Insect Population Dynamics, Pesticide Use, and Farmworker Health

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We address the impacts of regulations designed to reduce pesticide poisoning of farmers and farm laborers. Attention is concentrated on pre-harvest interval regulations that impose a time interval between pesticide application and harvest. The incidence of poisoning is determined by aggregate pesticide use, worker exposure, and toxicity. A dynamic, stochastic model of insect population growth is developed and used to measure the incentives for pesticide use. Increasing the pre-harvest interval has an ambiguous effect on the number of harvest worker poisonings. Pesticide taxation unambiguously reduces the number of worker poisonings. Theoretical results are quantified in a case study of mevinphos application on leaf lettuce in California's Salinas Valley.

Key words: farmworker health risk, interval regulation, pesticides, population dynamics, taxation.

Regulators and the public are becoming increasingly concerned with the adverse public health consequences of agricultural chemical use. One of the most serious concerns motivating pesticide regulation is the impact of these chemicals on the health of farmers and farmworkers. While there are a large number of potential regulations to address this problem, the typical response of state and federal regulators to concerns about farmworker poisoning is to ban the use of the product in question. This paper considers less extreme interventions, and uses a dynamic, stochastic model to assess their impacts on pesticide productivity, pesticide use, and, ultimately, worker health levels.

One potentially important type of agricultural chemical regulation that has received little attention in the economics literature is interval regulation. Two notable examples of interval regulation are the pre-harvest interval (PHI) and the re-entry interval (REI). The former governs the time between chemical application and harvest, while the latter sets the time between chemical application and the date workers can re-enter the field to perform various tasks (e.g., installing irrigation equipment). Since pesticides typically decay when exposed to sunlight, rain, and other environmental factors, a sufficiently long interval will reduce the chemical's toxicity.

While there is now a relatively large literature on the economic effects of banning pesticides (see for example Zilberman et al.; Lichtenberg, Parker, and Zilberman; and Sunding), there has been less analysis of the impacts of regulating the timing of pesticide applications. One notable exception is the pioneering 1993 study of Lichtenberg, Spear, and Zilberman, which presents a method for finding efficient re-entry intervals.[1] As a part of their analysis, Lichtenberg, Spear, and Zilberman consider farmer response to regulating the timing of pesticide applications. They assume a pest infestation of predetermined size and allow the farmer to choose between applying pesticides early (preventively) or on the last feasible day, defined as the desired harvest date minus the re-entry interval (reactively). They conclude that farmers may respond to increases in the re-entry interval by applying pesticides preventively. Furthermore, they conclude that increasing the re-entry interval unambiguously reduces the number of farmworker poisonings.

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[1] The econometric analysis of Hubbell and Carlson considers the effect of pre-harvest intervals on insecticide choice and the choice of application rate.

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Our paper takes a different approach. We formalize farmer response to interval regulations in a model with explicit insect population dynamics. In particular, we consider the impact of increasing the interval between chemical application and harvest (i.e., PHI) on pesticide productivity and the corresponding incentives for pesticide use. Indeed, a main goal of our work is to give micro-foundations to pesticide productivity by explicitly considering insect population growth. The impacts on pesticide use are then traced through a health risk generation model to measure the ultimate effect of interval regulation on harvest worker health. This approach yields substantially different results than the work of Lichtenberg, Spear, and Zilberman.

Insect growth rates are modeled as a geometric Brownian motion process. This formulation captures the well-established influence of stochastic weather conditions on insect reproduction and development. Given initial insect population levels and weather, as well as market and regulatory conditions, farmers form expectations about future insect populations and hence crop damage. Using this framework, it is possible to disentangle the various effects of changing the PHI.[2] Increasing the time between pesticide application and worker contact with pesticide residue gives the chemical more time to decay, thus benefiting workers. However, increasing the PHI also alters the productivity of the chemical and changes the optimal amount of pesticide application.

We show that there are cases in which increasing the PHI may increase total pesticide use. The ultimate effect of changing the PHI is then ambiguous theoretically and rests on the relative magnitudes of the pesticide use and pesticide decay effects. An extreme result of our model is that increasing PHI may actually increase the number of pesticide poisonings. This discussion shows a clear distinction between our approach and that of Lichtenberg, Spear, and Zilberman. Their analysis precludes the result that increasing the PHI increases pesticide use in that they assume farmers only choose when to apply pesticides, but not whether to apply pesticides at all. In their framework, increasing the PHI gives farmers greater incentive to apply the pesticide preventively, and benefits workers by giving the chemical additional time to decay. The Lichtenberg, Spear, and Zilberman result, by construction, ignores the possibility that increasing the PHI can increase the aggregate amount of pesticide use, thereby counteracting, to some degree, the salutary effect of increasing the decay interval.

We also apply our model to another important intervention: pesticide taxes. We measure the impact of taxation on worker health levels, and indicate its effectiveness relative to changing the pre-harvest interval. Conceptually and empirically, we demonstrate that pesticide taxes reduce pesticide use and the incidence of pesticide poisoning. The interesting empirical question is the magnitude of these impacts, and how they compare to the sensitivity of worker safety to pre-harvest interval regulation. This comparison is a first step toward a more general determination of efficient worker safety regulations that compares health impacts and social welfare costs.

Our paper contains a case study in which the theoretical model is applied to an actual case of pesticide regulation: controlling worker poisoning from the insecticide mevinphos in California's Salinas Valley. This case study demonstrates how the model can be made operational and discusses the types and sources of data needed to quantify the economic, biological, and toxicological components of the overall framework. The case study measures the impact on worker poisonings of changing the length of the pre-harvest interval for mevinphos, as well as imposing pesticide taxes. The case study is significant because it demonstrates that the integrated approach to farmworker protection proposed in this paper can be used to design policies in a practical and accurate way.

The Model

The model is sequential and the major stages are presented in Figure 1. In the initial period, the farmer observes the initial insect population, market parameters such as pesticide cost and output price, and regulations including the length of the pre-harvest interval. Using this information, the farmer forms expectations about profits at the date of harvest with and without pesticide application. He then chooses, in the second period, whether or not to apply chemicals. In the

[2] Note that we are considering the problem of harvest worker poisoning. In this case, there is effectively no difference between a REI and a PHI. In other applications, the two types of interval regulations may not be equivalent. Our theoretical results apply to these other cases as well.

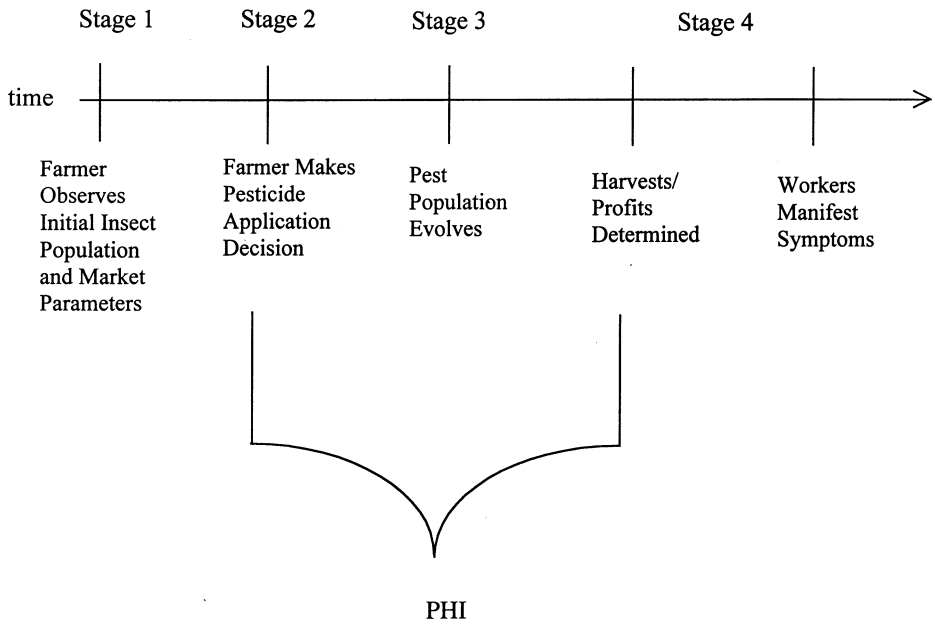

Figure 1. The sequence of events

third period, which is the period of the PHI, the insect population grows or declines. Finally, harvest occurs in the fourth period, at which time the farmer realizes some level of profit.

Damage Function

We suppose that the farmer operates in a competitive market. Insect damage is a function of the number of insects at harvest. Expected profits are determined according to the damage control specification of Lichtenberg and Zilberman. In particular, expected farm profits are equal to

$$(1) \quad E(\Pi) = PY(1 - D) - m$$

where P is the output price net of production costs except the pesticide, Y is potential output, and m is the pesticide cost including any taxes. The expectation is taken over the level of insect damage, D.

Insect damage is a function of the size of the insect population. We adopt a binary damage function where damage is zero if the insect population falls below some predetermined threshold for marketability. This specification is accurate for many fresh and frozen vegetable markets, including the case of leaf lettuce studied in some detail in the empirical section. For example, the USDA sets maximum allowable limits on the number of aphids that can be present on fresh lettuce, broccoli, cauliflower, and other crops. Making the model more general by working with a continuous, concave damage function does not change the basic comparative static results that are central to this paper, but does make the analysis much more complex.

Damage has a probabilistic interpretation as the likelihood that the insect population at harvest exceeds a maximum allowable level, in which case the harvest is a total loss. Formally, $D = \Pr(X_H > X_G)$, where X_H is the insect population at harvest and X_G is the maximum allowable insect population. We now turn to a discussion of insect population dynamics and more fully develop expectations about the terminal number of insects.

Insect Population Growth

The insect population growth rate is modeled as an increasing function of the current insect population and time. However, insect growth is a stochastic process, due in part to the influence of random factors such as weather on reproduction rates (Varley, Gradwell, and Hassell; Trumble; Minks and Harrewijn). Formally, insect population growth is modeled as a geometric Brownian motion process with a drift component as follows:

$$(2) \quad dX = \alpha X\, dt + \sigma X\, dz$$

where X is the current insect population, α is the intrinsic insect growth rate, dt is an increment of time, σ is a variance coefficient, and

$dz = \xi_t\sqrt{dt}$ is the increment of a Wiener process and ξ_t is standard normal.

The process in equation (2) has a number of features that make it appropriate for modeling insect population levels. The process is such that per-period growth rates are normally distributed; as we discuss in the empirical section below, this property matches closely with experimental evidence relating insect growth rates to environmental conditions. Since percentage changes in X are changes in the natural logarithm of X, population levels themselves are normally distributed in this formulation. Thus, X is bounded from below by zero, so that population levels can never be negative. Another important property of this process is that short-run changes in X are dominated by the volatility component of (2), whereas long-run changes are more influenced by the trend component.

For our purposes, it is important to characterize the insect population at some future date t. If the farmer does not use the pesticide, the distribution of the insect population at time t is obtained by applying Ito's lemma to equation (2) to obtain the following:

$$(3) \qquad X_t \sim \text{LN}\left[X_0 e^{\alpha t},\ X_0^2 e^{2\alpha t}\left(e^{\sigma^2 t} - 1\right)\right]$$

where X_0 denotes the initial insect population and X_t denotes the insect population at time t.

In the following section, it will prove useful to know the distribution of insects at harvest time, H, taken prior to the pre-harvest interval, or at date H-PHI. This distribution follows directly from (3) as

$$(4) \qquad X_t \sim \text{LN}\left[X_0 e^{\alpha\text{PHI}},\ X_0^2 e^{2\alpha\text{PHI}} \times\left(e^{\sigma^2\text{PHI}} - 1\right)\right].$$

For the remainder of this paper, X_0 denotes the insect population at time H-PHI. Equation (4) will now be used to derive a grower's chemical application rule conditional on his observation of the initial insect population.

Optimal Chemical Application

This section derives a farmer's decision rule for pesticide use. The basic structure of the pesticide application decision is straightforward: apply the pesticide or not at date H-PHI.[3] Denote the effectiveness of the pesticide as $(1-\mu)$, so that the insect population after application is μX_0.

The decision rule is best understood as a locus of parameter values for which the farmer is indifferent between using the pesticide or not. This locus is derived by equating expected profit levels with and without application of the pesticide. There are two damage probabilities of interest in the derivation of these expected profit levels, one that attains if the farmer applies the pesticide, and one that attains if he does not. Let D_{NA} denote the probability that $X_H > X_G$ if the farmer does not apply the insecticide, conditional on government regulations (e.g., product marketability, pesticide cost, and the length of the PHI), the initial insect population, and expected population growth rates. Similarly, the probability that the insect population at harvest exceeds the marketability standard if the farmer does apply the insecticide is D_{A}.

Equating expected profits with and without pesticide application, it follows that the threshold level of the insect population above which the farmer will apply the pesticide, $\tilde{X}$, is defined implicitly by the following equation:

$$(5) \qquad D_{\text{A}} + \frac{m}{PY} = D_{\text{NA}}.$$

This decision rule is used to describe how the farmer's pesticide application decision is altered by changes in the policy parameters.

Before proceeding, note that this basic framework can be extended to the situation where the farmer is choosing between two chemical alternatives, as opposed to the case considered in (5) where the choice is between a single pesticide and no treatment at all. It is straightforward to show that (5) becomes

$$(5') \qquad D_1 + \frac{m_1}{PY} = D_2 + \frac{m_2}{PY}$$

where the subscripts 1 and 2 denote the two alternative chemicals. As expected, pesticide choice hinges critically on relative effectiveness and relative cost. Since the empirical example presented later is captured by (5), we will pursue this formulation; note, how-

[3] This formulation assumes that the PHI constraint is binding.

ever, that many of the analytical results derived later go through with only obvious modifications if (5') is used.

Comparative Statics

Consider first the marginal effect of changing the pre-harvest interval. Totally differentiating (5) and applying the Implicit Function Theorem yields

$$(6) \quad \frac{d\widetilde{X}}{d\text{PHI}} = -\frac{\frac{\partial D_{\text{NA}}}{\partial \text{PHI}} - \frac{\partial D_{\text{A}}}{\partial \text{PHI}}}{\frac{\partial D_{\text{NA}}}{\partial \widetilde{X}} - \frac{\partial D_{\text{A}}}{\partial \widetilde{X}}}.$$

As we demonstrated in the section on insect population growth, the insect population at harvest time is distributed lognormally. When the farmer applies the pesticide, the number of insects at harvest is distributed as

$$(7) \quad X_H \sim \text{LN}\Big[\mu X_0 e^{\alpha \text{PHI}}, \mu^2 X_0^2 e^{2\alpha \text{PHI}}\Big(e^{\sigma^2 \text{PHI}} - 1\Big)\Big].$$

Recognizing that $\Pr(X_H > X_G) = \Pr(\ln X_H > \ln X_G)$, we convert the damage probabilities into z-scores utilizing the following relationship: $D = 1 - \Phi(z)$, where Φ is the standard normal distribution and z is a z-score. Given pesticide application, the z-score is

$$(8) \quad z_{\text{A}} = \frac{\ln X_G - \mu X_0 e^{\alpha \text{PHI}}}{\sqrt{\mu^2 X_0^2 e^{2\alpha \text{PHI}}\big(e^{\sigma^2 \text{PHI}} - 1\big)}}.$$

Similarly, the z-score for the insect population at harvest given no pesticide application is

$$(9) \quad z_{\text{NA}} = \frac{\ln X_G - X_0 e^{\alpha \text{PHI}}}{\sqrt{X_0^2 e^{2\alpha \text{PHI}}\big(e^{\sigma^2 \text{PHI}} - 1\big)}}.$$

For simplicity, consider the case where the pesticide is fully effective, or $\mu = 0$. The comparative static results below go through with only obvious modifications if μ is positive. If a grower applies the pesticide in the full-effectiveness case, then all the mass of the population density is concentrated at 0 and the derivative (6) can be rewritten as

$$(10) \quad \frac{d\widetilde{X}}{d\text{PHI}} = -\frac{\frac{\partial z_{\text{NA}}}{\partial \text{PHI}}}{\frac{\partial z_{\text{NA}}}{\partial \widetilde{X}}}.$$

Taking the relevant derivatives and manipulating yields the following expression:

$$(11) \quad \frac{d\widetilde{X}}{d\text{PHI}} = -\alpha \widetilde{X} + \frac{1}{2}\frac{\widetilde{X}\sigma^2\big(\widetilde{X} e^{\alpha \text{PHI} - \ln X_{\text{G}}}\big)}{\ln X_G\big(e^{\sigma^2 \text{PHI} - 1}\big)}.$$

A sufficient condition to sign the equation above is for the expression in parentheses in the numerator to be less than or equal to zero. This condition implies a negative sign for equation (11); i.e., increases in the pre-harvest interval result in a decrease in the critical population level, thus implying more pesticide use. This result is worth emphasizing: it is possible that increases in the PHI, which are designed to protect farmworkers, may actually provide incentives for increased pesticide application.

Given the importance of this result, it is useful to examine this sufficient condition more carefully. The negativity condition amounts to a requirement that $\ln X_G \geq X_0 e^{\alpha \text{PHI}}$. Noting that $\ln X_G$ is the transformed government marketability standard and that $X_0 e^{\alpha \text{PHI}}$ is the mean value of the insect population at harvest time, it follows that this condition will be satisfied when a farmer expects to meet the marketability standard without pesticide application. Equivalently, this condition holds when the probability of crop failure is less than or equal to one-half.

Alternatively, increasing the PHI could result in less pesticide application if $\ln X_G \leq X_0 e^{\alpha \text{PHI}}$ and the second term in expression (11) is greater in absolute magnitude than the first term. By similar logic, a necessary but not sufficient condition for this occurs when the probability of crop failure is greater than one-half.

The basic intuition behind these results hinges on the fact that, conditional on some initial insect population, lengthening the pre-harvest interval increases both the mean and the variance of the insect population at harvest. Thus, when there is only a "slight" chance of failing to meet the marketability standard, an increase in the mean and variance is undesirable and induces more pesticide applications. However, when the chance

of failing to meet the regulations is "large," increases in the variance of the insect population (more specifically increasing the variance faster than the mean) may increase the possibility of meeting the standard and result in a reduction of pesticide applications. Therefore, the ultimate impact of pre-harvest interval regulation on pesticide applications remains an open, empirical question.

Similar methods can be used to show how the farmer's pesticide application decision changes with respect to the other government policy variables. For example, the change in the threshold level of insects as the cost of pesticides changes, for example through a pesticide tax, is

$$(12) \quad \frac{d\widetilde{X}}{dm} = \frac{\widetilde{X}^2 e^{\alpha \mathrm{PHI}} \sqrt{e^{\sigma^2 \mathrm{PHI}} - 1}}{PY \ln X_G \varphi(z_{\mathrm{NA}})}$$

where $\varphi(z_{\mathrm{NA}})$ is the standard normal density evaluated at z_{NA}. It is clear that expression (12) is positive. Thus, an increase in pesticide costs increases the threshold pest population size above which it is optimal to apply pesticides, thereby confirming that pesticide use is decreasing in the cost of pesticides.

It is also interesting to examine how changes in the marketability standard affect the incentives for pesticide use and worker health levels. Given some fixed price of output, relaxing the marketability standard would clearly reduce the amount of pesticide use. However, it is unlikely that output price would remain fixed if the number of insects present at harvest were increased. A full understanding of how product quality standards affect pesticide use therefore requires specification of a hedonic demand function. It seems clear, however, that the effect of product quality on price would reinforce the pure effect of the marketability standard on pesticide use. Lowering the net price received by farmers for the crop further reduces the incentive to use pesticides. Consumers suffer in this case, however, since they value product quality and a reduced price of output simply reflects their diminished marginal valuation of the crop output.

The Health Risk Generation Process

In order to assess the effectiveness of pesticide regulation, we must first define the health risk generation process. The magnitude of health risk in a population of workers is defined as the probability of manifesting a physical ailment as a result of contact with a toxic substance used in the workplace. If there is a pre-existing incidence of this ailment in the population, then risk refers to the incremental risk incurred by human exposure to the contaminant. Health risk is most commonly represented as the product of three basic risk factors: (*a*) ambient contamination, (*b*) human exposure, and (*c*) the manifestation of physical symptoms, termed the dose–response relationship.

This multiplicative formulation is reasonable when the increased risk is relatively small, and is particularly germane to cases where there is pre-existing background risk (Van Ryzin, Crump and Howe, Krewski and Van Ryzin). In order to analyze the impacts of government policies on health risk, it is convenient to work with the following relationship:

$$(13) \quad R(m, \mathrm{PHI}) = C(m, \mathrm{PHI}) \cdot E(\mathrm{PHI}) \cdot D$$

where R is health risk, C is contamination (or deposited pesticide residue), E is exposure, and D is the dose–response parameter. Note that since the dose–response parameter simply maps exposure to illness, it is affected by physiology and not policy choices.

In this paper, we will largely confine ourselves to the problem of protecting harvest worker health. It is straightforward to apply the basic framework to other target populations including pesticide applicators, irrigation workers, and farmers themselves. There are numerous policies designed to improve farmworker health, including posting and training regulations, protective clothing, and protective equipment regulations (Sunding and Zivin). As mentioned earlier, in this paper we limit attention to two policies: the pre-harvest interval and pesticide taxation.

Pesticide taxation is designed to change the marginal incentives to use a toxic substance. To the extent that this regulation provides a marginal disincentive to use the toxic material (through higher costs), use is diminished and workplace injuries are reduced. In terms of the risk factors outlined in (14), taxation reduces pesticide use but leaves exposure unaffected. The pre-harvest interval regulation, by contrast, is primarily designed as an exposure regulation. As the PHI is lengthened, pesticides in the field have more time to decay, ultimately reducing farmworker exposure. However, our earlier economic analysis shows that PHI regulation also alters the

incentives for pesticide use by changing pesticide productivity. Thus, in terms of equation (14), PHI regulation affects both contamination and exposure. The net effect of these impacts will be discussed in detail in a later section.

The approach taken in this paper is predicated on basic toxicology and is consistent with risk assessment methods currently in use in the United States and other countries. That is, the health effects of pesticide use are supposed to result from the product of total pesticide use, worker exposure to the pesticide, and a dose–response, or toxicity, parameter extrapolated from controlled, replicable animal studies. There is some literature on pesticides and worker health in this *journal* that takes a different approach. For example, Antle and Pingali develop an econometric model of pesticide use and farmer health to measure the impacts of pesticide use on health levels and the impact of health on farm productivity in two regions of the Philippines. They demonstrate that pesticide use has a deleterious effect on farmer health and that productivity is increasing in the level of farmer health, implying that there may well be social gains from reducing pesticide use in this case study.[4] The econometric approach taken in their paper is a type of epidemiological analysis that characterizes the *ex post* statistical relation between health levels and various environmental factors.

We adopt the toxicology approach in this paper for several reasons. First, toxicology methods are used by the U.S. EPA and state agencies to set pesticide regulations. Indeed, many of the basic parameters used in our case study are taken directly from the risk assessments performed by the State of California prior to registration of the pesticide that is the subject of our case study. Clearly, if economic concepts are to be used to influence risk regulation, it is imperative that economists use similar methods and data as the rest of the regulatory community. Second, the epidemiological approach to risk regulation, while interesting from an econometric point of view, is out of favor with regulators for some valid reasons. Toxicological analysis is based on laboratory experiments and can thus be confirmed or denied in a systematic way. Epidemiological analysis is nonreplicable and subject to the usual problems of measurement error (e.g., under-reporting of injury) and misspecification of the underlying econometric model. Third, it is impossible to use an econometric model to examine the effects of changing PHI if there are no observed changes in this regulation in the sample. In order to avoid these problems, but most importantly to help build a bridge between economics and the rest of the risk regulation community, we adopt the toxicology approach to modeling health impacts of pesticide use.

[4] Other papers in this vein include Antle and Capalbo; Crissman, Cole, and Carpio; and Pingali, Marquez, and Palis; as well as the volume edited by Warborton, Pingali, and Palis.

Deposited Residue

In the context of pesticide application, it is sensible to treat contamination as deposited residue, defined as the amount of pesticide present per unit of plant surface area at the time of application. Deposited residue is determined in part by the amount of pesticide application, which is in turn affected by the marginal productivity of the pesticide. Deposited residue (or contamination, C) is the product of the share of acres treated with the pesticide (RS), the amount of the chemical used per acre treated (a) and a crop-specific coefficient (v) that converts per-acre application into deposited residue. Formally, $C(m, \text{PHI}) = \text{RS}(m, \text{PHI}) \cdot a \cdot v$.

Recall from the discussion on profit-maximizing pesticide application that a farmer will apply the pesticide if the number of insects exceeds some threshold level. Denote the probability density of the initial insect population across fields in a defined region as $f(x)$. The regional share of acres on which the pesticide is used, RS, is the probability that the initial insect population on a given plot will exceed the threshold population X_c. Formally, then,

$$\text{(14)} \quad \text{RS}(m, \text{PHI}) = \int_{\tilde{X}(m,\text{PHI})}^{\infty} f(X)\, dX.$$

This coefficient measures the probability that a randomly selected acre will be treated with the pesticide.

The coefficients a and v are exogenous, and are invariant with respect to the length of the pre-harvest interval. The amount of pesticide applied per acre, a, is specified on the product label, typically in terms of pounds of active ingredient per acre. The coefficient v maps pounds of chemical per acre into micrograms per square centimeter of surface area. This coefficient reflects the fact that only a fraction of all applied pesticides ends up as

deposited foliar residue that harvest workers can contact (Zweig, Leffingwell, and Popendorf), and is heavily influenced by plant shape and size.

Exposure

Harvest worker exposure to the pesticide is given as

$$E(\text{PHI}) = q \cdot e^{-k\text{PHI}} \tag{15}$$

where k is a residue decay parameter and q is an exposure coefficient. Exposure is measured in units of square centimeters of plant surface area per kilogram of body weight per day. The decay parameter converts units of deposited residue at the time of application into units of pesticide residue at harvest time, reflecting the fact that PHIs are primarily designed to give pesticides time to "cool down" before they are encountered by harvest workers. The decay parameter is a function of the type of pesticide applied, and may also be influenced by weather conditions such as humidity, temperature, and rainfall (Popendorf and Leffingwell).

The coefficient q in equation (15) simply denotes the amount of plant surface area that a worker contacts while on the job. The type of job performed by the worker influences this coefficient. For example, harvest workers have more exposure to plant surfaces than installers of irrigation equipment. The coefficient q is also heavily influenced by workplace safety measures such as mandatory protective clothing (Sunding and Zivin).

For the sake of clarity, it is wise to review the units in which contamination and exposure are measured. Contamination is expressed in terms of $\mu g/cm^2$, exposure is measured as $cm^2/kg/day$. Thus, the product of contamination and exposure has units of $\mu g/kg/day$. This measure conforms to the toxicological notion of an average daily dose, which is standard in the scientific literature. The dose–response parameter, denoted D in equation (14), maps average daily dose into numbers of acute poisoning cases. The dose–response relationship is fundamental to modern toxicology, and dose–response parameters are readily available for most pesticides.

Written in full, the health risk equation is as follows:

$$R(m, \text{PHI}) = \text{RS}(m, \text{PHI}) \cdot a \cdot v \cdot q \cdot e^{-k\text{PHI}} \cdot D. \tag{16}$$

A marginal increase in the pesticide tax unambiguously reduces the number of poisonings; deposited residue is reduced while exposure is unaffected. The marginal impact of lengthening the PHI is less certain theoretically since deposited residue may increase, although exposure must decrease. In any case, it is necessary to compute the effects of the PHI on both contamination and exposure to develop a true measure of health impacts.

Before proceeding, it is important to point out that most of the parameters of (16) will, in reality, only be known with some error, even though our analysis treats all these factors as known. Given the relatively undeveloped state of the interval regulation literature and the fact that we wish to emphasize how PHIs alter economic incentives for pesticide use, our treatment seems reasonable. In other applications, however, it will be important to account for this parameter uncertainty. At least two economic studies have identified methods for dealing with this problem when making environmental health regulations (Lichtenberg and Zilberman; Lichtenberg, Zilberman, and Bogen). As these papers indicate, parameter uncertainty will influence policy choices if the regulator is risk averse or if the risk factors are covariant (as may be the case if a number of them are affected by weather).

Case Study: Mevinphos Application in California's Salinas Valley

This section presents an application of the conceptual model to a specific case of worker protection regulation: farm worker poisoning from the pesticide mevinphos used on leaf lettuce in California's Salinas Valley, a prime vegetable-producing region located on the central coast of the state. Mevinphos (2-carbomethoxy-1-methylvinyl dimethyl phosphate) is an organophosphate insecticide used on a variety of crops, mainly vegetables. In California, mevinphos is applied primarily to head lettuce, leaf lettuce, cauliflower, broccoli, and celery. Mevinphos is a foliar insecticide used primarily to control aphids, although it is also effective against mites, grasshoppers, cutworms, leafhopper caterpillars, and other insects. The pesticidal activity of mevinphos is due to its inhibition of acetylcholinesterase activity.[5]

[5] Cholinesterases are a family of enzymes found throughout the body that hydrolyze choline esters. In the nervous sys-

There are numerous reported cases of worker poisoning involving mevinphos, most resulting from acute exposure. Indeed, mevinphos is responsible for more acute illness than any other insecticide currently in use (California Environmental Protection Agency 1996). The principal symptoms of mevinphos poisoning are nausea, diarrhea, vomiting, pinpoint pupils, tremors, and, in extreme cases, paralysis. Mevinphos is not known to be carcinogenic and is not believed to cause reproductive or developmental toxicity. In California, there were 548 reported cases of acute mevinphos poisoning involving farm workers from 1982 to 1991. There were sixty-eight cases involving one or more days of hospitalization and 201 cases involving one or more lost sick days during this period (O'Malley).

We now turn to a description of the data used to quantify the model in the case study. First consider parameters of the stochastic process describing the evolution of the insect population. In the theoretical discussion of the previous section, it was assumed that the insect population level (aphids in our case study) followed a GBM process. This assumption is especially appropriate for the case study chosen here. Recall that the GBM process is tantamount to assuming normally distributed population growth. Ruggle and Gutierrez show that the green peach aphid growth rate is linear in average daily temperature, and is given by the formula:

$$\begin{aligned}\%\ \text{daily growth} &= -53.48 + 1.11 \\ &\quad * \text{ average daily temperature.}\end{aligned}$$

Further, average daily temperature is normally distributed. Combining these two relationships, it follows that the percentage growth rate of the aphid population is normally distributed in the case study situation, just as assumed by the GBM framework.

To find the parameters of equation (2), it is helpful to rearrange this expression. This difference equation implies that

$$X_{t+1} - X_t = \alpha X_t + \sigma X_t \varepsilon_t$$

or that

$$(17) \quad \frac{X_{t+1} - X_t}{X_t} = \alpha + \sigma \varepsilon_t.$$

This expression implies that the percentage change in the aphid population is equal to an average growth rate plus a variance term, and that the estimates of α and σ are simply the sample mean and standard deviation. Utilizing the fact that the mean and standard deviation of average daily June temperatures in the Salinas Valley are 58.98 and 10.38 (National Oceanic and Atmospheric Administration), it follows that $\alpha = 0.1199$ and $\sigma = 0.1152$.

In order to describe the mevinphos application decision, it is also necessary to gather some information on leaf lettuce production in the Salinas Valley. Estimates of output price and per acre yields are based on averages calculated from eight years of data presented in the Monterey County Agricultural Commissioner's Reports (1989–96). Mean yield is 791.12 cartons per acre and mean farm gate price is \$6.71 per carton. Pre–harvest production costs (excluding the cost of mevinphos application) are taken from University of California Extension crop budgets and are set at \$1,590.09 per acre. Contracted harvesting costs are \$3.50 per carton (University of California Cooperative Extension). These cost figures imply a net price of \$1.20 per carton of lettuce. The cost of mevinphos application is \$50 per acre (Chaney). Total acreage of leaf lettuce in the Salinas Valley is set at 19,000 (Monterey County Agricultural Commissioner).

Lastly, the quantification of the application decision in equation (5) requires specification of status quo policy parameters. In particular, it is necessary to know current preharvest interval regulation and marketability standards for leaf lettuce. Initially, the pesticide tax is set at zero, so the cost of application is simply its market price. The state preharvest interval for mevinphos used on leaf lettuce is presently seven days (California Environmental Protection Agency 1994). A carton of lettuce (composed of twenty-four heads) is deemed unacceptable if more than three heads contain insect defects, where an insect defect is defined as any head containing five or more aphids (Chaney). Thus, we set the government marketability standard at fifteen aphids per carton, or 11,866.80 aphids per acre.

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tem, acetylcholinesterase (AChE) is involved in the termination of impulses across nerve synapses including neuromuscular junctions by rapidly hydrolyzing the neural transmitter acetylcholine. Inhibition of AChE results in overstimulation followed by depression or paralysis of the cholinergic nerves throughout the central and peripheral nervous systems (California Environmental Protection Agency 1994).

Given this basic biological and economic information, it is possible to determine an individual farmer's application decision conditioned on an initial aphid population [see equation (5)]. Recall that for an individual farmer, this decision is a discrete choice: apply the pesticide or not given the initial aphid population. Using equation (5) and retaining the assumption that $\mu = 0$ (Chaney), the critical initial aphid population is 614.85 per acre; that is, a farmer will apply mevinphos if he observes an aphid population above this level. Table 1 summarizes the exogenous parameters underlying the case study, as well as the endogenous values of the critical aphid population, the share of fields treated with mevinphos, and the number of harvest worker poisonings.

Table 1. Key Parameters of the Case Study

Exogenous	Value
P: net price ($/carton)	1.20
Y: yield (cartons/acre)	791.12
m: pesticide cost ($/acre)	50
α*: intrinsic growth rate*	0.1199
σ*: variance of growth rate*	0.1152
X_G*: quality threshold* (aphids/acre)	11,866.80
PHI (days)	7
Mean aphids/acre	1,265.80
S.D. aphids/acre	967.17
$a \cdot v$*: deposited residue* $\mu(g/cm^2)$	0.0660
q: exposure coefficient (cm^2/kg)	13.6300
k: residue decay ($\mu g/cm^2/day$)	0.0720
Bodyweight (kg)	70
Crew size (people)	60
Time to harvest an acre (hours/crew)	8
Acres	19,000
D: dose–response	3.85E-05
Endogenous	**Value**
$\tilde{X}$*: application threshold* (aphids/acre)	614.85
RS*: share of acres treated*	0.7512
R: poisonings (cases)	17.9176

To address aggregate mevinphos application under some configuration of policy parameters, it is necessary to know the occurrence of aphids across fields in the Salinas Valley. As part of a pest management study, the University of California Cooperative Extension office in Salinas has been collecting data on aphid populations on commercially farmed leaf lettuce plots in the region. We obtained data on aphid population levels from ninety plots farmed using standard (i.e., non-IPM) methods observed prior to June harvest. The aphid population level per acre is normally distributed with a mean of 1,265.79 and a standard deviation of 965.17. Given a PHI of seven days, it follows that the probability that a randomly selected field will be treated with mevinphos is $\Pr(X \geq 614.85) = 0.7512$, or 75.12%. This figure corresponds closely to the actual application rate of 74.57% of leaf lettuce acres in the Salinas Valley (California Environmental Protection Agency 1998).

We now turn to the parameters of the health risk generation process. Consider first the deposited residue or contamination component of the process. Deposited residue is the product of the application share, the amount of active ingredient applied per acre, and the foliar residue coefficient. The approved rate of mevinphos application stated on the label is 0.25 pounds per acre. Field studies by Spencer et al. demonstrate that mevinphos application on leaf lettuce at the prescribed rate results in an initial deposited foliar residue of 0.066 $\mu g/cm^2$.

As discussed earlier, exposure is based on values of q and k, and the length of time between application and exposure. In theory, the decay parameter k can be affected by weather conditions such as rainfall and temperature (Spear et al., Nigg et al.). However, Spencer et al. have demonstrated that for many vegetable crops produced under varying weather conditions in California, including leaf lettuce in the Salinas Valley, the mevinphos decay coefficient is in fact constant at $k = 0.072$ $\mu g/cm^2/day$.

The exposure parameter q is a function of some crop-specific dosing coefficient[6] that

[6] This coefficient varies by task and also according to protective clothing and equipment regulations. Typically, this dosing coefficient is broken down into a transfer component and an absorption component. Transfer components are often expressed as a clothing penetration measure or as an inhalation uptake, depending on the route of contact, and vary by type of clothing and equipment (see for example Fong, Brodberg, and Fong; Maddy et al.; Brodberg and Sanborn). Absorption coefficients are generally extrapolated from toxicological studies conducted

Table 2. Marginal Impacts of Policy Reforms

		Policy Change	
		dm	*d*PHI
Marginal Change in:			
Application threshold	$d\tilde{X}$	8.97 × E-4	−0.11
Application share	dRS	−1.70 × E-4	0.02
Contamination	dC	−1.12 × E-5	1.39 × E-3
Exposure	dE	0	−0.59
Poisonings	dR	−4.06 × E-3	−0.79
Expected profit	$d\mathrm{E}\pi$	−14,273	−1.45 × E6
Lost expected profit			
per poisoning averted	$d\mathrm{E}\Pi/dR$	3.51 × E6	1.84 × E6

relates the amount of active ingredient contacted to an hourly dose of poison, as well as certain worker characteristics, including the duration of contact and worker body weight. We assume that a workday consists of one eight-hour shift and that each worker has a body weight of 70 kg. Further, Formoli, Thongsinthusak, and Sanborn estimate the crop-specific dosing coefficient for mevinphos use in leaf lettuce to be 119.28 cm^2/hr. This figure is based on the assumption that harvest workers wear the standard uniform of long-legged pants, long-sleeved shirts, gloves, and a hat. Thus, the exposure parameter q for an individual harvester is 13.63 cm^2/kg. Recognizing that a sixty-person crew can harvest one acre of leaf lettuce in one eight-hour workday (University of California Cooperative Extension), we can calculate total worker exposure per acre. As mentioned earlier the pre-harvest interval for mevinphos used on lettuce in Monterey County is seven days.

The dose–response relationship D is a biological relationship that maps the amount of toxin taken up by an individual (i.e., the product of contamination and exposure, or average daily dose) into a manifestation of clinical symptoms requiring at least one lost day of work. Typically, this relationship is determined by extrapolating from animal models by adjusting for differences in body mass. Following O'Malley, we set D at 3.85×10^{-5}.

It is now possible to calculate the number of poisonings predicted by the model. Mutiplying the contamination parameter by the exposure parameter and the dose–response parameter, it follows that the predicted number of poisonings is 17.91 in the base case. Interestingly, this number corresponds closely to the reported number of mevinphos poisonings (17.78 cases) resulting from leaf lettuce harvest work.[7] Thus, both the application share (75.12%) and the number of poisonings (17.91) predicted by the model correspond closely to actual observations (74.57% and 17.78 cases, respectively).

Table 2 presents the results of the marginal impact analysis based on the parameters pertaining to the leaf lettuce case study. The second column denotes the marginal change in the threshold number of aphids above which the farmer will apply mevinphos. As indicated by expression (11), this threshold drops as PHI increases. Consequently, as shown in the third column, the fraction of growers applying mevinphos increases by 2%. Thus, increasing PHI increases contamination by 1.39 × E-3 $\mu g/cm^2$, as shown in the fourth column.

A marginal increase in the pesticide tax has the opposite effect on contamination. The second column of Table 2 shows the marginal impact of increasing the per acre cost of mevinphos on the application threshold. As predicted by equation (12), this threshold is increasing in the tax, which implies that RS is decreasing in the tax. The fourth column of the table shows the effect of an increase in the tax on deposited residue. Increasing the tax marginally reduces contamination by 1.12 × E-5 $\mu g/cm^2$.

The exposure parameter is affected only by the PHI. As discussed above, this effect is unambiguous since increasing the PHI gives the pesticide time to decay, which reduces

on laboratory animals, are specific to neither clothing nor equipment, and vary by route of exposure.

[7] This latter figure is derived by multiplying the total number of reported mevinphos poisonings (201) by the share of mevinphos applied to leaf lettuce in the Salinas Valley (8.85%) (California Environmental Protection Agency 1998).

worker injury per unit of pesticide application. Table 2 indicates that the marginal effect of PHI on per acre exposure is equal to -0.59 μg/kg/day.

Next, we calculate the marginal impact of the policy changes on the number of poisonings. These calculations are given in the sixth column of Table 2. The marginal effect of the PHI on worker health is especially interesting in this case study since we have exactly the type of situation in which increasing PHI can harm workers: namely, increasing the PHI increases the incentives to use mevinphos. The empirical analysis shows, however, that the drop in exposure outweighs the increase in contamination for the parameters of this case study, and as a result the number of mevinphos poisonings drops by 0.79 cases in response to a unit increase in the PHI. As expected, the pesticide tax also reduces the number of poisonings; Table 2 indicates that this marginal change is small (4.06 × E-3 cases). This latter result follows from the small impact of the tax on deposited residue (which follows from the modest marginal impact of a per acre tax on the percentage of growers using the pesticide).

It is of interest to evaluate the change in grower profit resulting from each of these marginal reforms since understanding how profit changes allows a comparison of policies in terms of lost profit per poisoning averted. Expected profit per acre is given as

$$\text{(18)}\quad \mathrm{E}\Pi = \int_0^{\tilde{X}} PY\left(1 - D_{\mathrm{NA}}\right) f(X)dX + \int_{\tilde{X}}^{\infty} [PY(1 - D_{\mathrm{A}}) - m] f(X)\, dX.$$

Taking the derivative with respect to the pesticide cost per acre, we have

$$\frac{\partial \mathrm{E}\Pi}{\partial m} = -\int_0^{\infty} f(X)\, dX + PY\left(1 - D_{\mathrm{NA}}\tilde{X}\right)\frac{\partial \tilde{X}}{\partial m} f(X) - [PY(1 - D_{\mathrm{A}}(\tilde{X})) - m] \times \frac{\partial \tilde{X}}{\partial m} f(X).$$

Using the definition of the critical insect population, it follows that marginal lost profit per acre is given by

$$\text{(19)}\quad \frac{\partial \mathrm{E}\Pi}{\partial m} = -[1 - F(\tilde{X})].$$

Similar analysis yields an expression for the marginal change in profit as PHI and the marketability standard increase. Taking the derivative of (19) with respect to PHI, we have

$$\frac{\partial \mathrm{E}\Pi}{\partial \mathrm{PHI}} = -\int_0^{\tilde{X}} PY \frac{\partial D_{\mathrm{NA}}}{\partial \mathrm{PHI}} f(X)\, dX - \int_{\tilde{X}}^{\infty} PY \frac{\partial D_{\mathrm{A}}}{\partial \mathrm{PHI}} f(X)\, dX$$

making use of Leibniz' Rule and the definition of $\tilde{X}$. This derivative can be expressed as

$$\text{(20)}\quad \frac{\partial \mathrm{E}\Pi}{\partial \mathrm{PHI}} = \int_0^{\tilde{X}} PY\varphi(z_{\mathrm{NA}}) \frac{\partial z_{\mathrm{NA}}}{\partial \mathrm{PHI}} f(X)\, dX + \int_{\tilde{X}}^{\infty} PY\varphi(z_{\mathrm{A}}) \times \frac{\partial z_{\mathrm{A}}}{\partial \mathrm{PHI}} f(X)\, dX < 0.$$

Table 2 shows the marginal change in grower profit and also the change in profit per poisoning averted. Increasing the PHI has a significant impact on profit since the probability of crop damage increases for all growers using mevinphos. The pesticide tax has a much smaller impact on profit since it does not affect expected crop damage at the margin. It is most interesting to consider the profit impact per poisoning avoided. The PHI is nearly twice as efficient as the pesticide tax by this measure, despite the fact that lengthening the PHI increases aggregate mevinphos use. Increasing the PHI reduces grower profit by $1.84 million per poisoning averted, while the pesticide tax lowers grower profit by $3.51 million per case. The basic reason for this disparity in our case study is the sensitivity of exposure to changes in the PHI.

Conclusions

This paper presents an explicit model of insect population dynamics and employs it to measure pesticide productivity, derive profit-maximizing pesticide application levels, and assess the impact of regulations intended to reduce worker pesticide poisonings. A main result of our conceptual analysis is that pre-harvest interval regulation affects both contamination and exposure, perhaps in opposite directions. Pesticide taxation unambiguously reduces the incidence of poisoning.

Regulators typically set pre-harvest intervals to separate farmworkers from health

hazards by increasing the time between pesticide application and exposure. The intent of increasing this interval is to allow more time for the pesticide to decay. Our paper shows that increasing the pre-harvest interval may increase the incentive for pesticide use, thereby resulting in reduced exposure but increased contamination. The impact of PHI regulation on pesticide use levels should be factored in to regulatory impact analyses rather than assuming that contamination is constant, as is typically the case.

It is important for economists to pay attention to insect population dynamics when assessing pesticide productivity and the impact of pesticide regulations, particularly regulations like the PHI that alter the timing of pesticide use. Without these dynamics, the pesticide use decision can only be motivated by the prevailing conditions at the time of application, and misses an important dimension of the pesticide problem. Our hope is that more economists will work with entomologists and other specialists to create even more elaborate and accurate models of insect population growth and develop solid microfoundations for the economics of pesticide regulation.

Future work should also focus on the multiplicity of regulatory instruments and how these can be combined most effectively to reduce the adverse public health consequences of pesticide use. There are many more potential interventions than the three considered here; training, posting, labeling, and protective equipment regulations are only a few examples. Given the likely decreasing marginal benefits of each regulation, it seems likely that a combination of instruments will be optimal in many cases.

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