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Research paper

Inoculum dose, diversity, dispersal, and damage: Simulating optimal economic control of hop powdery mildew at the landscape level

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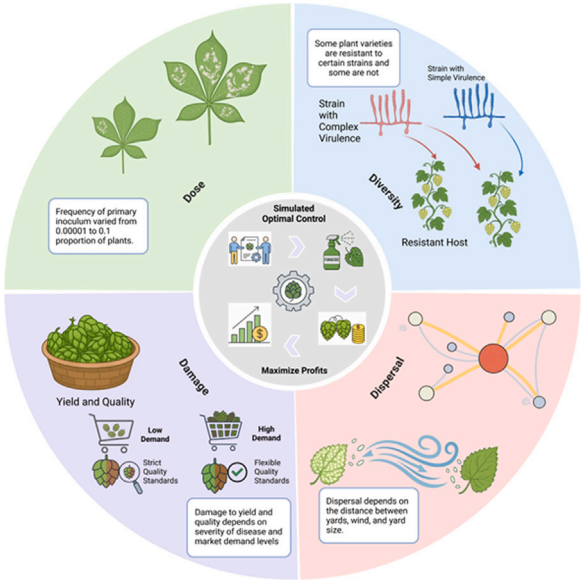
HIGHLIGHTS

- Mean profitability decreases as the likelihood of primary infection increases.
- Profitability is sensitive to where primary infection occurs in the landscape.
- Primary inoculum dose has a threshold-like response on simulated optimal control.
- Early intervention is essential for maximizing profitability region-wide.
- Simulated optimal controls are largely robust to varying market demand scenarios.

GRAPHICAL ABSTRACT



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ABSTRACT

CONTEXT Plant pathogens that disperse by airborne propagules may cause damage that extends beyond the borders of individual fields. Developing sound management strategies, therefore, requires consideration of heterogeneity in pathogen transmission, the effectiveness of control measures, host susceptibility and pathogen virulence, and the resulting economic outcomes that scale up at the regional level with coordinated management. OBJECTIVE We use hop powdery mildew as a motivating pathosystem to develop a coupled epidemiological-economic model to enable simulation of the impact of epidemic conditions and coordinated management interventions on profitability. This pathosystem is a well-suited case study because disease development may be limited by primary inoculum and fungicide applications, yet the pathogen can spread via long-distance dispersal between fields and rapidly damage both crop yield and quality. METHODS We

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parameterized the model using data collected from a census survey of commercial hop yards in Oregon during 2014 to 2017, including the monthly incidence of plants with powdery mildew, fungicides applied by growers, and estimated revenue depending on how the incidence of diseased hop cones affects yield and the likelihood of crop devaluation. We show that conditions in the early stages of epidemics related to primary inoculum dose, pathogen diversity, and the intensity of management intervention interact and determine the optimal regional control strategy. RESULTS AND CONCLUSIONS As the likelihood of primary infection increases, due to either the dose of primary inoculum or virulence of the pathogen population, mean profitability decreases. These effects are most pronounced when primary infection occurs in yards that are highly connected in the disease transmission network. The choice of how many fungicide applications to make in response to initial infection has little effect on profitability when the primary inoculum is relatively infrequent. However, as primary inoculum increases, targeted fungicide applications made in the early stages of epidemics are essential for maximizing profitability region-wide. These principles hold across a range of market demand scenarios that impose different crop quality standards, with the optimal number of early-season fungicide applications remaining largely consistent despite substantially different crop prices. SIGNIFICANCE Our analysis addresses a multifaceted challenge in agricultural disease management where epidemic control decisions must account for interactions between pathogen biology, management practices, market conditions, and regional-scale disease transmission. This research provides a framework for formally understanding factors that influence the cost of disease in complex systems where pathogens disperse across management units.

1. Introduction

There is increasing scrutiny of pesticides in agriculture and a need to develop management strategies and policies that reduce inputs while maintaining productivity and profitability (Pimentel, 2005; de Waard et al., 1993). Disease management efforts are most often directed at the scale of individual fields or farms, but plant pathogens do not respect management units or jurisdictional boundaries (Gilligan, 2008). Management may be suboptimal or ineffective when not properly matched to the scale of pathogen dispersal (Gilligan et al., 2007). The relative importance of inoculum produced endogenously within a given field or exogenously in other locations may dictate the mitigation strategies and the need for collective action in the form of area-wide management (Irwin, 1999).

Foundational theory in plant disease epidemiology suggests that mitigation efforts that reduce primary inoculum only delay epidemics, with the effect being inversely proportional to epidemic velocity (van der Plank, 1963). Yet, a number of empirical studies and modeling suggest that early intervention is critical for containing the spread of disease outbreaks (Cunniffe et al., 2015b; Fraser et al., 2004). Epidemics caused by pathogens capable of long-distance dispersal appear sensitive to the conditions in the initial outbreak area (Severns and Mundt, 2022; Estep et al., 2014). Final epidemic severity may depend on the size of the initial focus, pattern of initial inoculum, proportion of susceptible hosts in the population, and connectivity to other susceptible hosts (Ojiambo et al., 2017; Severns et al., 2014). The importance of these factors may depend sensitively on parameters controlling pathogen transmission and stochasticity in the earliest stages of an epidemic (Xu and Ridout, 1998). Concrete recommendations for control strategies also depend on the relative effectiveness and costs of controls (Margosian et al., 2009).

The need for modeling to explicitly express processes and current understanding of a system is clear (Pautasso et al., 2010; Lofgren et al., 2014). Bioeconomic models have been developed for multiple host-pathosystems, with the particular form of the model potentially including social (Etherton et al., 2023-03-01; Jeddi and DePaula, 2025) or biophysical processes (Jeddi and DePaula, 2025; Lazebnik et al., 2024) depending on the purpose of the model and specific system. The scale of prediction of bio-economic models varies as well, ranging from individual plants within a field to landscapes (Donatelli et al., 2017). Linking epidemiological models to crop damage and economic outcomes when pathogens potentially disperse over long distances is a challenging problem (Cunniffe et al., 2015a). Modeling dispersal at the mesoscale is especially challenging because microclimate, canopy architecture, and terrain and other landscape features influence aspects of inoculum dispersal, transport, and deposition (Aylor, 2017). The well known SIR and SEIR frameworks may require extensive adaptation or simply fail to capture the complexity of agricultural systems and unique properties of a given pathosystem (Lazebnik et al., 2024). Furthermore, estimation of R_0 to understand epidemic thresholds can be exceedingly difficult in SEIR models because high-quality data are rarely available and numerous assumptions must be made in models that capture the biological complexity of an entire system (Delamater et al., 2019). Agent-based process-based models offer an alternative (Donatelli et al., 2017), but these models also require many assumptions and carry heavy computational costs and data inputs that may be unavailable (Firester et al., 2018). Statistical network spreading models can offer a balance of biological realism and tractability needed to enable prediction of economically optimal outcomes in complex bio-economic systems with non-random mixing of sessile individuals (i.e., fields).

The motivating pathosystem for the research we present, hop powdery mildew, engenders many of these challenging aspects. The disease is caused by the fungus *Podosphaera macularis* and remains one of the most difficult and costly problems affecting hop producers in the western U.S. and other production regions. Success in breeding cultivars resistant to the disease has been met by the emergence of strains of the fungus that can overcome host resistance when the said resistance is broadly deployed in the landscape (Block et al., 2021; Gent et al., 2017; Royle, 1978; Wolfenbarger et al., 2016). Therefore, management still relies heavily on cultural practices that reduce inoculum density and fungicides to slow the rate of disease progression (Gent et al., 2019c; Mahaffee et al., 2009; Nelson et al., 2015; Probst et al., 2016; Royle, 1978).

This pathosystem has several biological and economic attributes that make it an exemplar for developing the modeling framework we propose herein, and also broadly of interest for other aerially-dispersed plant pathogens. First, the causal pathogen is an obligate biotroph with a host range limited to the plant family Cannabanaceae (Mahaffee et al., 2009), with hop being the dominant and most important host cultivated at the time of these studies (Rivedal et al., 2023). Consequently, it is possible to infer disease transmission between yards because other sources of inoculum are trivial. Further, this system is amenable to modeling dispersal between yards as *P. macularis* exhibits annual cycles of emergence, colonization, and extinction. The fungus persists from season to season in the Pacific Northwestern U.S. only in association with living host tissue, provisioned by infected crown buds, due to the absence of the sexual stage of the pathogen in this region (Gent et al., 2008; Weldon et al., 2021; Wolfenbarger et al., 2015). Bud infection and overwintering of the pathogen lead to highly focal infections owing to the emergence of heavily infected shoots in spring, the so-called flag shoots, which occur at a low frequency in commercial hop yards (Gent et al., 2019c; Laurie et al., 2023). From these focal infections, *P. macularis* disperses via wind and readily spreads within the network of

hop yards to create regional epidemics (Gent et al., 2019a). At the end of the cropping season, the pathogen becomes locally extinct in most yards due to its low overwintering survival (Gent et al., 2019c, 2018). Thus, a new realization of the disease development and transmission process can be observed annually.

Pathogenic variation is common in pathogens across plant and animal hosts (Burdon and Silk, 1997; Koelle et al., 2022) and has important implications for the dynamics of disease transmission and severity (Mundt, 2002; Ostfeld and Keesing, 2012). This is especially relevant in agricultural systems where *R*-gene-mediated resistance is deployed and creates a mosaic of resistant and susceptible hosts (McDonald and Linde, 2002). Host resistance to powdery mildew is commercially available in hop and multiple pathogenic variants (strains) exist in *P. macularis* in certain populations (Gent et al., 2017; Royle, 1978; Wolfenbarger et al., 2016) As hop plants are long-lived perennials (Neve, 1991), relatively stable patterns of resistant and susceptible cultivars are present in the landscape.

The pathosystem is of further interest because powdery mildew may cause economic losses by reducing both yield and crop quality (Gent et al., 2014; Neve, 1991; Royle, 1978). This is a common scenario in agricultural systems where crop damage is not attributed solely to direct losses in yield, but additionally or primarily from reductions in crop quality (Savary et al., 2006). This is particularly important for crops marketed directly to consumers with cosmetic value (Esker et al., 2013; Zadoks, 1985). In these situations, the financial loss incurred by the grower may be linked in complex ways to market demand (Zadoks, 1985). Indeed, this is the situation with hops and powdery mildew because the disease may reduce brewing value and cause conspicuous visual defects from degraded cone color. For this reason, growers apply fungicides repeatedly during the season with the goal of minimizing inoculum pressure for the critical cone phase of the disease (Nelson et al., 2015; Royle, 1978). Fungicide applications for powdery mildew may occur over a period of multiple months to minimize foliar infections in spring and then later in summer to minimize cone infection (Nelson et al., 2015; Twomey et al., 2015). Thus, growers must make multiple decisions on when to begin treatment and how intensively to treat.

Adding even more uncertainty, hops are sold almost entirely through marketing contracts that stipulate quality standards, usually with vague or subjective quality terms. Contracting is conducted for numerous agricultural commodities, and in 2017, 21 percent of total U.S. crop production was under a contract agreement (MacDonald and Burns, 2019). Price risk reduction is stated as a major incentive for contracting (Key, 2005). The potential for crop devaluation or, in the worst case, rejection, may substantially affect disease management choices since the producer assumes firstly the risk of crop damage and secondly the penalty for failing on a contractual obligation (Zhang et al., 2015). We expect that optimal disease control strategies may therefore be sensitive to contract structure when crop value or saleability is inseparably linked to crop quality metrics. Furthermore, the optimal control strategy may depend on practices in other fields and other farms given the potential for long-distance dispersal of the pathogen.

Motivated by this pathosystem, we draw upon an exceptionally rich data set collected from a census survey of hop yards in Oregon over a four-year period for our analyses (Gent et al., 2019a). We formulate an epidemiological model for the development and spread of powdery mildew at the regional level, including the effect of fungicides applied in a field of interest and all other potential source fields. We couple the epidemiological model to an economic model of expected revenue and costs associated with disease management, devaluation or rejection due to quality defects, and market conditions (Fig. 1). We then simulate varying conditions of the initial phases of epidemics related to primary inoculum dose, pathogen diversity, position in the disease transmission network of fields, and management intervention to identify control strategies that maximize profit under varying market demand scenarios.

2. Material and methods

2.1. Study system

Our study system is the hop production region in western Oregon. Oregon is one of the leading hop-producing regions in the U.S., and commercial production is concentrated in a few counties in the Willamette Valley in the western portion of the state (Barth, 1994). Hop powdery mildew was first confirmed in the field in Oregon in 1998 and has occurred annually each year since then (Mahaffee et al., 2003; Ocamb et al., 1999). Historically, powdery mildew has tended to occur most regularly and most severely in production regions in the eastern extent of the Willamette Valley, presumably due to the frequency of overwintered inoculum (Gent et al., 2019c; Turechek et al., 2001) and subsequent dispersal from the resultant disease foci (Gent et al., 2019a). We intentionally focused on hop farms in the eastern production regions for data acquisition. The diversity of cultivars and production practices in this region is summarized elsewhere (Laurie et al., 2023).

2.2. Biological data acquisition

We obtained monthly data on the incidence of plants with powdery mildew or primary infection (i.e., occurrence of a flag shoot) from a census survey of hop yards representative of the production region. Disease assessments were conducted monthly from April to July from 2014 to 2017, the period when powdery mildew readily spreads on foliage (Turechek et al., 2001). For brevity, a summary of the disease assessments methods is provided here; a complete description of the methods is given in (Gent et al., 2019a). There were 8 to 10 farms sampled per year, all within Marion County, Oregon, with a maximum distance between yards of 26 km. After cleaning to address duplicate entries or naming errors, data were available for 99 yards assessed in 2014, 113 in 2015, 116 in 2016, and 122 in 2017. All cultivars were evaluated, independent of their susceptibility to powdery mildew (Laurie et al., 2023). Sampling within yards to estimate the incidence of plants with powdery mildew was assessed using a modification of cluster sampling methods described previously (Turechek et al., 2001; Turechek and Mahaffee, 2004).

As noted previously, there are multiple pathogenic strains of *P. macularis* that differ in their ability to cause disease on hop plants with specific resistance genes (*R*-genes). Two strains of the pathogen were relevant at the time of the field surveys and are differentiated based on their ability to cause disease on plants with a resistance termed *R6* (Padgitt-Cobb et al., 2019; Wolfenbarger et al., 2016). The presence of *R6* resistance is analogous to immunization that provides complete, but strain-specific, protection from disease. The presence of *R6* in hop plants provides resistance to powdery mildew only when the pathogen lacks a corresponding virulence (*V*). For shorthand, we refer to strains that cannot infect plants with *R6* resistance as non-*V6*-virulent. Other strains of the pathogen may infect susceptible plants that lack *R6*, but importantly, can also infect plants that possess *R6*. These strains are dubbed *V6*-virulent. *V6*-virulent strains are promiscuous in the yards they may affect, as they infect hop cultivars that both possess or do not possess *R6*, analogous to a vaccine-evading strain of a pathogen.

When we detected powdery mildew in a given hop yard, it was necessary to match the strain of the pathogen present to potential yards where the pathogen could disperse to cause disease. Therefore, the initial strain of the pathogen present was determined to be *V6*-virulent or non-*V6*-virulent using bioassays as described previously (Gent et al., 2019a; Wolfenbarger et al., 2016).

2.3. Pesticide use and costs

We obtained pesticide application records from each grower for all yards sampled during 2014 to 2017 and interrogated their records to

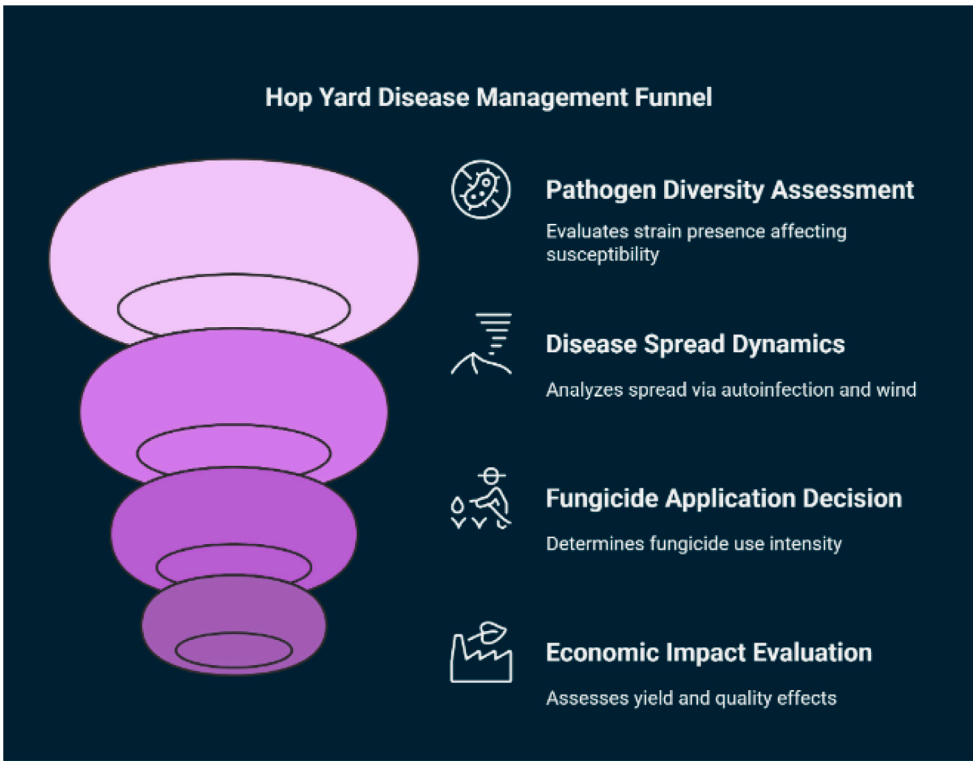

Fig. 1. Conceptual framework for disease management in the motivating pathosystem. This funnel diagram illustrates an idealized multi-stage decision-making process in disease management, showing the sequential evaluation of pathogen diversity assessment, disease spread dynamics, fungicide application decisions, and economic impact evaluation. Each stage represents a key component of the integrated epidemiological-economic model developed in this study.

determine the timing and dosage of applications of herbicides, fungicides with activity against powdery mildew, and adjuvant additives. We did not consider insecticides or miticides because these were not directly relevant for the present analysis of powdery mildew. We then estimated the January 2022 real costs of the relevant pesticides and adjuvants applied by requesting price quotes for each product from each of three vendors in western Oregon that service hop producers as described by (Hwang et al., 2024). We estimated real prices using January 2022 as the base by adjusting the nominal price by the producer price index for farm products for each year available from the U.S. Bureau of Labor Statistics, and then averaged over all available years to derive a single real price per unit. The mean and variance of the estimated real price per unit for each pesticide are provided in Supplemental Table S2.

2.4. General modeling approach

We introduce a linked epidemiological-economic statistical model of epidemic development and apply this model to hop powdery mildew to simulate economic outcomes due to disease management costs and crop damage from direct losses in yield and quality defects. The epidemic model contains both stochastic and deterministic components to estimate the development of the foliar phase of powdery mildew originating from initial occurrences of *P. macularis* via bud perennation, autoinfection at the scale of individual hop yards from inoculum endogenous to a given yard, and exogenous inoculum dispersed from other yards in the region. The population moment estimate of the incidence of plants with powdery mildew is derived from a function of the probability of a plant being diseased, as moderated by fungicide use. Profits are defined as revenue minus fixed and variable costs as influenced by fungicide inputs, and yield and crop devaluation due to quality defects as a function of disease incidence. External drivers are inputs of wind data, fungicide use, and initializing values of the probability of initial (primary) infection due to bud perennation, expected crop yield in the absence of disease, and price per unit of yield. The model is spatially explicit at the scale of individual hop yards and uses the actual location and size of hop yards in the data set for a given year; there is no attempt to account mechanistically for focus development or disease spread within hop yards.

We use the linked models to simulate profit levels resulting from varying epidemic conditions. Concretely, these conditions were related to initial infection frequency, pathogenic diversity of the initial strain of the pathogen, the location of the initial infections, and the number of fungicide applications made in the first month of the epidemic. We aggregate responses from individual yards to scale to the regional population of yards. We then ran each simulation scenario under three market demand scenarios to understand the sensitivity of the results to market conditions.

2.5. Submodels

2.5.1. Epidemic network model

An individual hop yard is considered a node in a network of yards in the spatial extent of interest (Fig. 2). Disease status of a yard in a given month is a nonlinear function of its disease incidence in the preceding month, susceptibility to two races of *P. macularis*, and disease spread from other nodes as influenced by their disease incidence and area (source strength), distance apart, and wind run in the preceding month (Gent et al., 2019a). We introduced two parameters that moderate disease in a given hop yard and disease spread from other hop yards based on the number of fungicide applications made in the prior month. We also generalized the model by expressing the dispersal kernel as a function of distance and source strength to accommodate various functional forms. The impact of exogenous inoculum depends jointly on the pathogen virulence in the source yards and susceptibility of the cultivar planted in the target yard.

The number of diseased plants is given by Y_i, among a sample of n_i plants, with $i = 1, \ldots, N$ indexing yard identity. The probability of Y_i,

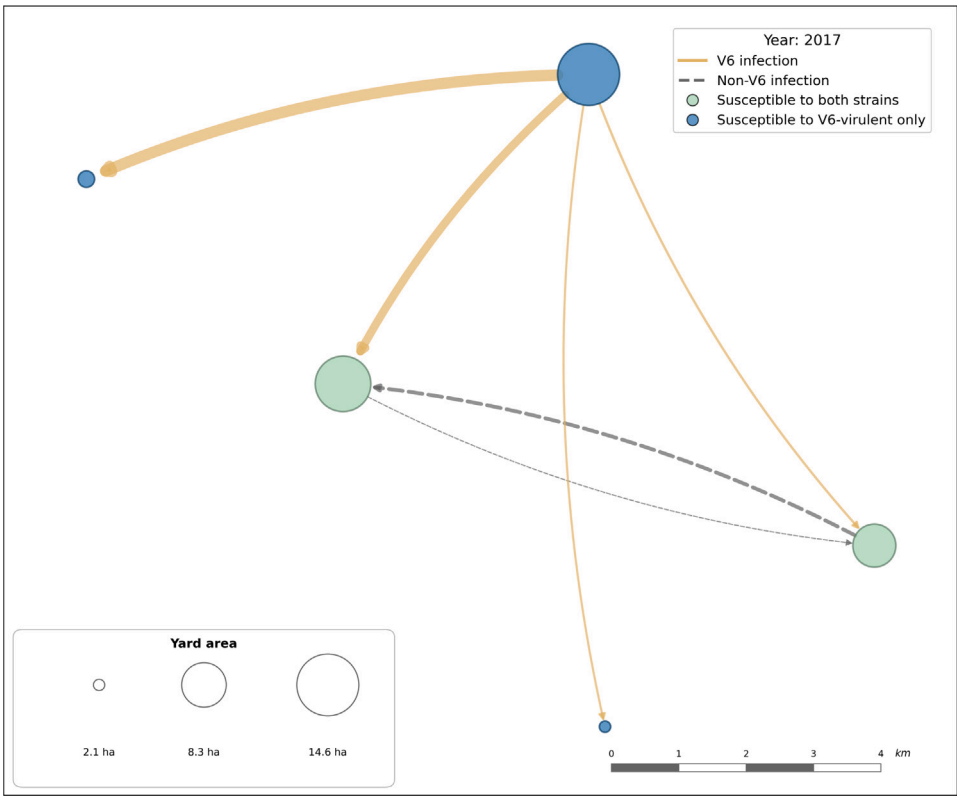

Fig. 2. Disease transmission network among sampled hop yards. A representative sample of hop yards (one per out-degree centrality quantile, 2017) connected by directed transmission edges. Node size is proportional to yard area. Green nodes denote cultivars lacking *R6* resistance (susceptible to both strains); blue nodes denote cultivars possessing *R6* (susceptible to *V6*-virulent strains only). Solid gold edges indicate *V6*-virulent source infection; dashed gray edges indicate non-*V6*-virulent source infection. Edge width reflects directed transmission weight (Eq. (1)); edges are omitted where strain–cultivar incompatibility precludes transmission. Refer to Section 3 for the definition of out-degree centrality and the quantile classification used.

is taken as binomially distributed with the predictor function expressed on the log-odds scale as:

$$\log\left(\frac{p_i}{1-p_i}\right) = \beta + \delta\left(\frac{\tilde{y}_i}{n_{\tilde{y}_i}} \cdot \exp\left(-\eta_1 s_i\right)\right) + \gamma \sum_{j=1}^{M_i}\left(\frac{a_j z_j}{n_{z_j}} \cdot w_{ij} \cdot \exp\left(-\eta_2 s_j\right) \cdot f\left(d_{ij};\alpha\right)\right) \quad (1)$$

Covariates and parameter interpretations are given in Tables 1 and 2. Parameters are allowed to vary for time transition periods from May to June and from June to July. The equation defines the model for disease development on foliage at the plant scale. We explored an exponential and also a power-law function for the dispersal kernel, but present results only for the power-law function (Eq. (2)), given theoretical considerations of expected dispersal characteristics of *P. macularis* (Severns et al., 2019).

$$f\left(d_{ij};\alpha\right) = \left(1 + d_{ij}\right)^{-\alpha} \quad (2)$$

2.5.2. Economic model

The profit function Π at time t over the growing season ($t = 0, \ldots, T$) measures the total profit per hectare across all hop yards $i = 1 \ldots N$:

$$\Pi(s_{it}) = \sum_{i=1}^{N}(\pi_{it})$$

where π_{it} is the profit per hectare at time t for yard i. This profit equation is conditioned on the number of fungicide sprays s_{it} across the season and subject to constraints imposed by the epidemic network model and by the price-quality relationships detailed below. Here, we assume risk neutrality of growers.

Table 1
Covariate notations for each yard of interest $i = 1, \ldots, N$ in a particular month, and all other yards $j = 1, \ldots, M_i$ relative to yard i.

Variable	Description
$\tilde{y}_i$	Number of diseased plants at yard i in the prior month
$n_{\tilde{y}_i}$	Number of plants sampled at yard i in the prior month
a_i	Area (hectares) of yard i
z_j	Number of diseased plants at yard j in the prior month
n_{z_j}	Number of plants sampled at yard j in the prior month
a_j	Area (hectares) of yard j
d_{ij}	Distance (kilometers) from centroids of yard i to yard j
w_{ij}	Wind vector on $j-i$ direction in the prior month
s_i	Number of fungicide sprays in yard i in prior month
s_j	Number of fungicide sprays in yard j in prior month

The profit π_{it} for each yard i at time t is defined as the difference between revenue and cost for each yard at time t:

$$\pi_{it} = R_t(q_i, v; p_i) - C_i(s_{it})$$

Revenue is only realized in the final period T, so that revenue is defined by $R_i(q_i, v; p_i) = R_T$, otherwise $R_i(q_i, v; p_i) = 0$. Revenue is a function of both yield q_i and price v, conditioned on the disease probability level p_i. Costs C_i, defined in more detail below, are a function of fixed costs and the number of fungicide sprays, s_{it}.

In the revenue function both yield quantity and price are adjusted by the incidence of cones with disease and associated quality defects as inferred from cone color. We used relationships reported in the literature to link the foliar phase of the disease to incidence of cones with powdery mildew, yield, and defects to cone color. First, the incidence of leaves with powdery mildew was estimated from the incidence of

Table 2
Interpretation of model parameters.

Parameter	Interpretation
β	Baseline log-odds of disease, after accounting for autoinfection and dispersal.
δ	Change in log-odds of disease associated with autoinfection at the yard scale, after accounting for disease spread.
γ	Distance-adjusted change in log-odds of disease associated with disease spread from other yards, after accounting for autoinfection.
α	Dispersal parameter providing distance adjustment to change in log-odds of disease associated with individual source yards.
η_1	Change in log-odds of disease associated with autoinfection at the yard scale, after accounting for fungicide sprays.
η_2	Dispersal parameter providing fungicide spray adjustment to change in log-odds of disease associated with individual source yards.

plants, p_i, with powdery mildew through the hierarchical relationship between disease at these scales (Turechek and Mahaffee, 2004):

$$\text{Leaf Incidence} = 1 - (1 - p_i)^{D/n}$$

where n is the number of leaves sampled per plant and D is the index of dispersion. We assume $n = 50$ and D as reported previously (Turechek and Mahaffee, 2004). The incidence of cones with powdery mildew was assumed to be a linear function of the incidence of leaves with powdery mildew (Turechek et al., 2001) as:

$$\text{Cone Incidence} = a_1 \times \text{Leaf Incidence} + b_1$$

Yield loss was then estimated assuming a linear relationship with cone incidence, which is in turn derived from leaf incidence (Gent et al., 2014):

$$\text{Yield Loss} = a_2 \times \text{Cone Incidence}$$

We considered cone color a surrogate for quality defects associated with powdery mildew that could lead to crop devaluation or rejection based on production contract standards. Cone color was modeled using an industry-accepted ordinal scale as an exponential decay function (Gent et al., 2014):

$$\text{Cone Color} = 10 + a_3 \cdot (1 - \exp(-b_3 \times \text{Cone Incidence}))$$

These empirical relationships are presented in Fig. 3. Parameter estimates for the damage functions are given in Supplemental Table S1.

Market conditions and production contracts also impact revenue as shown in Fig. 4. Production contracts were reviewed and plausible damage functions were derived from expert opinions solicited from two hop producers and a hop merchant for crop devaluation from moderate damage, salvage value if the crop was severely affected and was alternatively extracted for alpha-acids, or unsaleable due to extreme damage. We modeled these scenarios as a sigmoid function with varying thresholds dependent on low, medium, and high market demand, as:

$$\text{Adjusted Price} = \text{Initial Price} \times \sigma(\theta_0 + \theta_1 \cdot \text{Cone Color})$$

where σ is the sigmoid function and θ_0, θ_1 are parameters estimated for different market demand scenarios. Revenue in a given yard is then calculated as the product of Adjusted Price and expected yield. Yield in the absence of disease was taken as the average yield reported in 2020 by the National Agricultural Statistics Service for the four cultivars we considered, as we describe below. The initial price was taken from representative production contracts for each of the three estimated levels of demand.

The cost function for each hop yard C_i incorporates the costs associated with fungicide sprays along with fixed costs and other variable costs over the production season:

$$C_i(s_{i,t}) = C_F + C_V + \sum_{t=0}^{T}(C_{s,t} + C_{a,t})$$

where C_F is the fixed cost, C_V represents other variable costs, $C_{s,t}$ is the fungicide cost, and $C_{a,t}$ is the application cost at period t. Fixed and other variable costs were obtained from Galinato (Galinato, 2020).

2.5.3. Parameter estimation

The parameters in the epidemic network model were estimated using maximum likelihood. The likelihood function was constructed by assuming the number of diseased plants Y_i in each yard i follows a binomial distribution with probability p_i given by the inverse logit of η_i in the equation above. The log-likelihood of the parameters $\boldsymbol{\theta} = (\beta, \delta, \gamma, \alpha, \eta_1, \eta_2)$ given the observed data $\boldsymbol{D} = (y_i, n_i, \tilde{y}_i, n_{\tilde{y}_i}, a_i, s_i, z_j, n_{z_j}, a_j, d_{ij}, w_{ij}, s_j)$ where $i = 1 \ldots N$ and $j = 1 \ldots M_i$ is:

$$\ell(\boldsymbol{\theta}; \boldsymbol{D}) = \sum_{i=1}^{N} \left[y_i \log p_i + (n_i - y_i) \log(1 - p_i) \right]$$

where $p_i = \text{logit}^{-1}(\eta_i)$ and η_i is a function of $\boldsymbol{\theta}$ and the observed covariates.

The maximum likelihood estimator $\hat{\boldsymbol{\theta}}$ was obtained by maximizing $\ell(\boldsymbol{\theta}; \boldsymbol{D})$ subject to bounds on the parameters to ensure they were biologically plausible. This was done using the Sequential Least Squares Programming (SLSQP) algorithm, a gradient-based nonlinear constrained optimization method (Kraft, 1988). Analytical expressions for the gradient and Hessian of the negative log-likelihood were derived and provided to the algorithm to improve convergence. Multiple random initializations of the parameters were used to ensure a global optimum was reached. The model was fit using data for all years, but separately for the May to June and June to July transition periods to allow parameters to vary between these periods. Convergence was assessed by monitoring the magnitude of the largest gradient and the positive definiteness of the Hessian matrix at the obtained solution.

All analyses were conducted in Python, making use of the NumPy, SciPy, and Statsmodels libraries for numerical computing and statistical modeling.

3. Simulation experiments

For the simulation experiments described below, we constructed a synthetic landscape using the actual locations and sizes of hop yards in each of the years 2014 to 2017 from our data set. Each hop yard in the synthetic landscape was planted to one of four representative cultivars (Chinook, Simcoe®, Nugget, or Mosaic®), chosen because they were commercially relevant at the time of this research, either possessed or did not possess the *R6* resistance, and can be sold for direct use in brewing or alternatively can be processed to extract alpha-acids as a secondary market. We assumed equal susceptibility to powdery mildew given a compatible strain of the pathogen. Cultivar assignments were made at random for each yard, subject to an approximately 1:1 ratio of cultivars that possess *R6* or non-*R6* in each of the quantiles as we describe below. The same yard-level assignments were used across simulation runs for a given year.

We initiated epidemics at the start of May under varying scenarios by specifying an initial probability of disease, which governed the expected frequency of initial infections (flag shoots) in the population of hop yards. In reality, any given yard may or may not harbor flag shoots, making the true distribution of early-season disease unknown. We therefore modeled the occurrence of initial infections stochastically

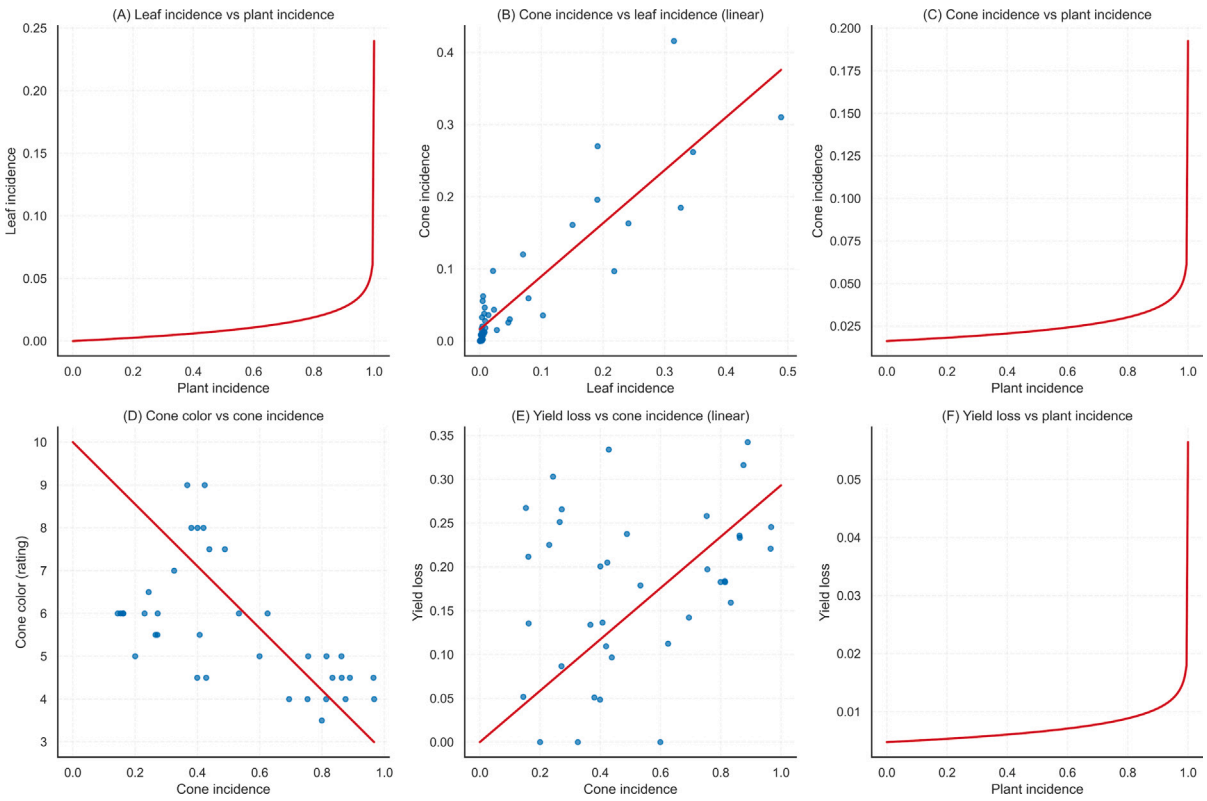

Fig. 3. Empirical relationships between disease incidence and crop damage. Six panels show the fitted relationships used in the economic model: (A) Leaf incidence as a function of plant incidence, (B) Cone incidence versus leaf incidence with linear regression, (C) Cone incidence versus plant incidence, (D) Cone color degradation as a function of cone incidence, (E) Yield loss versus cone incidence with linear relationship, and (F) Yield loss versus plant incidence. These empirical relationships link the epidemiological model outputs to economic damage estimates for yield quantity and quality metrics. Refer to Supplemental Table S1 for specific parameter estimates.

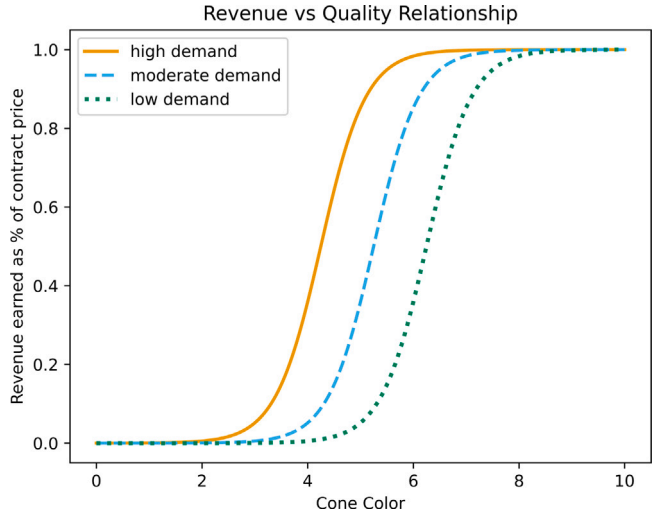

Fig. 4. Relationship between crop quality and revenue under different demand scenarios. Sigmoid functions model the relationship between hop cone quality (measured by cone color on a 10-point scale) and the revenue earned as a percentage of the contract price. The curves illustrate three distinct market demand scenarios: high, moderate, and low. Under low demand (green curve), stricter quality standards are enforced, meaning a higher cone color score is required to achieve the full contract price compared to moderate (blue curve) or high (red curve) demand conditions. These functions are used in the economic model to calculate crop devaluation based on simulated disease levels. Refer to Supplemental Table S1 for specific parameter estimates.

as follows. For each yard in the landscape, we first identified the proportion of plants there that were susceptible, which depends on whether a yard was planted with a cultivar susceptible to *V6*-virulent and/or non-*V6*-virulent strains. We also identified which quantile of the out-degree centrality distribution the yard occupied. Out-degree centrality was computed from a directed transmission network: for each ordered pair of yards, the edge weight from source yard i to target yard j is given by $a_i \cdot w_{ij} \cdot (1 + d_{ij})^{-2}$, incorporating source yard area, directional wind run, and a power-law distance decay (cf. Eq. (1)). The weighted out-degree measures a yard's potential to disperse inoculum to other yards in the landscape (Gent et al., 2019a). Denote this quantile by q_i for yard i, and let $0 \le q_i \le 1$ (discretized into 20% quantile bins).

Next, we specified a desired mean initial probability of disease across the entire landscape, denoted by p_0. Because yards differ in their centrality and susceptibility to a given strain of the pathogen, we introduced an adjustment factor so that the final landscape-wide fraction of plants with flag shoots would closely match p_0. Specifically, within each out-degree centrality quantile q, let:

$$\{\text{proportion susceptible in quantile } q\} = S_q.$$

$$\{\text{proportion of yards in quantile } q\} = P_q.$$

We then scaled p_0 by $\frac{1}{S_q}\frac{1}{P_q}$, effectively concentrating flag shoots into those yards that were actually susceptible in the particular quantile q (see Appendix 6.3 for a detailed derivation of this adjustment). Denote this adjusted probability by $p_0^*(q)$. For each yard i in quantile q, we then drew:

$$y_{0,i} = \text{Binomial}\big(n_i,\ p_0^*(q)\big),$$

where n_i is the number of plants in yard i. Thus, $y_{0,i}$ is the number of initial infections (flag shoots) in yard i at the start of May. $y_{0,i}$ was set

Table 3
Key parameters varied in the simulation, their tested ranges, and the rationale.

Parameter	Values Tested	Rationale/Data
Year	2014, 2015, 2016, 2017	Real yard size, distance, and wind patterns were available for each year, reflecting distinct conditions.
Simulations	100	Each scenario is replicated to capture stochastic variability (see text).
Quantiles	5	Yards are classified into five bins by out-degree centrality, used when varying location-based initial infections.
Pathogen-diversity	0.0, 0.25, 0.50, 0.75, 1.0	Varies fraction of initial inoculum from *V6*-virulent vs. non-*V6*-virulent strains.
Sprays in May	0, 1, 2, 3, 4, 5, 6, 7, 8, 9, 10	Extra sprays (beyond baseline) if a yard has flag shoots. Upper end (10) tests diminishing returns.
Initial infection probability (p_0)	$10^{-5}, 5\times10^{-5}, 10^{-4}, 5\times10^{-4}, 10^{-3}, 5\times10^{-3}, 10^{-2}$	Represents the probability of a plant harboring an overwintered infection. Spans extremely rare (10^{-5}) to moderately frequent (10^{-2}).
Market scenario	low, moderate, high	Captures three contrasting demand/price structures, impacting crop value and quality penalties.

to zero if the initial strain that was randomly placed was biologically impossible with the cultivar in that yard.

With these initial conditions set, we simulated disease progression through May, June, July, and into the early cone-development period using the network-based epidemiological model described in Section 2. In each monthly step, the incidence of infected plants in yard i was predicted using the fitted coefficients from the equation above. Fungicide applications served as an input to the model via coefficients that reduce the rate of new infections in both the focal yard and the potential source yards; the number of sprays in a yard was itself predicted by a Poisson regression fitted from the data on how many fungicide applications growers made in a yard in response to the incidence of diseased plants in the previous month. In May, the number of fungicide applications for each yard was drawn stochastically. If a yard *did not* contain disease, its spray count was drawn from a Poisson distribution with mean $\lambda_0 = 0.43$, the observed average for disease-free yards in the raw data, representing routine prophylactic spray behavior. If a yard *did* harbor at least one flag shoot (i.e. $y_{0,i} > 0$), its spray count was drawn from a Poisson distribution with mean λ_{may}, a user-specified parameter ranging from 0 to 10 to represent varying intensities of early-season intervention (Hwang et al., 2025). From June onward, the expected number of fungicide applications in each yard was predicted deterministically by Poisson regression models fitted to the grower survey data, with spray counts in each month conditional on disease incidence in the preceding month (Appendix 6.2). Late-season (post-July) applications were also predicted by the Poisson regression model; these contributed to management costs but did not further affect simulated disease progression.

After simulating disease progress through the end of July, we computed the incidence of diseased cones using the relationship in Fig. 3C. The economic model then used these incidence levels to estimate yield losses, potential quality reductions (via the cone-color model), and resulting net returns after accounting for fixed costs, variable costs, and the cost of sprays. We repeated these simulations for a range of p_0 values, different fractions of *V6*-virulent strains present in the initial inoculum, and different yard-level centralities in which the initial flag shoots occurred. For the primary inoculum dose, we varied p_0 from 10^{-5} to 10^{-2}, a large but plausible range of primary infection levels (Laurie et al., 2023). The proportion of initial infections caused by*V6* or non-*V6*-virulent strains was varied from 0% to 100% in 25% steps. For out-degree centrality, we placed the primary inoculum into each 20% quantile (Table 3). Each scenario was run 100 times to capture stochastic variability arising from three sources: binomial draws of initial flag shoots ($y_{0,i}$), Poisson draws of May fungicide applications, and random assignment of *V6*-virulent strain presence across yards. We report either the mean or percentiles of the relative change in profit per hectare compared to a disease-free baseline in which yards still receive routine prophylactic fungicide applications but no disease is present.

Table 4
Parameter estimates for epidemic model.

Parameter		May–June	June–July
β_1	Baseline log-odds	−2.06	−2.79
β_2	Baseline log-odds	−4.15	−3.88
δ_1	Autoinfection effect	2074.35	2.94
δ_2	Autoinfection effect	120.46	8.04
γ_1	Dispersal effect	34 358.91	1390.71
γ_2	Dispersal effect	14 536.03	286.8
α_1	Dispersal kernel	0.71	1.0
α_2	Dispersal kernel	2.67	2.04
η_{11}	Fungicide effect	3.51	0.02
η_{12}	Fungicide effect	1.62	0.31
η_{21}	Fungicide effect	2.9	0.95
η_{22}	Fungicide effect	0.27	0.37

(a) Subscripts on parameters index cultivars susceptible only to strains with *V6*-virulence or cultivars susceptible to all strains.

3.1. Summary of simulation parameters

Beyond these parameters, the simulation used yard-level attributes (area, distance matrices, wind data) and the fitted model coefficients described in Sections 2 and 3. In each scenario, the model outputs include time series of disease incidence and final net profit estimates under the specified market-demand conditions.

4. Results

4.1. Parameter estimates for epidemic model

To understand the temporal dynamics of the epidemic model, we compared the parameter estimates for the two key transition periods: May–June and June–July (Table 4). Several notable differences emerge between these periods.

The baseline log-odds of disease (β_1, β_2) were lower (more negative) in May–June (−2.06, −4.15) than in June–July (−2.79, −3.88), indicating a lower baseline risk of disease early in the season. The effect of autoinfection (δ_1, δ_2) was much larger in May–June (2074.35, 120.46) compared to June–July (2.94, 8.04), suggesting that local sources of inoculum are most influential at the start of the epidemic. The effect of dispersal from other yards (γ_1, γ_2) was also much greater in May–June (34 358.91, 14 536.03) than in June–July (1390.71, 286.8), highlighting the importance of regional spread early in the epidemic. The dispersal kernel parameters (α_1, α_2) were larger in June–July (1.0, 2.04) than in May–June (0.71, 2.67), indicating a possible shift in the spatial scale of dispersal that increases longer distance dispersal as the season progresses. The fungicide effect parameters (η_{11}, η_{12}, η_{21}, η_{22}) were generally larger in May–June, especially η_{11} (3.51 vs 0.02), suggesting that fungicide applications are more impactful on reducing disease early in the season versus later. Overall, these differences

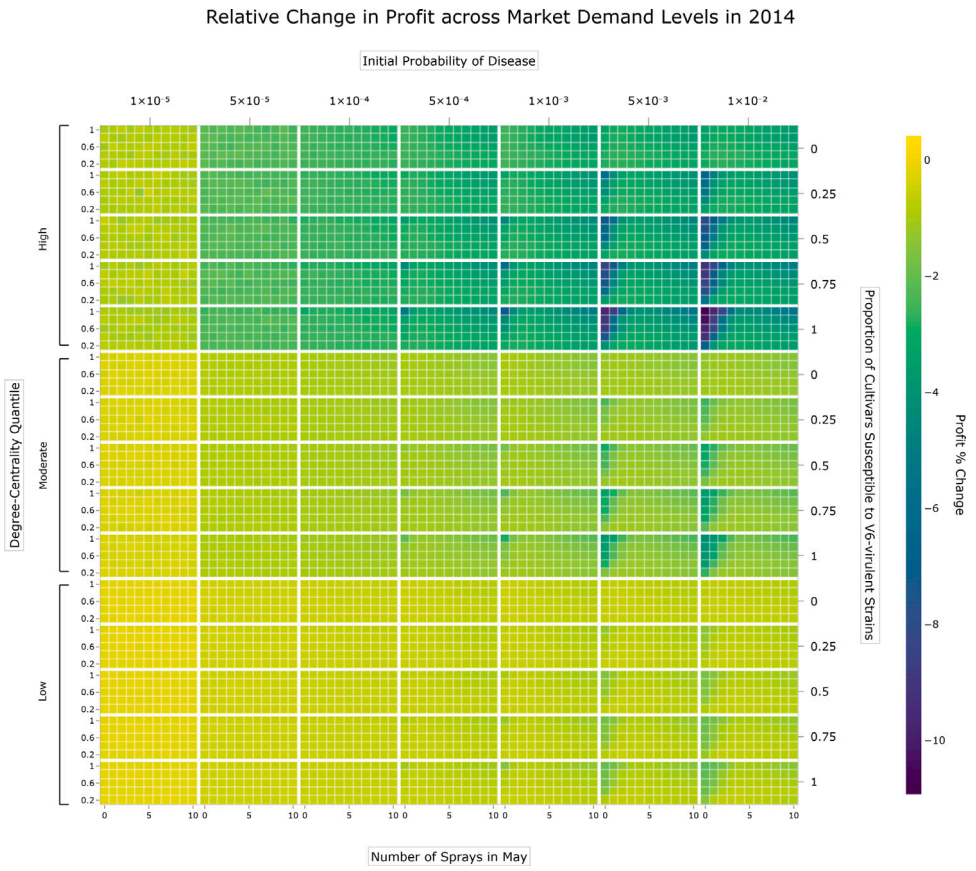

Fig. 5. Relative change in profit for high, moderate, and low market demand in 2014. This comprehensive heatmap shows the profit percentage change across different initial probability of disease (p_0) values (rows), number of sprays in May (columns), and out-degree centrality quantiles (panels within each market scenario) for varying pathogen V6-virulence percentages (0%, 25%, 50%, 75%, 100%) under three market demand scenarios (high, moderate, low). Yellow indicates minimal profit impact, while cooler colors represents more substantial profit losses.

indicate that both local and regional sources of inoculum are most important in the early epidemic phase, and that fungicide applications have their greatest impact when applied earlier in the epidemic. As the season progresses, the epidemic becomes less sensitive to these factors.

4.2. Simulation experiments

The simulation results reveal how profitability depends on initial epidemic conditions, specifically the initial inoculum dose (p_0), pathogen diversity (proportion of *V6*-virulent strains), out-degree centrality quantile of the initially infected yards, and market demand (Fig. 5). All simulated scenarios resulted in negative relative profit changes, indicating that any level of disease incidence reduces profit relative to the disease-free baseline. We present results primarily for 2014, the year with the highest observed disease incidence, and note that patterns were qualitatively consistent across all four years of the study period (Supplemental Figures S2, S3, and S4). Across the four years, the maximum profit loss in the worst-case scenario (high demand, 100% *V6*, highest degree-centrality quantile, no sprays, $p_0 = 10^{-2}$) ranged from −6.7% (2015) to −10.7% (2014).

4.2.1. Impact of initial inoculum dose

The initial probability of disease (p_0) was the dominant factor determining the magnitude of profit loss and the value of fungicide intervention (Fig. 5).

Low inoculum ($p_0 \leq 10^{-4}$): When the initial probability of disease was very low, profit changes were small and insensitive to the number of fungicide sprays, out-degree centrality quantile, or pathogen diversity. In 2014, profit losses at $p_0 = 10^{-5}$ ranged from −0.1% to −1.1% under high demand and remained below −0.7% under low demand, with no systematic response to increasing spray numbers. This indicates that at sufficiently low inoculum levels, the epidemic fails to develop regardless of management inputs, and additional fungicide applications represent unnecessary costs.

Moderate to high inoculum ($p_0 \geq 10^{-3}$): As p_0 increased, profitability became sensitive to management decisions. At $p_0 = 10^{-2}$ under high demand with no sprays, profit losses ranged from −2.9% (lowest out-degree centrality quantile, 0% *V6*) to −10.7% (highest out-degree centrality quantile, 100% *V6*). Applying fungicides substantially reduced these losses: 5 sprays in May was optimal under high demand for the worst-case scenario, reducing losses from −10.7% to −4.3%. However, additional sprays beyond this optimum were counterproductive — 10 sprays yielded a loss of −5.4%, reflecting diminishing disease control benefits offset by rising fungicide costs.

4.2.2. Effect of pathogen diversity

The proportion of *V6*-virulent strains in the pathogen population strongly modulated profit outcomes because they can infect a broader range of cultivars, effectively increasing the susceptible host population. Under the most severe epidemic scenario ($p_0 = 10^{-2}$, no sprays, highest out-degree centrality quantile, high demand), increasing the *V6*-virulent proportion from 0% to 100% more than tripled the profit loss, from −3.3% to −10.7%. The relationship was monotonically increasing: intermediate proportions of 25%, 50%, and 75% *V6* yielded losses of −6.3%, −7.8%, and −9.2%, respectively. This gradient was present across all market demand levels, though attenuated in magnitude under low demand (−0.7% at 0% *V6* to −2.2% at 100% *V6*).

4.2.3. Influence of out-degree centrality of initial inoculum

Profit losses increased monotonically with the out-degree centrality quantile of initially infected yards. Under high demand at $p_0 = 10^{-2}$ with 100% *V6* and no sprays, losses ranged from −6.7% (Q1, lowest) to −10.7% (Q5, highest), with the steepest increase between Q1 and Q2 (2.5 percentage points) and progressively smaller increments thereafter. This pattern was consistent across all four years, with the Q1–Q5 gap ranging from 3.6 to 4.4 percentage points (pp). The centrality effect depended strongly on pathogen diversity: at 0% *V6* the gap was only 0.4 percentage points (−2.9% vs. −3.3%), but it widened to 4.0 percentage points at 100% *V6* as the expanded susceptible host pool enabled highly-connected yards to propagate disease more effectively.

The interaction between centrality and fungicide management was non-monotonic. From the no-spray baseline (4.0 pp gap), the gap initially widened at 1 spray (5.8 pp) because a single application suppressed the epidemic in low-centrality yards but was insufficient in high-centrality yards with more outgoing transmission pathways. With additional sprays the gap narrowed to 2.5 pp at 3 sprays and 1.2 pp at 5 sprays (−3.2% at Q1 vs. −4.3% at Q5), indicating that adequate fungicide management can largely neutralize the disadvantage of initial infections in highly-connected yards.

4.2.4. Market demand conditions

Market demand conditions shifted the magnitude of profit losses and, in some cases, the optimal management strategy. Under the most severe epidemic conditions without sprays, losses declined from −10.7% under high demand to −4.2% under moderate and −2.2% under low demand. At the optimum, 5 sprays minimized losses under high (−4.3%) and moderate (−1.8%) demand, while 4 sprays was sufficient under low demand (−1.0%). The greater sensitivity of profit to disease under high demand reflects the higher per-unit crop value and the consequent larger revenue penalty from quality defects. These patterns were consistent across all four years of the study period (Supplemental Figures S2, S3, and S4).

5. Discussion

Our analysis addresses a multifaceted challenge in agricultural disease management where epidemic control decisions must account for complex interactions between pathogen biology, management practices, market conditions, and regional-scale disease transmission. Our motivating pathosystem represents a complex scenario for economic epidemiology, combining the spread of a highly dispersible pathogen capable of long-distance transmission, potential damage to both yield and quality metrics, market-sensitive quality standards, and strain-specific cultivar resistance in a heterogeneous landscape of host connectivity. This complexity necessitates a modeling approach that simultaneously captures network-scale disease transmission, multi-dimensional crop damage functions, and market-dependent economic outcomes.

Three key findings emerge from our simulations. First, there exists a critical threshold in initial inoculum pressure (approximately $p_0 = 10^{-4}$) below which economic losses are negligible regardless of management, but above which profitability becomes acutely sensitive to early-season fungicide timing and intensity, suggesting opportunities for area-wide interventions targeting primary inoculum reduction. Second, above this threshold, the economic impact of disease is modulated by the network position of initially infected yards and pathogen diversity, with initial infections in highly-connected yards and virulent strains amplifying losses and requiring more intensive intervention. Third, quality-sensitive market conditions sustain near-maximum fungicide intensity even under low demand, with the optimal spray count falling by only one application despite substantially lower crop prices, challenging traditional economic injury level concepts when crop value is tied to quality standards rather than yield alone.

5.1. Early intervention and threshold effects

One of the most striking findings from our simulations is the threshold-like response in simulated optimal control strategies as initial inoculum probability increases. When the initial probability of disease remained below 10^{-4}, economic losses were minimal regardless of fungicide application intensity, pathogen strain diversity, or initial infection location. Above this threshold, however, the system exhibited dramatic sensitivity to early-season intervention, with the optimal number of fungicide applications in the first month becoming the dominant factor determining economic outcomes. This pattern aligns with theoretical predictions for epidemics where containment efficiency depends critically on early-stage contact rates and intervention timing (Margosian et al., 2009; Severns et al., 2019). For spatially-explicit epidemics with long-distance dispersal capabilities, small changes in primary inoculum can cascade through the transmission network, leading to nonlinear responses in final epidemic size (Cunniffe et al., 2015a). Epidemic threshold behavior has been documented across diverse host-pathogen systems, from livestock diseases (Hartfield and Alizon, 2013) to forest pathogens (Lofgren et al., 2014). Similarly, the sensitivity to early intervention aligns with the "golden hour" concept in human disease outbreak response, where rapid action in the initial stages determines overall outbreak severity (Fraser et al., 2004).

In most susceptible–infected–recovered (SIR) epidemic models, there is a critical value of R_0 below which epidemics cannot occur (Keeling and Eames, 2005; Diekmann et al., 2013). The critical value of R_0 depends on transmission and recovery dynamics, but also the structure of the host population and mixing of healthy and infected individuals. Calculation of a threshold R_0 becomes increasingly difficult with more complex epidemic models. Thresholds occur not only for epidemic development, but also for management intervention in social dilemma problems, such as collective action in the form of area-wide pest management (Lence and Singerman, 2022; Garcia-Figuera et al., 2024). Identifying participation thresholds in complex social-ecological systems is not trivial, and often participation thresholds for successful area-wide pest management are unknown due to uncertainty on factors such as transmission dynamics of the pathogen or vector, heterogeneity in host connectivity, and efficacy of the intervention effort at different scales (Garcia-Figuera et al., 2024).

Our simulation experiments with a time-transition epidemic model suggest that there exists a primary inoculum threshold effect in the motivating pathosystem and, very likely, other pathosystems where epidemics are the result of inoculum and intervention efforts that influence disease spread both within and among management units. We expect that the observed initial inoculum threshold results from the specific combination of pathogen transmission and landscape connectivity idiosyncratic to the specific conditions in the motivating pathosystem. How these specific factors influence the dynamics of landscape-level epidemics is an obvious area for future investigation.

Our study indicates that epidemic mitigation may not require intensive early-season intervention when the primary inoculum at the landscape level is sufficiently low. Indeed, we observed that a 10-fold reduction in initial inoculum from the levels observed in practice (Gent et al., 2018) could largely eliminate economic damage from powdery mildew industry-wide. Further work is needed to understand whether area-wide interventions targeting primary inoculum reduction may be more cost-effective than field-level fungicide intensification. This might involve strategic deployment of cultural control measures or host resistance that is race-nonspecific.

When the primary inoculum dose is not limiting, we show it is possible to calibrate intervention efforts to observed or expected primary inoculum dose and location. More intensive early-season intervention is warranted when primary inoculum occurs in individual fields that are more highly connected in the disease transmission network or when the initial infections are caused by virulent strains that can infect more cultivars. The importance of intervention in highly-connected nodes is

well established in network epidemiology across physical and biological systems (Pastor-Satorras and Vespignani, 2001; Cross et al., 2004). Minimizing economic loss was sensitive to the number of fungicide applications made in the earliest stage of epidemics, and this was particularly the case when primary inoculum first occurred in the most highly-connected yards. In our analysis, under severe epidemic conditions ($p_0 = 10^{-2}$ and 100% *V6* strain under high market demand), the optimal number of early-season fungicide applications was up to two higher when primary inoculum was concentrated in the most connected yards versus the least connected in the network, and even at the optimal spray level the most connected yards still incurred greater losses (−4.3% vs. −3.1%). We observed similar changes in the optimal simulated number of fungicide applications when the pathogen population increased from a single, less virulent strain to entirely a more virulent strain that could infect all cultivars. Notably, a single early-season fungicide application can be counterproductive in this context: it may suppress disease in less-connected yards while providing a false sense of adequate control in high-centrality yards where the epidemic persists through numerous transmission pathways, temporarily widening the disparity in outcomes across the network. Only with sustained fungicide pressure does this disparity narrow, eventually equalizing outcomes across centrality quantiles. Degree centrality is calculated from yard size, distance to other yards, and average wind run, features that are known or can be estimated prior to epidemic development. In some instances, pathogen virulence may be known with certainty depending on which cultivar develops disease (Gent et al., 2019b). Given this, one may estimate the management units that we predict are most critical for disease spread and therefore prioritize intensified management efforts prior to or in the earliest stages of a disease outbreak.

5.2. Market sensitivity and economic injury level paradigms

The optimal spray strategy remained largely consistent across different market demand scenarios, typically realizing 4–5 applications for interventions during the critical early epidemic period when primary inoculum pressure was substantial. This is notably more than growers' standard practices during the same period (Hwang et al., 2025). The simulated optimum number of sprays was slightly higher under high market demand conditions but decreased modestly under moderate and low demand scenarios, with low demand scenarios realizing a decrease by an average of 1 spray. Across the market demand scenarios, the highest profits were consistently associated with overtreatment rather than undertreatment. This asymmetry in marginal profit has important implications for understanding the economic consequences of suboptimal spray decisions. While overtreatment of fungicide applications led to diminishing returns and net economic losses through higher input costs, these losses from overtreatment were lesser in magnitude than the losses incurred from a modest undertreatment relative to the simulated optimum.

It is important to note that incorporating uncertainty or risk aversion into the profit maximizing decision process predicts increased applications of pesticides relative to a risk neutral grower (Feder, 1979). Furthermore, more stringent quality standards and increased uncertainty of the likelihood of pathogens also tend to increase pesticide use in crop production (Marsh et al., 2000).

This pattern reflects a broader challenge in crops where farm profit depends on meeting specific quality criteria in contracts rather than simply maximizing yield. When crop rejection or severe devaluation becomes possible, risk-averse growers may adopt more intensive management strategies that exceed the classical EIL optimization (Feder, 1979). Under high pathogen pressure in our simulations, the economic penalty for under-treatment became as severe as over-treating by two to three times the simulated optimal level. The sensitivity of private optimal strategies to market conditions has important implications for pest management recommendations and policy. Management recommendations are often targeted based on disease pressure and tacitly assume static market demand. Our results suggest that market context should also inform management decisions (Marsh et al., 2000). Furthermore, vague terms in production contracts regarding quality standards or price premiums may exacerbate this issue by increasing uncertainty and encouraging more conservative (intensive) disease intervention.

5.3. Study limitations and future directions

Several limitations should be considered when interpreting our results. We recognized that the damage functions linking disease incidence to cone color and subsequent price adjustments rely on relationships derived from literature rather than direct observation. This may introduce uncertainty in the precise magnitude of the economic effects we estimate in the damage functions. However, the directional and relative importance of the epidemic factors and management interventions we report remain valid. Ambiguous language in production contracts is problematic and also complicates model parameterization, but there are no easy solutions to this problem. Indeed, this may be the largest source of uncertainty in our modeling and also one of the most difficult to model precisely.

We focused on annual profit maximization at the landscape-level as our outcome of interest. This may be appropriate for producers with productivist ideology (McDowell et al., 2018) but ignores grower risk aversion and externalities whose costs are not borne directly by producers. We also do not identify the optimal strategy for an individual grower or an individual yard. Many other production or environmental objectives could be considered, and at time steps longer than a single crop year. Nonetheless, the coupled epidemiological-economic modeling framework we developed provides a foundation for analyzing other production objections or interventions in complex pathosystems and production scenarios. Additionally, our optimization is partial: only fungicide applications in May are treated as decision variables, while later-season sprays follow deterministically from a fitted model based on observed disease state, meaning the full seasonal spray program is not jointly optimized. The proportion of *V6*-virulent strains is also held uniform across all yards within a given simulation run, whereas in practice strain composition likely varies spatially and may shift over the course of a season. Extensions to the model to accommodate more complex pathogen race structure are possible, which may be informative since a third pathogen race has evolved since the original data acquisition (Gent et al., 2017). We expect the overall findings of this research will hold across more complex patterns of pathogen virulence conditioned by *R*-genes that provide qualitative resistance.

CRediT authorship contribution statement

Joshua F. Pedro: Writing – review & editing, Writing – original draft, Visualization, Software, Methodology, Investigation, Formal analysis. **Sharmodeep Bhattacharyya:** Writing – review & editing, Supervision, Methodology, Investigation, Conceptualization. **Shirshendu Chatterjee:** Writing – review & editing, Supervision, Resources, Methodology, Investigation, Conceptualization. **Thomas L. Marsh:** Writing – review & editing, Methodology, Investigation, Formal analysis, Conceptualization. **Jae Young Hwang:** Writing – review & editing, Investigation. **David H. Gent:** Writing – review & editing, Supervision, Resources, Project administration, Methodology, Investigation, Funding acquisition, Conceptualization.

Declaration of competing interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: David H. Gent reports financial support was provided by National Institute of Food and Agriculture. Sharmodeep Bhattacharyya reports financial support was provided by National Institute of Food and Agriculture. Thomas L. Marsh reports financial support was provided by

National Institute of Food and Agriculture. If there are other authors, they declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary material related to this article can be found online at https://doi.org/10.1016/j.agsy.2026.104792.

Data availability

Code and data available at: https://github.com/joshfpedro/hops-project-2025.

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