

## ARTICLE

# Developing practical measures of the price of pesticide resistance: A flexible computational framework with global sensitivity analysis

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## Abstract

Pesticide resistance poses an increasing challenge for agricultural sustainability. Pesticide susceptibility is a depletable biological resource, but resistance management rarely quantifies marginal, forward-looking economic costs to users of depletion. To facilitate the development of such costs, we use a generic stochastic bioeconomic model of resistance evolution in a crop pest population, stochastic dynamic programming, and global sensitivity analysis to analyze the “marginal user costs” of resistance. The most impactful parameters are population density dependence and pesticide prices. The least impactful is the fitness cost of resistance, which is noteworthy because of prior emphasis on this parameter in the resistance management literature.

## KEYWORDS

pesticide resistance, population genetics, resource valuation, sensitivity analysis

## JEL CLASSIFICATION

Q11, Q18, Q20, Q57

## 1 | INTRODUCTION

Evolution plays a critical role in the control of insect pests and weeds. Despite pesticides continuing to be essential inputs in agriculture, resistance in major agricultural pests has arisen to every major pesticide (Gould et al., 2018) and poses a challenge to future agricultural sustainability (Living with

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Resistance Project, 2018). For many years, holistic management paradigms, namely insect resistance management (IRM), integrated pest management (IPM), and integrated weed management (IWM) have been developed to help farmers decrease the burden of pesticide resistance (Abrol & Shankar, 2012; Korres et al., 2019; Onstad, 2013; Onstad & Crain, 2019). A key component of these practices is to diversify control tactics and to base pesticide applications on information about the pest population and resistance levels, to both limit the near-term growth of the pest population and long-term selection for further resistance.

Yet, the effective uptake of these practices among farmers has remained low (Alwang et al., 2019; Gould et al., 2018; Hurley & Frisvold, 2016; Lefebvre et al., 2015; Moss, 2019). Economists have identified the common-pool nature of resistance as one reason for this (Miranowski & Carlson, 1986). Common-pool resources create a dilemma for collective action, in which individual resource users have an incentive to consume the resource beyond what would be collectively optimal. In the case of resistance, the resource is the level of pesticide susceptibility in the pest population.

The economist's prescription for collective resource problems is to institute incentives that result in self-interested resource users choosing levels of consumption that are socially optimal. Such incentives all rely explicitly or implicitly on putting a current price on the resource that captures the future benefits we lose if we consume one unit of the resource today. This price is practical, in that its value can be used as an incentive directly by adding it as a markup to the market price of the resource under previously open-access conditions, or as an important indicator of the efficiency of a quantity-based permit or quota trading system. In other collective action problems, including climate change (Cai & Lontzek, 2019), fisheries management (Pascoe et al., 2016; Yun et al., 2017), and regional water and air pollution (Keeler et al., 2016; Keiser & Muller, 2017), the computation of such prices using dynamic integrated assessment models (IAMs) is proving instrumental for effective resource policy. These resource prices come by various names, depending on the application: the social cost of carbon (SCC, in climate change), the social cost of nutrient pollution, marginal social value, shadow prices, or marginal user costs (MUCs) (in the general resource economics literature; Clark, 2010; Conrad, 2010). In each case, these prices are advanced as summary economic measures that can be used to inform and design more economically efficient policies.

While there is a rich history of dynamic bioeconomic modeling of pesticide resistance (Brown et al., 2013; Feder & Regev, 1975; Grimsrud & Huffaker, 2006; Hueth & Regev, 1974; Hurley et al., 2001; Laxminarayan & Simpson, 2002; Livingston et al., 2004; Qiao et al., 2008), results from these bioeconomic models still rarely inform the governance of pesticide resistance in any technically substantive way. This contrasts notably with other topics in resource economics, for example, climate change, where IAM-based SCC estimates have seen extensive implementation in policy (Pizer et al., 2014). Recently, however, there have been multiple calls for the use of such applied economic analyses in resistance management: Gould et al. (2018) point to a lack of quantified user costs of resistance as an important barrier to effective governance. An EPA scientific advisory panel (Environmental Protection Agency, 2018) noted that key policy questions regarding the permitting of transgenic pesticidal crops be submitted to benefit–cost analysis that consistently accounts for the costs of resistance (and the benefits of pest reduction) over time. The World Health Organization (2021) has noted that a similar approach would be useful in accounting for insecticide resistance in public health programs to control disease-transmitting mosquitoes. Moreover, resistance management policy is ripe for direct input from bioeconomic modeling because biomathematical modeling is already being used to inform IRM (e.g., Martinez & Caprio, 2016) but without quantitative economic and social objectives built into these models.

There are many potential reasons for the gap between the concepts analyzed in the economics literature and the practical implementation of a bioeconomically informed pesticide resistance policy (Hurley & Frisvold, 2016). In the US context, Masur and Posner (2015) argue that the executive branch shows a general reluctance to implement Pigouvian taxes, despite apparently

having the authority to do so. This reluctance appears even greater for Pigouvian taxes on farmers (Shortle & Uetake, 2015). In European countries that have implemented Pigouvian taxes on pesticides—Denmark, Norway, Sweden, Germany—these taxes have been limited to correcting external costs on the environment and human health, and have been effective at doing so, but have not been used to address resistance concerns (Böcker & Finger, 2016, Finger et al., 2017, Nielsen et al., 2023).

Zilberman and Millock (1997) note that multidimensionality and heterogeneity are the most challenging components of developing a practical financial incentive for resistance management. This is despite the fact that implementation of corrective incentives for resistance management should theoretically be easier than for pesticide-related environmental externalities since pesticide resistance imposes more direct—albeit collective and delayed—production costs on farmers than do environmental externalities. This recognition points to another important potential role for practical measures of resistance costs: as a communication device for farmers. In our own experience, a significant amount of agricultural extension efforts among entomologists and weed scientists is devoted to resistance management, yet little attempt is made in these extension activities to quantify and communicate to farmers the expected, collective economic benefits to farmers from adopting resistance management recommendations. Of course, communicating these quantities requires both a scientifically credible and practical framework for their computation.

Given this state of research and policy on pesticide resistance, this paper seeks to develop a better understanding of how the user costs of resistance are potentially determined by the interactions of heterogeneous bioeconomic factors that vary by context. We provide the first systematic numerical analysis of model-based user costs of pesticide susceptibility, that is, the price of resistance, and their variation across different bioeconomic contexts. We extend a generic bioeconomic model of insecticide resistance developed by Hurley et al. (2001), which was originally developed for the management of resistance in European corn borer (ECB; *Ostrinia nubilalis*) to transgenic pesticidal corn, which contains toxin-producing genes from *Bacillus thuringiensis* (Bt) species of bacteria. The basic structure of this model and methods for analyzing it have been revisited and extended in various ways in subsequent research. In particular, Grimsrud and Huffaker (2006) and Qiao et al. (2008) conduct dynamic optimization of versions of this model structure, using optimal control theory. This generic framework, which models resistance as a single gene mutation and in which individual pests can be either homozygous resistant or susceptible or heterozygous, has also been applied to model the evolution of glyphosate resistance in major weed species in US cropland (Gustafson, 2008; Livingston et al., 2015). However, a large-scale sensitivity analysis (SA) of this framework has never been conducted, so far as we know.

In this paper, we therefore first extend this work methodologically by incorporating environmental stochasticity into the pest population dynamics (e.g., via weather shocks) and by using stochastic dynamic programming (SDP) as our optimization method, and then subject the model to a computationally intensive global SA using large ranges for model parameters. The SDP approach allows us to examine optimal pesticide use levels and user costs not just as calendar-based schedules (which are of limited use in a stochastic setting), but in terms of how these quantities depend on the current state of pest population and resistance. Our global SA is significant because, whereas Hurley et al. use parameters specific to Bt resistance in ECB, we present this paper's findings as more broadly relevant for the economics of resistance management in general.

Our analysis first confirms a claim made by Feder and Regev (1975), but rarely discussed since, regarding the theoretically ambiguous sign on the MUCs of pesticides. This ambiguity arises because of the tension between using more pesticides early to limit population growth and spread (i.e., greater areawide pest suppression, as highlighted by Hutchison et al., 2010 and Dively et al., 2018, among others) versus using less pesticide early on to delay the evolution of resistance. Our extension to a stochastic model of resistance also reveals that the *steady-state* distribution of these MUCs includes both positive and negative values, depending on the current state of the pest population density and resistance. This is important because it suggests that even the qualitative policy question

of whether to tax or subsidize pest control in the steady state still requires dynamic updating based on the current state of the population genetics.<sup>1</sup>

Findings from global SA indicate that parameter-induced variations in dynamically optimal pesticide use and the “price of resistance” are most strongly determined by density dependence of pest population growth, the contemporaneous costs of the pesticide, genetic dominance of resistance mutations, and the economic discount rate. Other biological and economic parameters included in the SA but that have negligible effects on these outcomes are: the (biological) fitness costs of resistance, the degree of stochasticity in the pest population, and the correlation of this stochasticity between different pest genotypic subpopulations. The lack of model sensitivity with regard to fitness costs is especially notable, given that this parameter controls whether pesticide susceptibility is a renewable or nonrenewable resource and has been highlighted in previous bioeconomic analyses as a key factor determining the nature of optimal IRM (Laxminarayan & Simpson, 2002). The next section below details the bioeconomic model and how we analyze it. We then present and discuss results, before concluding with a discussion about how these findings help advance the use of forward-looking model-based estimates of resistance costs in IRM policy.

## 2 | THE BIOECONOMIC MODEL AND SENSITIVITY ANALYSIS

The bioeconomic model used in this analysis combines a stochastic biomathematical model of pesticide resistance evolution with a model of the flow of net economic damages from the pest, within an agricultural, crop cultivation context where the objective is to maximize the expected net present value (ENPV) of farm profits. To solve this optimization problem, we use SDP. The SDP solutions provide the optimal pesticide use level as a function of the current state of the pest population and resistance levels. We also use the SDP solutions to derive the marginal net costs of pesticide resistance. Full details of these components are provided in the Supporting Information.

We adopt a two-state, discrete-time model of resistance to a single crop and single pesticide, in which resistance is generated from a single gene mutation  $R$  relative to the original wild-type gene  $S$  that left the pest susceptible to the pesticide. Hurley et al. (2001) were the first to apply the most basic form of this model in agricultural economics, in a case study of ECB resistance to Bt crops. That implementation treated pest susceptibility to Bt as a nonrenewable resource that evolved following a standard population genetics model according to the relative fitness of the three relevant genotypes:  $SS$ ,  $RS$ , and  $RR$ . Subsequent bioeconomic models (e.g., Grimsrud & Huffaker, 2006; Laxminarayan & Simpson, 2002; Qiao et al., 2008) included fitness costs of the  $R$  mutation, thereby permitting pest susceptibility to regenerate and converting the problem from one of a nonrenewable resource to that of a renewable resource. In addition to modeling resistance evolution, all of these bioeconomic models track the dynamics of the overall pest population, since this is a critical input into the economic models of crop damage and thus a key component of determining the economic efficiency of different resistance management policies. Purely biological models of resistance that are used in forming most IRM policies have typically focused only on the prevalence of the resistance mutation and often ignored overall pest densities (e.g., Martinez & Caprio, 2016).

The biological model tracks the state of the total pest population density  $N_t$  and the frequency  $p_t$  of the resistance gene allele within that population at time  $t$ . Gathering the biological state variables in the vector  $\mathbf{x}_t = [N_t \ p_t]$ , the dynamics of these state variables can be summarized as a generic stochastic Markov process  $\mathbf{x}_{t+1} = F(\mathbf{x}_t, q_t, \epsilon_t, \theta_F)$ , where  $F(\cdot)$  is the transition function,  $q_t$  is the pesticide application level,  $\epsilon_t$  is a vector shock terms with probability density function  $f(\epsilon_t | \theta_\epsilon)$ , and

<sup>1</sup>Note that we focus here on the farm-production user costs of resistance excluding environmental externalities, a limitation we discuss at the end of the paper.

$\theta_x = [\theta_F \ \theta_\epsilon]$  are parameter vectors governing the transition dynamics and distribution of shocks. In brief, higher pesticide use  $q_t$  decreases  $N_t$  when  $p_t$  is low, but also selects for increasing  $p_t$ . At high  $p_t$ ,  $q_t$ 's effects on  $N_t$  diminish. As detailed in the Supporting Information, these effects are controlled by the parameters in  $\theta_x$ , which include the relative effectiveness of the pesticide at killing SS genotypes, potential fitness costs of RR genotypes, and the genetic dominance of the resistance gene in RS genotypes, as well as the variances and covariances of the shocks to each genotype (with  $\epsilon_t$  being distributed mean-zero multivariate normal).

The pest population's effects on farm profits are summarized via the per-period profit function  $\pi(x_t, q_t, \theta_\pi)$ , where  $\theta_\pi$  is a vector of parameters in this model and with its formulation detailed in Supporting Information: In brief, higher pest densities  $N_t$  decrease profits and higher pesticide applications  $q_t$  increase yields but are more costly with an assumed price/marginal cost of application of  $w$ . Higher resistance levels  $p_t$  decrease the marginal productivity of the pesticide input  $q_t$ .

In our model, we define the net MUCs of resistance as the net adjustment factor to the marginal cost of the pesticide that would induce a self-interested farmer ignoring pest population dynamics and evolution to make a decision in the collective interest of farmers as a whole (see detailed rationale for this definition in Supporting Information). The collectively optimal outcome is characterized as a solution to the following discrete-time, infinite-horizon Bellman equation

$$V(x, \Theta) = \max_{q \in [0, \bar{q}]} \{ \pi(x, q, \theta_\pi) + \beta \mathbb{E}[V(x', \Theta) \mid x, q, \theta_x] \}, \quad (1)$$

where  $V(x, \Theta)$  is the maximized expected NPV of profits given the current state  $x$  of the pest population and aggregated parameter vector  $\Theta = [\theta_\pi \ \theta_x]$ ,  $x'$  is the uncertain next-period state,  $\beta \in (0, 1)$  is the discount factor, and  $\bar{q}$  is the upper bound on pesticide use (e.g., if the input is specified as the fraction of acreage treated then  $\bar{q} = 1$ ). When an interior level of pesticide use is optimal, then the first-order condition (FOC) holds

$$0 = \left[ \frac{\partial \pi}{\partial q} + \beta \frac{\partial \mathbb{E}[V(x', \Theta) \mid x, q, \theta]}{\partial q} \right]_{q=q^*(x, \Theta)}. \quad (2)$$

A myopic, self-interested farmer ignoring biological dynamics in this model would simply choose  $q$  to maximize profits in each period, that is,  $q^o(x, \theta_\pi) = \arg \max_{q \in [0, \bar{q}]} \pi(x, q, \theta_\pi)$ , with FOC is  $0 = \partial \pi / \partial q|_{q=q^o(x)}$ . The net MUC of resistance therefore brings the FOC of the self-interested farmer in line with the collective interest. Thus,

$$MUC(x, \Theta) = -\beta \frac{\partial \mathbb{E}[V(x') \mid x, q]}{\partial q} \Big|_{q=q^*(x, \Theta)}. \quad (3)$$

Imposing this MUC as a price-based policy incentive would bring the myopic, self-interested farmer's FOC in line with that of the collective optimum. Note that this MUC may be positive or negative. To see this, the MUC can be decomposed using the Chain Rule

$$MUC(x, \Theta) = -\beta \mathbb{E} \left\{ \left( \frac{\frac{\partial V}{\partial N'}}{\text{pop. suppression gains}} \frac{\partial N'}{\partial q} + \frac{\frac{\partial V}{\partial p'}}{\text{resistance costs}} \frac{\partial p'}{\partial q} \right) \Big| N, p, q \right\}. \quad (4)$$

Additional pesticide decreases population growth ( $\partial N'/\partial q < 0$ ) and increases the evolution of resistance ( $\partial p'/\partial q > 0$ ), and in general the marginal values of higher pest pressure and more resistance are negative ( $\partial V/\partial N, \partial V/\partial p < 0$ ). Therefore, if the MUC in Equation (4) is positive, it must be due to the expected profit losses from increased future resistance, whereas if it is negative then the dynamic incentives to limit future population growth dominate those to limit resistance.

Because the SDP model above cannot be solved analytically, we rely on numerical analysis. For numerical specification, we initially parameterize this model for the well-studied case of potential resistance in ECB to pesticidal Bt corn. This parameterization is described in the Supporting Information.

However, our main goal in this paper is to explore the behavior of the SDP solutions across a large extent of the parameter space that hopefully captures a wide range of other possible contexts outside of the ECB-Bt case. The full set of model's 11 parameters in  $\Theta$  is summarized in Table 1, seven of which are varied in SA (with rationale for this selected subset  $\Theta^*$  provided in the Supporting Information).

We apply global SA methods described by Saltelli and Annoni (2010), Saltelli et al. (2010), and Saltelli (2008). We conduct Latin hypercube sampling of 78,125 uniformly distributed points in the seven-dimensional parameter space, with the bounds listed in Table 1. At each point, we solve the SDP problem in Equation (1). From this large volume of numerical output, we compute sensitivity indices for each parameter. These include an index constructed from standardized ordinary least squares (OLS) regression coefficients, as well as first, second, and total order Sobol indices. The formulas for these indices and their interpretation are detailed in the Supporting Information. Together, they are used to assess the relative importance of each parameter and interactions in determining model output, as well as the extent of nonlinearity in parameter effects.

TABLE 1 Model parameters.

Parameter	Notation	Units	Minimum	Maximum
<i>Economic parameters</i>				
Corn price	$P$	\$ per Mg	100	100
Maximal yield	$\bar{Y}$	Mg per ha	12	12
Bt price premium	$w$	\$ per ha	\$1	\$50
Economic discount factor	$\beta$	\$ next year/\$ this year	0.85	0.99
<i>Biological parameters</i>				
Density dependence	$K$	Larvae per 100 plants	25	400
Maximal net growth rate	$r$	% per year (/100%)	0.791	0.791
Pesticide effectiveness	$\mu_s$	% per year per 1% Bt	2.28%	2.28%
Dominance of resistant gene	$h$	[weight]	0	1
Fitness cost of resistance	$\lambda$	% per year (/100%)	0	0.15
Variance of shocks	$\sigma^2$	[% per year (/100%)] <sup>2</sup>	0.1	1.5
Correlation of shocks	$\rho$	Covariance/variance	0	0.99

Note: Gray rows are parameters not varied in sensitivity analysis, with reasonable values obtained from Hutchison et al. (2010) for ECB Bt resistance case. See the Supporting Information for full parameter descriptions.

Abbreviations: Bt, *Bacillus thuringiensis*; ECB, European corn borer.

### 3 | RESULTS

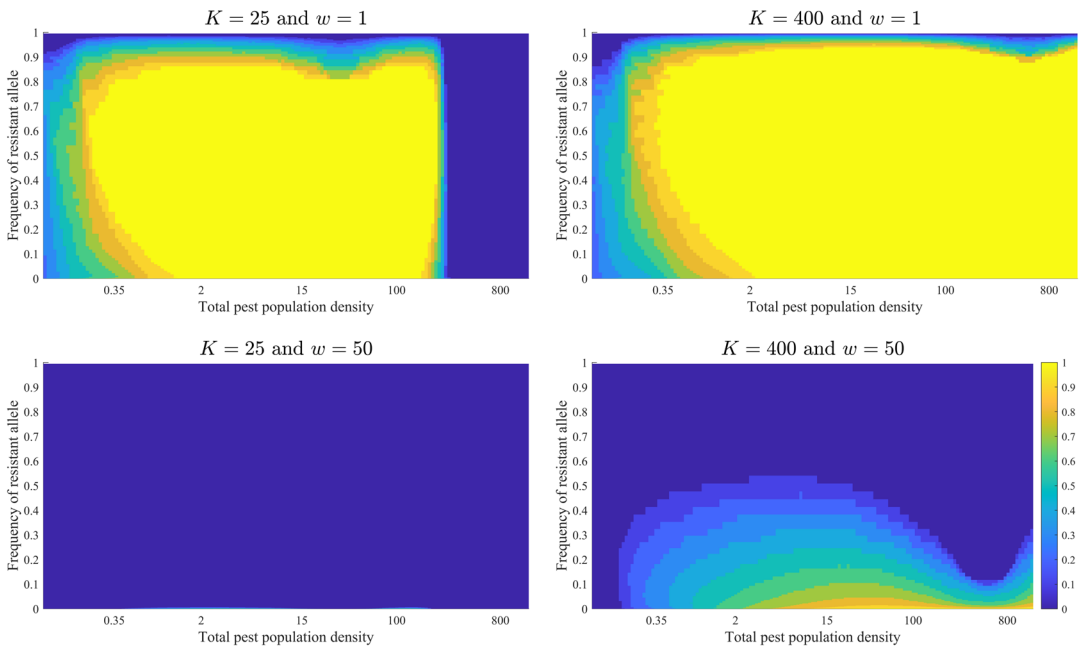
Table 2 reports the first-order effects revealed by the SA. The optimal pesticide policy (averaging over the state space) exhibits the highest first-order sensitivity to density dependence, pesticide costs, and the genetic dominance of resistance. The  $R^2$  from OLS shows that the linear correlation coefficients only capture 35% of the variation in optimal Bt use, whereas the sum of the first-order Sobol indices captures 61% of the variation. This means that there is a lot of nonlinear dependency between optimal pesticide use and the parameters since the first-order Sobol indices capture univariate nonlinearities not captured by OLS.

Figure 1 provides visualizations of the shape of the policy function in the state space, and examines in more detail how the nature of the optimal policy varies with key parameters. The figure shows an intuitive relationship between pesticide use and current levels of resistance, with greater resistance associated with lower optimal pesticide use. Meanwhile, the figure shows important nonmonotonicities between pesticide use and pest density, as well high dependence on key parameters (as implied by Tables 2 and 3). For a fixed level of resistance, optimal pesticide use is nonmonotonic in pest population density. Visually, we find that the easiest way to interpret these plots is, for a given level of pest density, to focus on the threshold level of resistance above which it is no longer optimal to use the pesticide and to examine how this threshold changes across different pest density levels. At low and high levels of pest density, it is generally optimal to sustain pesticide use even at higher levels of resistance. At medium-density levels, however, optimal pesticide use is more conservative. These medium-density levels are in the region of the approximate carrying capacity  $K$  of the population (which is 25 larvae per 100 plants in the left column of the figure and 400 larvae per 100 plants in the right column). The optimal policy is therefore responding to the effects of density dependence: When densities are far below carrying capacity, uncontrolled population growth is likely to be high, and it is therefore optimal to use more pesticides early, to limit future pest growth and damage, even if that means increasing resistance. However, around carrying capacity, the pest population is naturally self-limiting, and little future growth is expected; therefore, optimal pesticide use is more conservative since its marginal effect on future population growth is limited. When current densities are higher than carrying capacity (which is possible in this

**TABLE 2** Standardized OLS regression coefficients, first-order, and total Sobol indices.

Parameters	Optimal Bt use			MUCs		
	OLS coefficients	First-order Sobol	Total Sobol	OLS coefficients	First-order Sobol	Total Sobol
Economic						
$\beta$	-0.0990	0.0176	0.1103	<b>-0.1446</b>	<b>0.0540</b>	0.1498
$w$	<b>-0.4737</b>	<b>0.3172</b>	<b>0.6390</b>	<b>0.5167</b>	<b>0.2854</b>	<b>0.4035</b>
Biological						
$h$	<b>-0.1632</b>	<b>0.0342</b>	<b>0.1779</b>	-0.0382	0.0302	<b>0.1567</b>
$K$	<b>0.2862</b>	<b>0.2353</b>	<b>0.5166</b>	<b>-0.1081</b>	<b>0.3530</b>	<b>0.4928</b>
$\rho$	-0.0853	0.0082	0.0793	-0.0842	0.0125	0.0651
$\sigma^2$	-0.0156	0.0019	0.0462	-0.0216	0.0084	0.0588
$\lambda$	-0.0034	0.00004	0.0124	0.0158	0.0022	0.0244
Variation explained	0.3503	0.6143		0.3088	0.7458	

Note: Averages over state space reported. Numbers in bold are the three largest-magnitude values in each column. Abbreviations: Bt, *Bacillus thuringiensis*; MUC, marginal user cost; OLS, ordinary least squares.



**FIGURE 1** Optimal pesticide policy function, varying pesticide cost ( $w$ , \$/ha) and density dependence ( $K$ , larvae/100 plants). Color axis is fraction of corn planted using *Bacillus thuringiensis* trait. Value of other parameters in this simulation:  $h = 0.5$ ,  $\beta = 0.92$ ,  $\sigma^2 = 0.8$ ,  $\rho = 0.495$ ,  $\lambda = 0.075$ .

model due to stochastic fluctuations), then the near-term expedient to limit large-scale damage becomes most pressing and optimal pesticide use is higher as a result.

Figure 1 also shows how the shape of the optimal policy function changes with respect to the two most impactful parameters found in the SA: density dependence ( $K$ ) and pesticide costs ( $w$ ). As expected, these parameters have opposing effects on optimal pesticide use (as implied by the signs of their correlation coefficients in Table 2), with cost predictably decreasing the area of the state space over which it is optimal to use the pesticide (and decreasing optimal intensity levels) and carrying capacity having the opposite effect. These effects compound: With both increased costs and reduced carrying capacity (i.e., higher density dependence), it is never optimal to use the pesticide at any point in the state space. The importance of this compounding effect suggested in Figure 2 is confirmed in Table 3, which shows the second-order Sobol indices for two-way parameter interactions. This table largely mirrors Table 1, in that the parameters with the highest first-order effects are also those with the highest second-order effects. The most influential two-way interaction on optimal pesticide use is the  $w \times K$  effect. Turning to the user costs of resistance, Table 3 shows the MUC sensitivity exhibits similar patterns as with optimal pesticide use. The only notable difference is that here the discount factor is more influential than genetic dominance, which is intuitive given that discounting has direct effects on evaluating the ENPV of future pest damages.

Table 2 also presents the mean values for the total Sobol index with respect to the outputs for each of the parameters varied. As in Tables 2 and 3, the magnitudes of the total Sobol indices can be interpreted as the fraction of the variance in the output explained. Qualitatively, the results here regarding optimal Bt use match those in Table 1, with variations in  $w$  explaining the most variation, followed by  $K$  and then  $h$ . Index values with respect to the MUC show a slightly different qualitative pattern compared to the first-order indices in Table 2: Here, total variation including interactions in the genetic dominance of resistance  $h$  explains slightly more variation in the MUC than does the discount factor  $\beta$ , which was found to be more influential in the first-order indices (Table 2).



TABLE 3 Second-order Sobol indices of interaction effects.

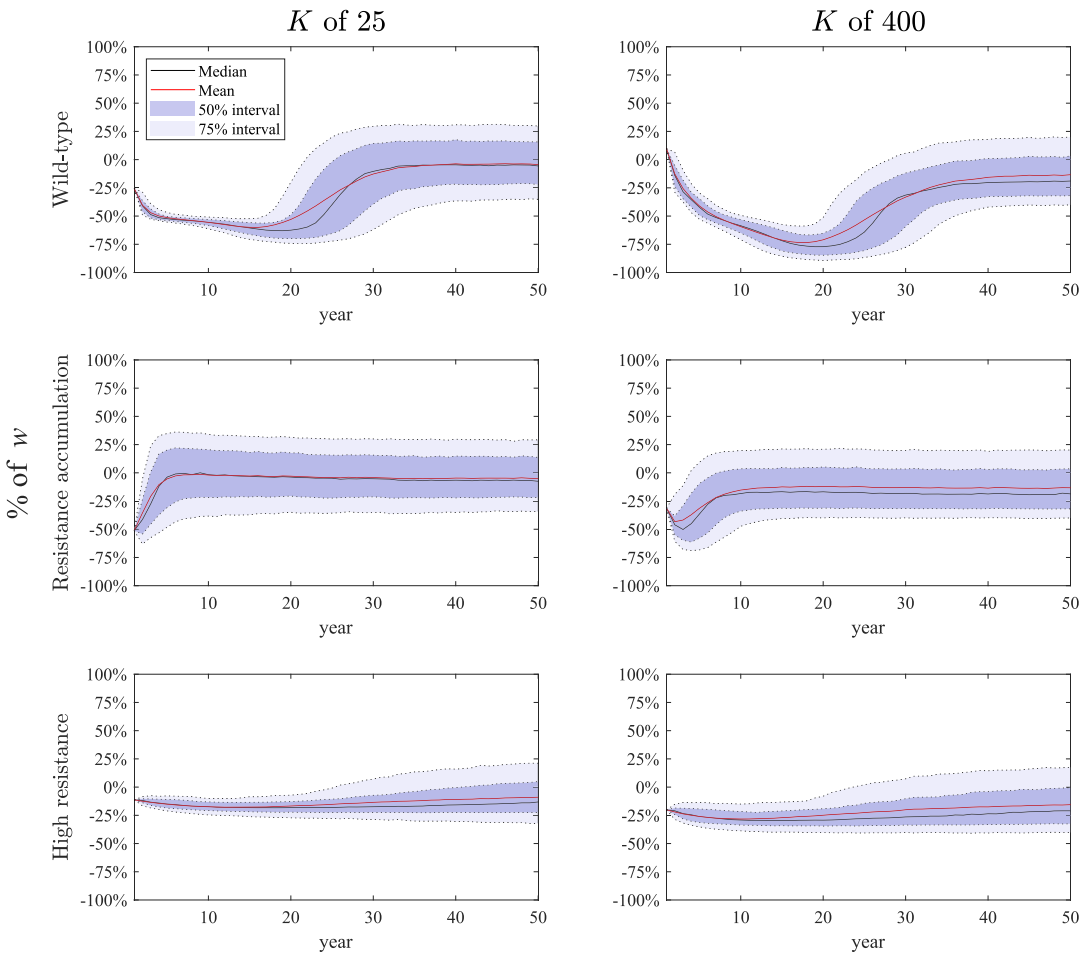
	$\beta$	$K$	$h$	$w$	$\sigma^2$	$\rho$
Optimal Bt use						
$\beta$						
$K$	0.0162					
$h$	0.0011	<b>0.0204</b>				
$w$	0.0173	<b>0.1459</b>	<b>0.0387</b>			
$\sigma^2$	0.0005	0.0015	0.0019	0.0019		
$\rho$	0.0021	0.0040	0.0039	0.0114	0.0018	
$\lambda$	0.0003	0.0001	0.0001	0.0004	0.0001	0.0001
Variation explained	0.2698					
MUC						
$\beta$						
$K$	<b>0.0247</b>					
$h$	0.0075	<b>0.0342</b>				
$w$	0.0172	0.0222	<b>0.0268</b>			
$\sigma^2$	0.0015	0.0111	0.0052	0.0076		
$\rho$	0.0040	0.0036	0.0088	0.0052	0.0018	
$\lambda$	0.0027	0.0014	0.0010	0.0044	0.0002	0.0001
Variation explained	0.1911					

Note: Numbers in bold are the highest three in each matrix.

Abbreviations: Bt, *Bacillus thuringiensis*; MUC, marginal user cost.

However, the difference in the  $\beta$  and  $h$  indices with respect to the MUC is small in both the first-order and total Sobol indices, leading to our interpretation that these parameters are roughly equivalent in the magnitude of their influence on the MUC. However, the total indices are much larger than their first- and second-order counterparts (and their sum), suggesting the importance of higher levels of interaction effects among the parameters.

To further visualize how the MUC is affected by these parameters, and to examine the model from another aspect, Figure 2 shows Monte Carlo simulations of MUC values over time for three different scenarios with different initial conditions regarding pesticide resistance, corresponding to each row of subplots in the figure. Supporting Information S1: Figure A1 shows the corresponding MUC function for these scenarios plotted in the state space. In Figure 2, the first row corresponds to a “wild-type” scenario in which the pest population has almost no resistance and is around its carrying capacity. This scenario is supposed to capture a situation in which the policymaker has a brand new, effective pesticide at hand, and wishes to institute an efficient incentive to effectively manage the evolution of resistance to the new product. The second row corresponds to a “resistance accumulation” scenario, in which resistance has arisen and accumulated somewhat but has not yet limited the effectiveness of the pesticide so that the overall population is still kept low. Finally, the third row corresponds to “high resistance” scenario in which resistance is so high that it has eroded the effectiveness of the pesticide. The pest population carrying capacity  $K$  is varied between subplot columns.



**FIGURE 2** Marginal user cost simulations, varying density dependence ( $K$ , across columns, larvae/100 plants) and initial conditions (across rows). Value of other parameters in this simulation:  $h = 0.5$ ,  $w = 25.5$ ,  $\beta = 0.92$ ,  $\sigma^2 = 0.8$ ,  $\rho = 0.495$ ,  $\lambda = 0.075$ . Time in years. MUC as % of pesticide cost,  $w$ .

Figure 2 plots simulated MUC timepaths expressed as a percentage of pesticide costs  $w$ .<sup>2</sup> Across all subplots of the figure, the mean and median of the MUC is nearly always negative, indicating—as per Equation (3)—that in this *hypothetical* scenario, the marginal dynamic gains from the pesticide suppressing population growth generally outweigh the marginal costs of increased resistance. Recall that negative MUC values imply it would be optimal to subsidize pesticide use beyond what a myopic farmer would choose to use on their own. The “wild-type” scenario with initially low resistance exhibits the most dynamic variation in the MUC, initially decreasing sharply (especially in the case when carrying capacity is high). In contrast, the “resistance accumulation” and “high resistance” scenarios exhibit more stability in the mean/median MUC over time. This would suggest that allowing for dynamic adjustments in IRM policies may be most relevant early in the introduction of a new pesticide, and that a time-invariant policy (which better reflects the current reality of Bt refuge mandates) may be more innocuous once resistance has accumulated to high

<sup>2</sup>The  $w = \$25.50$  per ha value used in these scenarios (see Figure 2 caption) is the value that has previously been estimated to pertain to Bt traits in corn for corn (e.g., Hutchison et al., 2010).

levels. However, a caveat to this interpretation is that the stationary distribution of the MUC, at the end of the 50-year time horizon, still shows significant variation, with the interquartile range containing positive MUC values, meaning that optimal policy—even in equilibrium—still responds to stochastic conditions. This provides a more nuanced picture of the sign of the MUC compared to the deterministic optimal control model of Feder and Regev (1975): In their model, the sign of the MUC is fixed in the steady state, determined by the balance between pest suppression and resistance dynamics, whereas in our stochastic model the MUC can be both positive and negative in its stationary distribution, depending on the current conditions of the overall pest population and resistance. Just as with Figure 1, it is important to emphasize that these outputs pertain to a single parameter set (varying only the carrying capacity  $K$ ). So, this output is not presented as a general argument to subsidize pesticide use, but rather as an illustration of the range of possible output from the model.

The fitness cost parameter  $\lambda$  is consistently the lowest impact parameter on the model outputs. Its Sobol indices, in Tables 2 and 3, explain a miniscule fraction of variation in optimal pesticide use and MUC. This means that in this model the optimal resistance management policy is insensitive to the degree of fitness costs in resistant pests. Fitness cost effects (or, evidently, lack thereof) are noteworthy because of the general attention paid to these parameters in resistance management (Gassmann, 2023; Gassmann et al., 2009), as well as theoretical results from Laxminarayan and Simpson (2002) finding that the relative magnitude of fitness costs versus the discount rate should determine whether sustaining any pest susceptibility, in the long run, is optimal.

We also find that the model output is insensitive to the parameters  $\sigma$  and  $\rho$  governing the degree of stochasticity. The total Sobol indices in Table 2 show that, even when accounting for all possible interactions with the other varied parameters, these two parameters each can explain no more than 8% of the total variation in the optimal policy and MUC functions. This finding has practical relevance for resistance management because it suggests that, even when information is lacking on the degree of stochasticity in the population and resistance dynamics, few costs would be incurred from employing an optimal policy function with feedback (but *not* a predetermined calendar-based schedule) from a deterministic model of resistance (e.g., Livingston et al., 2015).

Finally, to illustrate the practical utility and interpretability of the MUC, we provide possible point estimates for the MUC specifically for the Bt-ECB case analyzed by Hutchison et al. (2010). Supporting Information S1: Table A3 shows MUC point estimates for different values of the discount factor, and different initial resistance levels, ranging from  $p_0 = 1 \text{ e-}4$  to  $p_0 = 0.4$ . Note that there is considerable interest in resistance management in distinguishing and monitoring variations at very low levels of resistance since these seemingly small differences in initial conditions can lead to large differences in the time until economically consequential resistance levels arise (Andow & Ives, 2002; Environmental Protection Agency, 2018). For illustration, we calculate these scenarios mainly for the Minnesota, 1996–2009 ECB larval data from Hutchison et al. (2010) using parameter values specific to that context, assuming initial ECB density is at its average over that time period: 30 larvae per 100 plants in non-Bt fields. For this scenario, when the discount rate is low at  $\approx 1\%$ , then if the frequency of the resistant gene allele is 1 in 1000 ( $p_0 = 1 \text{ e-}3$ ), then the MUC is  $a + 60\%$  the marginal cost of the Bt trait (assumed at \$25.50/ha in Hutchison et al., 2010), meaning the collective importance of limiting resistance dominates that of limiting population growth, whereas when the resistant gene frequency is higher, at one in 100, then the MUC amounts to  $-77\%$  the cost of the Bt trait, meaning collective incentives to limit population growth dominate those associated with resistance. Interestingly, this change in sign of the MUC between  $p_0 = 1 \text{ e-}3$  and  $p_0 = 1 \text{ e-}2$  is exactly the range in which Andow and Ives (2002) located the minimum resistance detection level ( $p^* = 5 \text{ e-}3$ ) needed to achieve an effective resistance management response. However, at higher discount rates (5% or 15%), the MUC is always positive, meaning that collective incentives to limit resistance always dominate.

## 4 | DISCUSSION

This paper analyzes a candidate metric for the forward-looking, model-based estimates of the costs of pesticide resistance, as measured by the efficient level of adjustment to pesticide prices to induce collectively optimal use across the farm. Such a measure would improve on existing estimates of the costs of resistance, which typically take a back-of-the-envelope approach tallying up retrospective increases in pest damages or pesticide expenditures over time (e.g., Palumbi, 2001). The “price of resistance” concept we propose is more appropriate for communicating (a) to policymakers the degree of correction for the open-access market failures associated with pesticide resistance in conjunction with the local public good of pest suppression and (b) to pesticide users the magnitude of expected future consequences of not addressing these market failures, in practical terms (i.e., relative to the \$ cost of frequently used inputs).

While this analysis provides important information for moving the MUC of resistance from theory to practice, additional work remains to put into practice. Such an agenda is analogous to the decades of research on climate-economy IAMs that led to the development of SCC estimation, as well as ongoing research developing IAMs for the social cost of water pollution (Keiser et al., 2020). To be credible one would theoretically have to estimate a MUC for each pesticide, for each pest targeted by that pesticide, and for each agroecological zone. Moreover, the MUC is dynamic and would require regular updating with the current state of resistance and pest density.

Although this may strike readers as a daunting effort, we note that the use of pest-specific economic injury/action thresholds is a key pillar of IPM. These thresholds have been widely calculated and used by agricultural extension entomologists and specialists for communicating IPM recommendations to farmers (e.g., Higley & Pedigo, 1996; Ramsden et al., 2017). Recent calls by IPM experts for the significant need to update these economic thresholds (Leather & Atanasova, 2017; Peterson et al., 2018; Ramsden et al., 2017) provides an opportunity to improve the underlying methodology for calculating the economic impacts to farms of farmers' pest control decisions. Writing in *American Entomologist*, Peterson et al. (2018) call for both updated economic/decision tools and a renewed focus on resistance and evolution to reinvigorate IPM adoption.

Were MUCs calculated for a panel of pesticides  $\times$  pests  $\times$  location cases, it is unclear whether communicating this information directly to pesticide users would improve outcomes. There is some reason to be cautiously optimistic in this regard, despite the incentives for individual pesticide users to free ride on others' actions. Kotchen and Segerson (2020) review several studies on collective payments for ecosystem services given to groups of farmers or resource users. These studies generally find that collective rewards are most effective in groups with strong social ties and open communication channels. Transferring these conclusions to the context of resistance management suggests that communicating the localized collective economic impacts of individual pesticide use decisions could be most effective among closely knit or more farming communities with high levels of intragroup social capital. Singerman et al. (2017) and Lence and Singerman (2023) reach similar conclusions in their studies of the effectiveness of areawide management of citrus greening among Florida citrus growers.

Evaluating whether communicating MUCs to growers enhances the effectiveness of agricultural extension is thus an important applied question for future empirical research. However, such research first requires credible MUC estimates. This paper aims to initiate the development of these estimates, but important extensions are likely required to obtain credible estimates. Our model makes simplifying, potentially consequential assumptions in both the biomathematical and economic components. These include the single-gene representation of resistance, as opposed to polygenic evolution, as well as the possibility of compensatory mutations that can ameliorate fitness costs (Hawkins et al., 2019). In addition, spatial dynamics across a landscape are an important aspect of resistance management. Spatially explicit models would generate spatially varying MUCs (e.g., Brock & Xepapadeas, 2010), akin to what has been pointed out for estimates of the social cost of nutrient pollution (Keeler et al., 2016).

Related to this are important omitted aspects related to farm size heterogeneity and the degree of pest mobility, both determinants of the extent to which farmers would be expected to internalize pest dynamics (Ambec & Desquilbet, 2012; Carrière et al., 2020). In terms of economics, while our generic framework allows other farm inputs besides the target pesticides, these factors are omitted in the numerical and SA. Additionally, the environmental externalities of pesticide use (both from the pesticide posing resistance problems and its substitutes) are completely omitted from the present analysis. We expect that these elements could interact in complex ways with the costs of pesticide resistance, especially when alternative pest control tools are available which differ in their environmental impact. This is a broad topic we aim to analyze further in future work; the analysis in this paper is a necessary first step in this broader agenda.

Beyond improvements to estimating the costs of pesticide resistance, our SA is an important precursor for identifying what types of scientific information would be most valuable for improving resistance management. For example, while much prior research has been devoted to the study of the fitness costs of resistance, our results suggest this factor does not substantially affect optimal pesticide use or the MUC in our model. In contrast, the model is most sensitive to density dependence, pesticide costs, genetic dominance of resistance, and discounting. Discount rates and density dependence are probably factors for which better information would be most useful for management. Pesticide costs are easily ascertained from the market. Genetic dominance is conceptually straightforward—albeit laborious—to determine in laboratory studies (e.g., Jin et al., 2018). However, the difficulty of determining the appropriate social discount rate (i.e., in collective action problems) is a recurring theme in resource economics and policy (Arrow et al., 2013).

Density dependence is likewise a perennially vexing topic in pest ecology. In contrast to genetic dominance, it is typically more difficult to study in a lab, can vary widely between contexts, and the mechanisms generating it (resource competition, increased disease, etc.) are often difficult to pinpoint (e.g., Johnson et al., 2022). Most relevant information about in situ density dependence probably comes from direct experience controlling the pest in applied settings (e.g., the large-scale population models for ECB estimated by Hutchison et al., 2010). From this perspective, incorporating the value of information about this parameter in the above model using an adaptive management framework is another promising direction for future research, which could provide an avenue to renew prior efforts among applied entomologists to move towards “adaptive resistance management” paradigms (Andow & Ives, 2002; Downes et al., 2010).

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## DATA AVAILABILITY STATEMENT

The data that support the findings of this study are openly available in openICPSR at <https://www.openicpsr.org/openicpsr/> under ID# openicpsr-193166. The computer code for replicating the analysis in this paper is available at <https://www.openicpsr.org> under project ID openicpsr-193166.

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## SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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