft² or 5.22 acres. Vine and column spacing are 5 and 8 feet, respectively.

is random for periods beyond t because it depends on the vine's infection state $s_{i,j,t}$.

Given each vine's state $s_{i,j,t}$ and an infection state transition matrix \mathbf{P} , its expected infection state $E(s_{i,j,t+1})$ at time $t + 1$ is computed according to the following infection-state transition equation:

$$(1) \quad E(s_{i,j,t+1}) = \mathbf{P}^T s_{i,j,t}$$

where E is the expectation operator and \mathbf{P}^T is the transpose of matrix \mathbf{P} . $E(s_{i,j,t+1})$ is a $5 \times I$ vector with a probability of staying in the current state, a probability of transitioning to the next state, and zeros elsewhere.

Disease diffusion is spatially constrained by the vineyard's horizontal (equation 2a) and vertical boundaries (equation 2b) as follows:⁴

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

$$(i + 1) \in \{2, \dots, I\};$$

$$(2b) \quad (j - 1) \in \{1, \dots, J - 1\};$$

$$(j + 1) \in \{1, \dots, J - 1\}.$$

We now describe how the infection state transition probability matrix \mathbf{P} governs disease diffusion. Vines in state *Healthy* (H) are susceptible to infection. They get exposed to the virus with a neighborhood-dependent probability b . At this point, they enter a latency period during which they are non-symptomatic and noninfective. At first, the virus population in the vine is below levels that can be detected by virus tests and the vine is in state *Exposed-undetectable* (E_u). The virus population reaches detectable levels with probability c (i.e., the vine transitions to state *Exposed-detectable*, E_d). The transition to state *Infective-moderate* (I_m) happens with a probability d 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 moderate severity state first (I_m) and transition to a state of high severity later, or *Infective-high* (I_h), with a probability

f . Mathematically, \mathbf{P} can be expressed as follows:⁵

$$(3) \quad \mathbf{P} = \begin{bmatrix} (1 - b) & b & 0 & 0 & 0 \\ 0 & 1 - c & c & 0 & 0 \\ 0 & 0 & (1 - d) & d & 0 \\ 0 & 0 & 0 & (1 - f) & f \\ 0 & 0 & 0 & 0 & 1 \end{bmatrix}.$$

In equation (3), b is the *Healthy to Exposed-undetectable* transition probability conditional on previous own, and neighborhood infection states. It can be expressed as

$$(4) \quad b = \Pr(s_{i,j,t+1} = E_u | s_{i,j,t} = H)$$

$$= \begin{cases} 0 & \text{if } s_{N_{i,j,t}} = (NI, NI, NI, NI) \\ 1 - e^{-\beta} & \text{if } s_{N_{i,j,t}} = (NI, NI, I, NI) \\ 1 - e^{-\beta} & \text{if } s_{N_{i,j,t}} = (NI, NI, NI, I) \\ 1 - e^{-2\beta} & \text{if } s_{N_{i,j,t}} = (NI, NI, I, I) \\ 1 - e^{-\alpha} & \text{if } s_{N_{i,j,t}} = (I, NI, NI, NI) \\ 1 - e^{-(\alpha+\beta)} & \text{if } s_{N_{i,j,t}} = (I, NI, I, NI) \\ 1 - e^{-(\alpha+\beta)} & \text{if } s_{N_{i,j,t}} = (I, NI, NI, I) \\ 1 - e^{-(\alpha+2\beta)} & \text{if } s_{N_{i,j,t}} = (I, NI, I, I) \\ 1 - e^{-\alpha} & \text{if } s_{N_{i,j,t}} = (NI, I, NI, NI) \\ 1 - e^{-(\alpha+\beta)} & \text{if } s_{N_{i,j,t}} = (NI, I, I, NI) \\ 1 - e^{-(\alpha+\beta)} & \text{if } s_{N_{i,j,t}} = (NI, I, NI, I) \\ 1 - e^{-(\alpha+2\beta)} & \text{if } s_{N_{i,j,t}} = (NI, I, I, I) \\ 1 - e^{-2\alpha} & \text{if } s_{N_{i,j,t}} = (I, I, NI, NI) \\ 1 - e^{-(2\alpha+\beta)} & \text{if } s_{N_{i,j,t}} = (I, I, I, NI) \\ 1 - e^{-(2\alpha+\beta)} & \text{if } s_{N_{i,j,t}} = (I, I, NI, I) \\ 1 - e^{-(2\alpha+2\beta)} & \text{if } s_{N_{i,j,t}} = (I, I, I, I) \end{cases}.$$

In equation (4), $s_{N_{i,j,t}}$ is the infectivity state of a vine's von Neumann neighborhood.⁶ For example, $s_{N_{i,j,t}} = (I, I, I, NI)$ is the state of a neighborhood composed of two *Infective* (I) neighbors in the same column (within-column neighbors), one *Infective* neighbor in the adjacent column (across-column neighbor), and one *Noninfective* (NI) neighbor in the other adjacent column (across-column neighbor). Given that each of the four neighbors can be in one of two infectivity states (I or NI), $s_{N_{i,j,t}}$ can be in one of the 2^4 states listed in equation (4). Parameters α and β are the within-column and across-column

⁴ These spatial constraints are formulated by defining the set of indices that vine (i, j) 's within-column (equation 2a) and across-column neighbors (equation 2b) can have. They ensure that the disease does not spread beyond the vines situated at the borders of the vineyard.

⁵ \mathbf{P} reads from row (states H, E_u, E_d, I_m , and I_h at time t) to column (states H, E_u, E_d, I_m , and I_h at time $t + 1$).

⁶ This type of neighborhood represents the most common vertical trellis system where a vine is in contact with its four neighbors in the cardinal directions. In contrast, a horizontal trellis system favors contact with up to eight neighbors (Cabaleiro and Segura 2006) and could be represented by a Moore neighborhood.

transmission rates with $\alpha > \beta > 0$, suggesting that *Infective* vines transmit the disease to their neighbors within the grid column at a higher rate than they transmit it to their neighbors situated in the adjacent grid column. We choose this neighborhood-based infection state transition to reflect patterns of GLRD diffusion observed in spatial analyses where the disease is shown to spread preferentially along grid columns (Habibi et al. 1995; Le Maguet et al. 2013). We assume that within- and across-column infections occur independently with rate parameters α and β . That is, the time a vine stays in the *Healthy* state before transitioning to the *Exposed-undetectable* state is a random variable, exponentially distributed, with rate α for within-column state transitions and rate β for across-column state transitions. In each time step, a random variable u_t determines whether the *Healthy* to *Exposed-undetectable* state transition happens or not. A *Healthy* vine that has one within-column *Infective* neighbor (e.g., $s_{N_{i,j,t}} = I, NI, NI, NI$) receives the infection at time $t + 1$ if $u_t < 1 - e^{-\alpha}$, where u_t is a random draw from $U \sim (0, 1)$. Conversely, the disease is not transmitted if $u_t \geq 1 - e^{-\alpha}$. Similarly, a *Healthy* vine that has one across-column *Infective* neighbor (e.g., $s_{N_{i,j,t}} = NI, NI, I, NI$) receives the infection at time $t + 1$ if $u_t < 1 - e^{-\beta}$ and is not transmitted if $u_t \geq 1 - e^{-\beta}$. When two or more transmission types are realized—for example, when a vine has one *Infective* within-column neighbor and one *Infective* across-column neighbor—the disease transmission is determined by the shortest of the waiting times (Cox 1959).⁷

The probability of transition from *Exposed-undetectable* (E_u) to *Exposed-detectable* (E_d) is given by c as follows:

$$(5) \quad c = \Pr(X < x) = \begin{cases} 0 & \text{if } x < m_1 \\ \frac{(x - m_1)^2}{(m_2 - m_1)(m_3 - m_1)} & \text{if } m_1 \leq x \leq m_3 \\ \left(1 - \frac{(m_2 - x)^2}{(m_2 - m_1)(m_2 - m_3)}\right) & \text{if } m_3 \leq x < m_2 \\ 1 & \text{if } x > m_2 \end{cases}$$

⁷ We compare the random variable with the transition probability in each time step because the state of a vine’s neighborhood is changing over time, thus changing the probability that a vine receives the infection in each time interval.

This transition happens after a vine has spent a period X in state *Exposed-undetectable*. Cabaleiro and Segura (2007) and Constable et al. (2012) report minimum (m_1), maximum (m_2), and most common (m_3) values for this period. With no further knowledge on the distribution of this period, we assume it is drawn from a triangular distribution with parameters m_1, m_2 , and m_3 . The probability that the transition happens in less than x time steps, or $\Pr(X < x)$, can then be written as a function of these parameters (Kotz and van Dorp 2004).

The *Exposed-detectable* to *Infective* state transition probability is given by d as follows:

$$(6) \quad d = \Pr(s_{i,j,t+1} = I_m | s_{i,j,t} = E_d) = \begin{cases} 1 - e^{-1/L_y} & \text{if } a_{i,j,t} = \text{Young} \\ 1 - e^{-1/L_m} & \text{if } a_{i,j,t} = \text{Mature} \\ 1 - e^{-1/L_o} & \text{if } a_{i,j,t} = \text{Old} \end{cases}$$

This probability depends on a vine’s age category. Younger vines have shorter latency periods (Pietersen 2006), i.e. $L_y < L_m < L_o$, where subscripts y, m , and o denote young (0–5 years), mature (6–20 years), and old (>20 years) vines, respectively. The waiting time after a vine enters state *Exposed-detectable* (E_d) and before it transitions to state *Infective moderate* (I_m) is a random variable, exponentially distributed with fixed rate parameter $1/L_y$ for young vines, $1/L_m$ for mature vines, and $1/L_o$ for old vines.

Finally, once a vine is in state *Infective moderate* (I_m), symptom severity increases over time and reaches a high level after a period *Inf*. That is, the waiting time until a vine transitions from *Infective-moderate* (I_m) to *Infective-high* (I_h) is a random variable, exponentially distributed with fixed rate parameter $1/Inf$. Thus, the probability that a vine transitions from I_m to I_h is $f = \Pr(s_{i,j,t+1} = I_h | s_{i,j,t} = I_m) = 1 - e^{-1/Inf}$.⁸ Symbols, definitions, values, and references for the model parameters are presented in table 3.

The objective of a risk-neutral vineyard manager is to maximize the vineyard ENPV by choosing an optimal disease control strategy from a discrete set of spatial and

⁸ Note that we do not need to compare a random variable with the probabilities at each time interval for the E_u -to- E_d , E_d -to- I_m , and I_m -to- I_h transitions because the rates and waiting times in these transitions do not depend on the neighborhood state and are therefore fixed.

Table 3. Model Parameters

Parameter	Description	Value	Unit	Sources
α	Within-column H to E_u transition rate	4.2 ^a	month ⁻¹	Calibration experiment based on Charles et al. (2009).
β	Across-column H to E_u transition rate	0.014 ^a	month ⁻¹	
L_y	Latency period for young vines	24	months	Age-specific latency periods based on Jooste, Pietersen, and Burger (2011).
L_m	Latency period for mature vines	48	months	
L_o	Latency period for old vines	72	months	
m_1	Minimum of undetectability period	4	months	Cabaleiro and Segura (2007); Constable et al. (2012).
m_2	Maximum of undetectability period	18	months	
m_3	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.
τ_{max}	Period from planting until productivity	36	months	White (2008).
ρ	Discount factor	0.9959	month ⁻¹	Assumed. Equivalent to an annual discount rate of 5%.
$c_{u_{ij}}$	Unit cost of vine roguing (removal) and replacement	7.25	\$/vine	Based on White (2008) and Atallah et al. (2012).
$c_{v_{ij}}$	Unit cost of vine virus testing	2.61	\$/vine	AC Diagnostics (2012) for the material cost based on 1,000 samples; Luminex (2010) for the labor time.

^aTransition 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.

nonspatial strategies, \mathcal{W} . Strategies are based on vines' age-infection states. Each strategy translates into two binary decisions for each vine in cell (i, j) in each time step. The first vine-level decision a manager faces is whether to rogue a vine and replace it with a new, virus-free vine⁹ ($u_{w_{ij,t}} = 1$ if roguing and replanting takes place, 0 otherwise). The other decision is whether to test for the

that allocates disease control effort over space and time so as to yield the maximum ENPV improvement over the strategy of no disease control. Letting E be the expectation operator over the random vine-level revenue $r(w_{ij,t})$ and ρ^t the discount factor¹⁰ at time t (in months) where $t \in \{0, 1, 2, \dots, 600\}$, the objective of a vineyard manager is to maximize the ENPV as follows:

$$(7) \quad \max_w E \sum_{t \in T} \rho^t \sum_{(i,j) \in G} \left\{ \left(1 - \sum_{\tau=0}^{\tau_{max}} u_{w_{ij,t-\tau}} \right) r(w_{ij,t}) - (u_{w_{ij,t}} c_{u_{ij}} + v_{w_{ij,t}} c_{v_{ij}}) \right\}$$

virus ($v_{w_{ij,t}} = 1$ if virus testing takes place, 0 otherwise).

The optimal strategy \mathcal{W}^* is the sequence of vine-level control variables $\{u_{w_{ij,t}}, v_{w_{ij,t}}\}$

subject to the infection state transition equation (equation 1) and the spatial constraints to disease diffusion (equations 2a and 2b). Note that the objective function not only takes into account the total amount of control realized under each strategy but also

⁹ We assume that the vineyard manager follows industry recommendations and uses virus-tested vines when replacing an infected vine. We also assume that virus-tested vines are virus-free.

¹⁰ $\rho^t = \frac{1}{(1+r)^t}$, where r is the discount rate.

the timing, intensity, and location of that control. The first expression in the curly brackets of equation (7) represents the revenue of a vine in location (i, j) and in age-infection state $w_{i,j,t}$ at time t . If a manager has decided to rogue and replant vine (i, j) in the last τ_{max} periods, then $u_{w_{i,j,t-\tau}}$ is equal to 1 and the revenue is pre-multiplied by zero for a period of τ_{max} until the replant bears fruit, where $\tau \in \{1, 2, \dots, \tau_{max}\}$, and $\sum_{\tau=0}^{\tau_{max}} u_{w_{i,j,t-\tau}} = \{0, 1\}$.¹¹ The second expression in the curly brackets represents the cost of roguing and replanting ($c_{u_{ij}}$) and the cost of testing ($c_{v_{ij}}$), premultiplied by their corresponding binary decision variables.

Model Initialization

At the beginning of a simulation, 2% of the grapevines (including those situated at the border of the vineyard) are randomly chosen from a uniform spatial distribution $U(0, 5720)$ to transition from the *Healthy* to the *Exposed* state. This reflects findings in GLRD studies indicating that primary infection sources are randomly spatially distributed (Cabaleiro et al. 2008) and that initial disease prevalence is typically between 1% and 5% (Atallah et al. 2012). Thereafter, GLRD spreads to uninfected vines.

Model calibration and parameterization

To select the disease transmission parameter values (α and β in table 3), we first define a calibration objective function that minimizes the difference between the total number of infected vines over time obtained from our computational model (under no disease control) and the total number of infected vines over time from temporal disease progress data in Charles et al. (2009). Next, we use an optimization engine (OptQuest) that varies the values of α and β in each of the Monte Carlo simulations according to an algorithm combining Tabu search,¹² scatter search, integer programming, and neural networks to find the optimal parameter values (step 2c in table 1). We finally validate that the expected time until 50% disease prevalence (vineyard half-life) and expected time to 100% disease

prevalence measures from the calibrated model fall within ranges of temporal disease diffusion curves reported in the GLRD literature (Cabaleiro and Segura 2006; Cabaleiro et al. 2008) (step 2d in table 1). For the other parameters, we choose values reported in the literature after consultation with GLRD experts (table 3).

We choose a monthly time step because it gives the disease diffusion model a temporal resolution that is consistent with the magnitude of the disease diffusion parameters. With no information on diffusion seasonality, we do not model seasons and assume for simplicity that disease diffusion and control can take place in any month.¹³

Experimental Design

We design and implement Monte Carlo experiments to evaluate nonspatial and spatial disease control strategies by comparing their bioeconomic outcomes to those resulting from a strategy of no disease control. Each experiment consists of a set of 1,000 simulation runs, over 600 months, on a vineyard of 5,720 grapevines. Experiments differ in the disease control strategy they use. Outcome realizations for a run within an experiment differ because of random spatial initialization and random spatial disease diffusion. Data collected over simulation runs are the expected values of the bioeconomic outcomes under each strategy (step 3 in table 1). Finally, we conduct statistical tests to rank the disease control strategies and find the optimal strategy (step 4 in table 1). The model is written in Java and simulated using the software AnyLogic (XJ Technologies). Software documentation and Java code are available in a supplementary appendix online.

Disease Control Strategies

The discrete set of disease control strategies, \mathcal{W} , includes eighteen spatial and nonspatial strategies, in addition to the strategy of no disease control. In the subset of *nonspatial strategies*, the vineyard manager decides whether to rogue and replace symptomatic vines based on their symptoms (*Infective-moderate*; *Infective-high*) with or without

¹¹ 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.

¹² Tabu search is a metaheuristic procedure for solving optimization problems designed to guide other methods to avoid the trap of local optimality.

¹³ Note that, although simplifying, this assumption is consistent with the fact that, in reality, both disease diffusion and disease control take place during the same months (the growing season).

Table 4. Disease Control Strategies: Definitions and Acronyms

Disease Control Strategies		Acronyms
<i>Nonspatial strategies</i> ^a		
1	Roguing and replacing all vines that are <i>Infective</i>	I
2	Roguing and replacing all vines that are <i>Infective-moderate</i>	I_m
3	Roguing and replacing all vines that are <i>Infective-high</i>	I_h
4	Roguing and replacing vines that are <i>Infective-moderate</i> and <i>Young</i>	I_mY
5	Roguing and replacing vines that are <i>Infective-moderate</i> and <i>Mature</i>	I_mM
6	Roguing and replacing vines that are <i>Infective-moderate</i> and <i>Old</i>	I_mO
7	Roguing and replacing vines that are <i>Infective-high</i> and <i>Mature</i>	I_hM
8	Roguing and replacing vines that are <i>Infective-high</i> and <i>Old</i>	I_hO
<i>Spatial strategies</i> ^b		
9	Roguing and replacing <i>Infective-moderate</i> vines in addition to testing their two within-column neighbors, then roguing and replacing those that test positive	I_mNS
10	Roguing and replacing <i>Infective-moderate</i> vines in addition to testing their two across-column neighbors and two-within column neighbors, then roguing and replacing those that test positive	I_mNSEW
11	Roguing and replacing <i>Infective-moderate</i> vines in addition to testing their four within-column neighbors and two across-column neighbors, then roguing and replacing those that test positive	I_mNS2EW
12	Roguing and replacing <i>Infective-moderate</i> vines in addition to testing their four within-column and four within-row neighbors, then roguing and replacing those that test positive	$I_mNS2EW2$
13.	Roguing and replacing <i>Young, Infective-moderate</i> vines in addition to testing their two within-column neighbors, then roguing and replacing those that test positive	I_mY-NS
14.	Roguing and replacing <i>Mature, Infective-moderate</i> vines in addition to testing their two within-column neighbors, then roguing and replacing those that test positive	I_mM-NS
15.	Roguing and replacing <i>Old, Infective-moderate</i> vines in addition to testing their two within-column neighbors, then roguing and replacing those that test positive	I_mO-NS
16.	Roguing and replacing <i>Young, Infective-moderate</i> vines in addition to testing their two across-column neighbors and two-within column neighbors, then roguing and replacing those that test positive	$I_mY-NSEW$
17.	Roguing and replacing <i>Mature, Infective-moderate</i> vines in addition to testing their two across-column neighbors and two-within column neighbors, then roguing and testing those that test positive	$I_mM-NSEW$
18.	Roguing and replacing <i>Old, Infective-moderate</i> vines in addition to testing their two across-column neighbors and two-within column neighbors, then roguing and replacing those that test positive	$I_mO-NSEW$

^aNote that strategies 4 to 8 are the age-structured counterparts of strategies 2 and 3.

^bNote that strategies 13 to 18 are the age-structured counterparts of strategies 9 and 10.

considering their age (*Young*: 0–5 years; *Mature*: 6–20 years; *Old*: >20 years). There are eight nonspatial strategies (strategy 1 to strategy 8 in table 4).¹⁴ In the subset of *spatial strategies*, the vineyard manager decides whether to rogue and replant vines as soon as they develop symptoms (*Infective-moderate*), then test their neighbors and rogue and replace them if they test positive.

¹⁴ 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 five years to transition to the *Infective-high* state.

Figure 1 shows the four types of neighborhoods considered in disease control (panels a, b, c, and d). If a vine tests positive, it is removed in the same period. There are ten spatial strategies (strategy 9 to strategy 18 in table 4). The scenario of no disease provides a baseline to compute the expected disease economic cost under each candidate strategy.

Bioeconomic Outcomes Measured and Ranking of Control Strategies

To find the optimal disease control, we use the objective function (equation 7) to rank

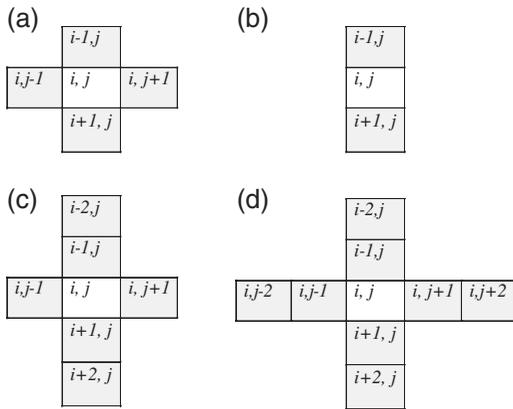


Figure 1. Types of grapevine neighborhood

the vineyard net present values under the alternative strategies. In addition, we collect simulated data on the expected total disease control costs, the expected total number of grapevines rogued and replaced, the expected average vineyard age, and the expected half-life. The latter is defined as the expected time period to reach 50% disease prevalence. It is a measure of the ability of a control strategy to slow down disease diffusion.

Results and Discussion

Overall, we find that spatial strategies are superior to nonspatial strategies. In fact, none of the nonspatial strategies improve the ENPV over the strategy of no disease control under the base model parameter values. We also find that age-structured disease control improves a vineyard ENPV compared with non-age-structured control. However, such improvements are not present with spatial strategies. Among nonspatial strategies, targeting young, moderately infected vines (strategy I_mY) yields the highest vineyard ENPV. The spatial strategy of targeting symptomatic vines and their four immediate neighbors (strategy I_mNSEW) is optimal, maximizing the vineyard ENPV.

Nonspatial Strategies

Our simulations indicate that the vineyard’s ENPVs over a 50-year period are higher when nonspatial disease control strategies are structured by age (I_mY , I_mM , I_mO , I_hM , I_hO) compared with the strategies that are not (I_m , I_h , I) (table 5). Structuring strategies by vine age increases revenues by reducing

the number of unproductive replants and the disease control costs compared with non-age-structured strategies. The strategy of targeting young, moderately infected vines (strategy I_mY) yields the highest ENPV. This result is significant because a vineyard manager might be inclined to wait until a productive vine is more severely infected and older before roguing and replacing it. Doing so, however, causes roguing and replanting to occur too late and less frequently, thus reducing the vineyard ENPV.

Under the base model parameter values, none of the nonspatial strategies yield a positive ENPV improvement over the strategy of no disease control. This finding suggests that current industry recommendations for roguing and replanting all symptomatic vines might not be ENPV-improving if the within-column disease transmission is high enough. Our result depends critically on the baseline value of the within-column transmission parameter α . We decrease this value to find a threshold that renders the best nonspatial strategy I_mY (targeting young, moderately infected vines) ENPV-improving. We find that, when α equals 1.169, strategy I_mY yields an ENPV that is 3% higher than the ENPV of no disease control. This improvement is statistically significant at the 1% level.

Spatial Strategies

Among the non-age-structured spatial strategies (strategies 9 through 12), the one that targets symptomatic vines and their four immediate neighbors (strategy I_mNSEW) yields the highest ENPV improvement over the strategy of no disease control (\$59,000 or 19%; table 5). This improvement is statistically significant at the 1% level. This strategy reduces the vineyard-level disease cost to \$88,000 over 50 years, which is smaller than the cost of \$147,000 when the disease is not controlled and the cost of \$148,000 when the best nonspatial strategy is used (strategy I_mY). The second-best strategy is I_mNS , which targets symptomatic vines and their two immediate neighbors. This strategy yields an ENPV improvement of \$58,000 and reduces the disease cost to \$89,000. This improvement is statistically significant at the 1% level. These results underscore the benefits of spatial disease control strategies in comparison with nonspatial strategies. Spatial strategies I_mNS and I_mNSEW increase the

Table 5. Disease Control Strategies: Expected Net Present Value Improvements

Disease Control Strategies	Acronym	Expected Net Present Values ^a				
		Value \$1,000	Disease Cost ^b \$1,000	Improvement over no disease control ^c		
				\$1,000	%	
No disease		463 (0) ^d	N/A	N/A	N/A	
No disease control		316 (4)	147	N/A	N/A	
<i>Nonspatial strategies</i>						
1	All <i>Infective</i>	<i>I</i>	-147 (16)	610	-463***	-147
2	<i>Infective-moderate</i>	<i>I_m</i>	-146 (16)	609	-462***	-146
3	<i>Infective-high</i>	<i>I_h</i>	284 (4)	179	-32***	-10
4	<i>Infective-moderate</i> and <i>Young</i>	<i>I_mY</i>	314 (3)	148	-1***	0
5	<i>Infective-moderate</i> and <i>Mature</i>	<i>I_mM</i>	309 (4)	154	-7***	-2
6	<i>Infective-moderate</i> and <i>Old</i>	<i>I_mO</i>	308 (4)	155	-8***	-2
7	<i>Infective-high</i> and <i>Mature</i>	<i>I_hM</i>	295 (4)	168	-21***	-7
8	<i>Infective-high</i> and <i>Old</i>	<i>I_hO</i>	310 (4)	153	-6***	-2
<i>Spatial strategies</i>						
9	Two within-column neighbors of <i>Infective-moderate</i> vines	<i>I_mNS</i>	374 (6)	89	58***	18
10	Two within- and two across-column neighbors of <i>Infective-moderate</i> vines	<i>I_mNSEW</i>	374 (5)	88	59***	19
11	Four within- and two across-column neighbors of <i>Infective-moderate</i> vines	<i>I_mNS2EW</i>	309 (4)	154	-7***	-2
12	Four within- and four across-column neighbors of <i>Infective-moderate</i> vines	<i>I_mNS2EW2</i>	295 (5)	168	-21***	-7
13.	Two within-column neighbors of <i>Young, Infective-moderate</i> vines	<i>I_mY-NS</i>	215 (5)	248	-101***	-32
14.	Two within-column neighbors of <i>Mature, Infective-moderate</i> vines	<i>I_mM-NS</i>	316 (5)	147	0.1***	0
15.	Two within-column neighbors of <i>Old, Infective-moderate</i> vines	<i>I_mO-NS</i>	303 (4)	159	-13***	-4
16.	Two within- and two across-column neighbors of <i>Young, Infective-moderate</i> vines	<i>I_mY-NSEW</i>	-59 (14)	521	-374***	-119
17.	Two within- and two across-column neighbors of <i>Mature, Infective-moderate</i> vines	<i>I_mM-NSEW</i>	317 (4)	146	1***	0
18.	Two within- and two across-column neighbors of <i>Old, Infective-moderate</i> vines	<i>I_mO-NSEW</i>	302 (4)	161	-14***	-4

Note: N/A is not applicable.

^aExpectations are obtained from 1,000 simulations for the 5-acre vineyard.

^bExpected Disease Cost = ENPV (no disease) - ENPV (strategy).

^cENPV improvement = ENPV (strategy) - ENPV (no disease control).

^dStandard deviations in parentheses.

***Difference is significant at the 1% level.

vineyard ENPV through early detection and control of nonsymptomatic grapevines situated in the neighborhood of a symptomatic grapevine. Consequently, the *Infected-high* state is never reached, and the highest yield reduction (75%; table 2, *I_h*) is never realized. At the terminal period, disease prevalence

is 13% under strategy *I_mNS* and 4% under strategy *I_mNSEW*.

The disease economic cost under the optimal strategy *I_mNSEW* is approximately \$25,000 per hectare by year 25 and contrasts with previous GLRD estimates of approximately \$8,000 per hectare over 20 years

(Nimmo-Bell 2006) and \$7,000 per hectare over 25 years (Atallah et al. 2012). These studies assume that a strategy consisting of roguing and replanting all symptomatic vines is capable of reducing the disease to a prevalence of 1% (Atallah et al. 2012) or eradicating it (Nimmo-Bell 2006). Such assumptions are valid if all infected vines are symptomatic and can therefore be rogued and replaced. If we set the undetectability and latency periods to zero to replicate such an assumption, the expected disease economic costs are approximately \$4,000 per hectare over 25 years when all symptomatic vines are rogued and replaced (strategy I).

We find that expanding the search for *Exposed* vines beyond the immediate neighbors (Figure 1, panels c and d) leads to higher disease control costs and is not economical. For instance, strategy I_mNS2EW worsens the ENPV improvement relative to the strategy of no disease control (table 5). The estimated ENPVs become even more negative if disease search includes two additional across-column neighbors (strategy $I_mNS2EW2$; table 5). Interestingly, strategies featuring a search for *Exposed* vines beyond immediate neighbors (strategy I_mNS2EW and strategy $I_mNS2EW2$) yield lower expected half-life measures (i.e., they speed rather than delay disease diffusion) compared with the strategies testing only immediate neighbors (strategy I_mNS and strategy I_mNSEW). Although the identification of a larger amount of infected, nonsymptomatic vines (*Exposed*) and their removal before they become *Infective* slows disease diffusion, beyond a certain level of roguing and replanting, the effect is reversed. Grapevine roguing and replanting implies replacing infected grapevines with younger healthy ones that have shorter latency periods. Once replants get infected, they become *infective* in a relatively short period and contribute to further disease diffusion.¹⁵

Among age-structured spatial strategies (strategies 13 through 18), only those targeting mature vines (strategy I_mM-NS

and strategy $I_mM-NSEW$) yield positive ENPV improvements over the strategy of no disease control (table 5). Note that age-structured spatial control strategies (strategies 13 through 18) perform worse than their non-age-structured (strategies 9 and 10) counterparts. In contrast with non-spatial scenarios, structuring strategies by age in spatial scenarios reduces total revenues relative to non-age-structured strategies. This relative decrease in revenues is caused by the disease diffusion generated by those *Exposed* vines left undetected because they do not neighbor *Infective-moderate* vines in the targeted age category. Note that the channels through which structuring strategies by age affects the revenues are different in non-spatial and spatial strategies. In the non-spatial scenarios, revenue increases stem from reductions in the number of unproductive replants. In spatial scenarios, structuring disease control by age decreases revenues by lowering the level of early detection.

Disease Management Insights

Our results offer new insights to vineyard managers. Alternative disease control strategies yield different results through their different allocation of disease control effort over time and space. A manager deciding when and where to control GLRD (i.e., what age, infection, and location states to target) faces tradeoffs between the ecological benefits and drawbacks of controlling earlier, more frequently, and in a more extended neighborhood. Two types of costs incentivize vineyard managers to postpone roguing depending on their discount rates. One is the expenses incurred in testing, roguing, and replacing vines, and the other is the opportunity cost of roguing an infected but still-productive vine. The latter cost consists of the forgone revenues during the period replants are unproductive. Postponing those costs has to be balanced with two types of ensuing damages—namely, the continued reduction in revenues of uncontrolled infected vines and the expected economic losses due to infected vines spreading the infection to healthy ones. Our results show that it is beneficial to incur the costs of disease control earlier to avoid future damages and realize the benefits of a healthy, productive vineyard—that is, for non-spatial strategies, it is better to target younger vines in their earlier infection stages. For

¹⁵ For example, we find that the final number of vines rogued and replanted under strategy I_mNS2EW is almost twice as under strategy I_mNS . Consequently, the vineyard age under strategy I_mNS2EW is three to seven years lower than under strategy I_mNS (by the twenty-fifth and fiftieth year, respectively). As a result, the expected vineyard half-life is 1,639 months (approximately 137 years) under strategy I_mNS2EW , compared with 2,533 months (approximately 211 years) under strategy I_mNS .

spatial strategies, testing the neighborhood of all symptomatic vines reduces uncertainty through early detection and control. Finally, our analysis shows that incurring virus testing costs is justified for strategies testing the immediate neighbors of symptomatic vines (strategy I_mNSEW and strategy I_mNS).

Sensitivity Analyses

We use the ENPVs from the simulations with alternative parameter values, together with the ENPVs calculated using

the baseline parameters, to guide GLRD research investments by plant scientists, plant pathologists, and entomologists. Our sensitivity analyses deal with two critical questions. First, what parameter values make eradication possible and optimal? Second, what is the threshold expenditure for a virus test cost beyond which the optimal spatial strategy becomes cost-ineffective?

Eradication Feasibility and Optimality

We first focus on finding parameter values that make eradication possible and optimal.

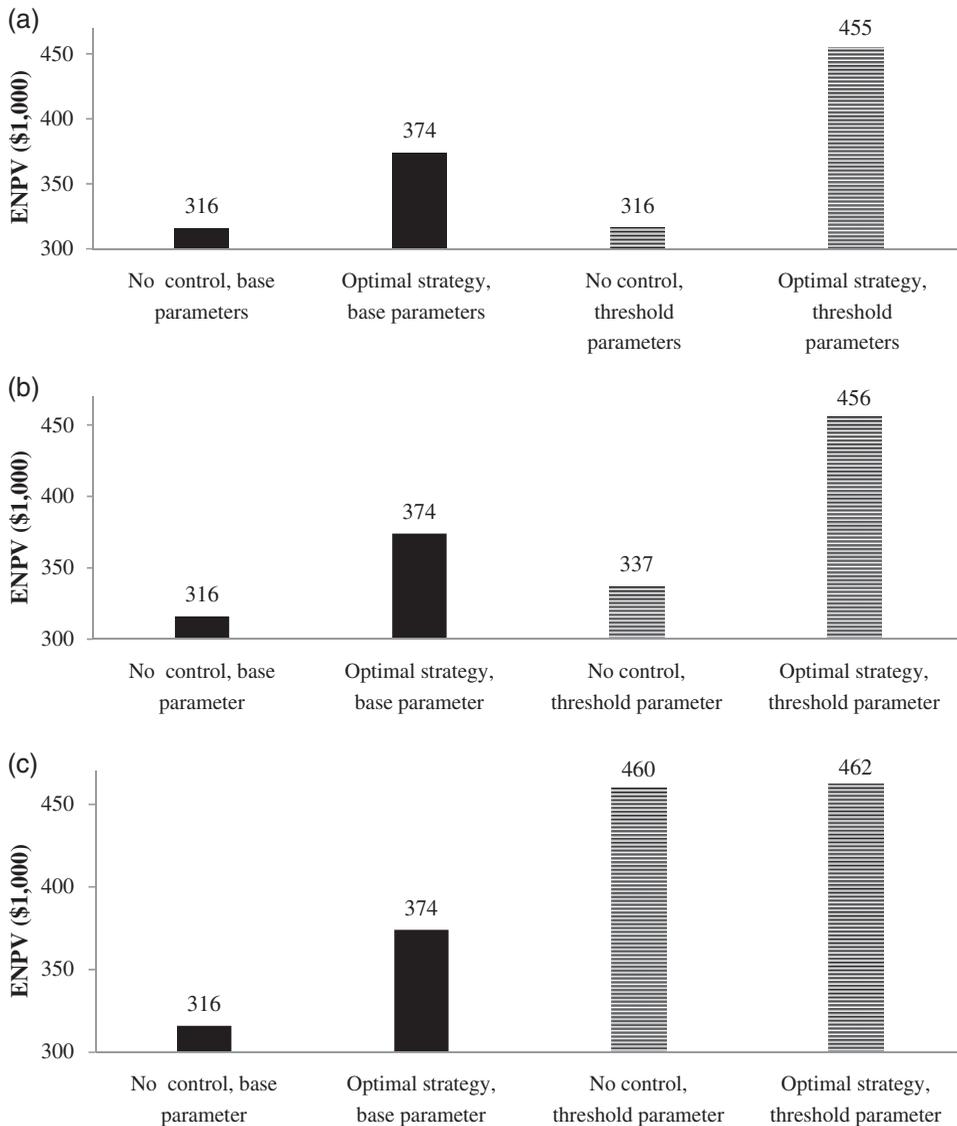


Figure 2. Sensitivity of expected net present values (ENPVs) to the virus undetectability period (a), within-column transmission (b), and initial infection level (c) parameters

We find that strategy I_mNS and strategy I_mNSEW achieve eradication with 99% and 100% probability, respectively, when the minimum (m_1), maximum (m_2), and mode (m_3) of the undetectability period's probability density function are substantially reduced. We simulate reductions from four, eighteen, and twelve months in the baseline model (values in Cabaleiro and Segura 2007; Constable et al. 2012; Tsai et al. 2008) to approximately one, four, and two months, respectively (figure 2(a)). Eradication is achieved under optimal strategy I_mNSEW at these threshold parameter values, yielding an ENPV improvement of \$139,100 (the difference between \$455,100 and \$316,000 in figure 2(a)) over the strategy of no disease control. The ENPV improvement is statistically significant at the 1% level. The reduction in the undetectability period parameters might be achieved through new technology able to detect the virus one month after infection. Using the ENPVs in figure 2, we estimate the value of such technology at \$81,000 under optimal strategy I_mNSEW (the difference between \$455,000 and \$374,000 in figure 2(a)).¹⁶

Eradication is also possible and optimal under strategy I_mNS , with a probability of 97% if the within-column transmission rate (α) is reduced from 4.2 to 0.1. At this threshold rate, strategy I_mNS yields an ENPV improvement of \$119,000 over the strategy of no disease control (the difference between \$456,000 and \$337,000 in figure 2(b)). This ENPV improvement is statistically significant at the 1% level. Reducing the within-column transmission rate to 0.1 per month has a value of \$82,000 under optimal strategy I_mNS (the difference between \$456,000 and \$374,000 in figure 2(b)). Reduction in within-column transmission is theoretically possible through vector management. However, monitoring and controlling mealybugs through biological control methods and pesticides has proven ineffective to date (Daane et al. 2012).

The initial infection level is a third critical parameter that may affect the possibility and optimality of eradication. Lowering the initial infection level from the base value (2%) to

the lowest possible level (0.02% or one initially infected vine) achieves eradication with a probability of 50% under optimal strategy I_mNSEW . At this initial infection level, strategy I_mNSEW yields an ENPV improvement of \$2,000 over the strategy of no disease control (difference between \$462,000 and \$460,000 in figure 2(c)). The ENPV improvement is statistically significant at the 1% level. A research program targeting reductions of initial infection levels has a value of \$88,000 under optimal strategy I_mNSEW (difference between \$462,000 and \$374,000 in figure 2(c)). For example, this result suggests that a sanitary program ensuring that 99.98% of the supplied planting material are virus-free (i.e., 0.02% initial infection level) may be justified if the cost is less than \$88,000 for a 5.2-acre vineyard (e.g., the National Clean Plant Network of the U.S. Department of Agriculture, Johnson 2009).

Disease Control Costs

Finally, we find that the ENPV improvement under the second-best strategy (I_mNS) is less sensitive to increases in disease control costs than under optimal strategy I_mNSEW . When the virus test cost increases twofold (from \$2.61 to \$5.22 per vine), Strategy I_mNS becomes optimal. The ENPV improvement for the originally optimal, more testing-intensive strategy I_mNSEW , decreases by 8 percentage points (from 19% to 11%; figure 3). In contrast, the ENPV improvement for strategy I_mNS decreases by just 4 percentage points (from 18% to 14%; figure 3). Beyond a fivefold cost increase (from \$2.61 to \$13 per vine), strategy I_mNSEW becomes cost-ineffective (figure 3). We also vary the costs of roguing and replanting and find that, when the unit cost of roguing and replanting is 1.2 times higher than the base value, strategy I_mNSEW becomes cost-ineffective. Strategy I_mNS , on the other hand, retains its cost-effectiveness up to a break-even parameter value of \$30 per vine (fourfold increase over the base value).

For vineyard managers, these sensitivity results highlight that, although strategy I_mNSEW is optimal under the base parameter values, second-best strategy I_mNS is less sensitive to changes in the costs of disease control. For scientists working on the disease, these results indicate that, although disease eradication can be optimally achieved if an

¹⁶ Recall that our unit of analysis is a 5.2-acre vineyard with 5,720 vines.

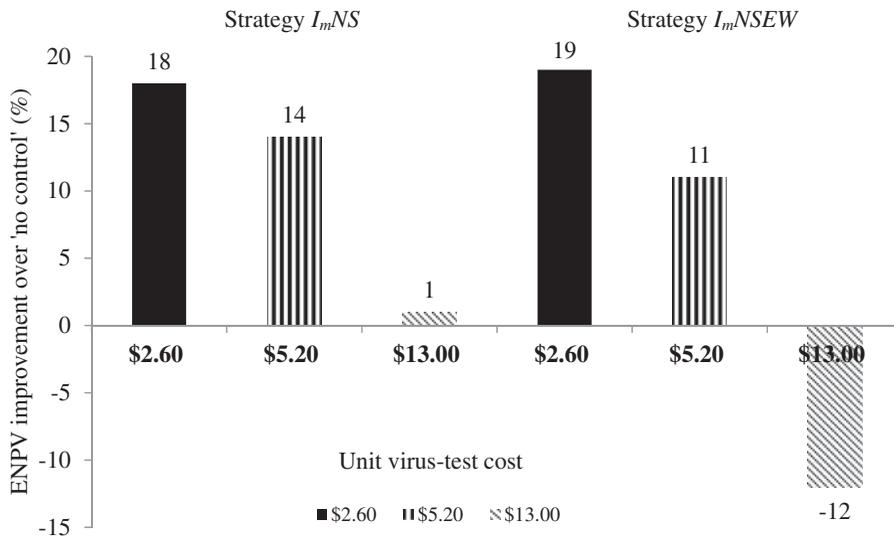


Figure 3. Sensitivity of the expected net present value to the unit virus-test cost

early detection technology is developed, the cost of this technology should not exceed \$13 per vine for it to be cost-effective in spatially controlling GLRD.

Conclusions and Directions for Future Research

There is growing interest in researching the economics of integrated spatial-dynamic processes in general and pests and diseases in particular. This article features a plant-level, bioeconomic model of GLRD diffusion and control in a vineyard. We analyze alternative disease control strategies that would not be possible using classical approaches. The originality of the results lies in the computational method's ability to model a large number of bioeconomic, plant-level state variables.

Our results show a general feature of spatial-dynamic processes: optimal policy interventions are those that achieve the temporally, spatially, and quantitatively optimal allocation of inputs. The results are particularly valuable for vineyard managers because they highlight the superiority of spatial strategies over nonspatial strategies recommended to the industry. We also estimate the expected value of research programs aimed at decreasing or increasing the critical model parameters. These results can help guide research efforts of disease ecologists,

plant pathologists, entomologists, and plant scientists involved in GLRD research.

This model has wider application possibilities and can be adapted to other crop diseases characterized by spatial-dynamic processes after adjustments for spatial configuration and input data. In particular, it can be used to inform profit-maximizing disease management in horticultural crops such as apple or citrus trees.

There are opportunities to extend the model as well. One extension would incorporate temporal price dynamics. If grape prices are substantially lower or higher than the mean price in the first year, optimal disease control strategies might be different than the ones identified in this article. Another extension would allow for spatial externalities caused by the flow of vectors from neighboring infected vineyards left uncontrolled. We expect this situation to yield strategies that alter the spatial configuration of the vineyard in a way that slows down disease progression. One such strategy might involve the creation of barriers to disease diffusion (e.g., Brown, Lynch, and Zilberman 2002; Foroutan 2003; Sharov and Liebhold 1998). Establishing "fire breaks" from an adjacent, infected vineyard may result in immediate yield losses that will need to be measured against the expected value of lower disease damages in the future. If cost-efficient, these designs might be recommended for the establishment of more disease-resistant vineyards and orchards with higher ENPVs.

Supplementary material

Supplementary material is available at http://oxfordjournals.org/our_journals/ajae/online.

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