# Bioeconomic Analysis of Pesticide Demand

By L. Joe Moffitt and Richard L. Farnsworth*

## Abstract

The ability of insects to develop resistance to specific pesticides affects pesticide demand However, the affect of resistance on demand cannot be observed or measured This analysis substitutes an expression for the unobserved resistance variable in a pesticide demand model and then illustrates the model's potential by estimating demand for DDT To arrive at the expression characterizing the unobserved resistance variable a biological resistance model is constructed then incorporated into the dosage response curve Resistance development is hypothesized to be directly dependent on cumulative pesticide use

## Keywords

Pest resistance, econometrics, pesticide demand

An efficient and economical crop protection system is essential to agricultural production For the past three decades, the primary pest control tactic used by agricultural producers has been to apply toxic chemicals However, people have become more concerned over possible adverse effects of pesticide use on the environment, wildlife resources and human health Their concern has led to research that investigates each chemical's properties, especially with respect to cancer and mutations identifies optimal timing and application rates, restricts or bans hazardous products, assesses impacts on gene pools of affected organisms, and investigates declining pesticide effectiveness from pest resistance

The potential of econometric demand analysis to assess grower behavior and, thus, to increase our understanding of pesticide use appears promising as the observed quantity demanded of a pesticide reflects both economic and technical factors Understanding the decline in effectiveness of the pesticide arsenal is vital from a resource management standpoint An important characteristic of biological populations is their ability, by evolutionary adaptation, to develop resistance An econometric analysis of pesticide use can help us understand resistance development

A recent bibliographical review of pest control economics literature (*11*) cites several studies (1, 8) which indicate the significance of resistance in pesticide use decisions but cites only one econometric demand study (*3*) which incorporates resistance [1] Carlson's method (*3*) involves specifying a log-linear demand function which, in addition to the standard explanatory variables such as own price and price of a substitute includes an index of pest resistance Carlson developed this pest resistance index from treatment figures contained in various published research reports and noted the potentially crude approximation of an index computed in this manner Considering the current lack of data on resistance and the meager prospects of future availability of such data, we must consider alternatives methods of estimating pesticide demand The approach described here involves specifying a resistance development structure that permits an econometric assessment of the impact of pesticide use on resistance development Parameters in the explanatory equation can be estimated by nonlinear least squares

Objectives of this research are threefold (1) to present an explicit mathematical formula for resistance development consistent with models used by biologists and economists

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[1] Italicized numbers in parentheses refer to items in the references at the end of this article

(2) to incorporate the expression for resistance, in place of an unobservable resistance variable, in a pesticide demand model, and (3) to illustrate the model's potential by estimating the demand for the insecticide DDT

Tolerances to different pesticide dose levels are assumed to follow a Weibull density function The extent to which the killing efficiency of pesticides decreases as resistance increases is defined in the function The dosage-response or kill efficiency function is the cumulative distribution function of the Weibull density Borrowing from biologists' S-shaped resistance development models, we hypothesize that resistance development can be modeled using the S-shaped logistic function and that the most important factor affecting resistance development is directly related to cumulative pesticide use We then substitute the resistance development model into the pest tolerance, or Weibull density, function to provide a mechanism for measuring changing pest tolerances as resistance develops over time Because resistance is unobservable, the expression characterizing resistance as a function of cumulative pesticide use is substituted into the demand equation We then estimate a demand function for DDT, the results illustrate the applicability of the resistance model Finally we show the limitations of this study and suggest a direction for further research

## Tolerance Density, Dosage Response, and a Characterization of Resistance

Talpaz and Borosh depicted pest tolerances to pesticides using the Weibull density function The Weibull density of tolerances indicates the proportion of the pest population that is susceptible to pesticide dosage level I but that is not susceptible to any lesser dosage (For example, some pests, through genetic factors, possess a detoxifying enzyme which provides immunity )

The Weibull density is

$$w(I|a,\lambda) = a\lambda(aI)^{\lambda-1}\exp[-(aI)^{\lambda}] \text{ if } I \geq 0 \qquad (1)$$

where I is pesticide dose measured in pounds per acre and a and $\lambda$ are parameters which determine the shape of the density function For example, if $\lambda > 1$, the Weibull density function is unimodal and exhibits other usual characteristics of probability density functions

The dosage-response or kill efficiency function relates cumulative pest mortality to differing pesticide dosages Available evidence beginning with Finney's suggests that the dosage-response relationship increases monotonically and is sigmoidal shaped beginning at the origin and asymptotically approaching 1 as pesticide levels increase Thus, pest mortality increases first at an increasing, and then at a decreasing rate as pesticide dosage levels increase

Given a Weibull density of tolerances, equation (1), the dosage-response function (proportion of the cumulative pest population susceptible to dosage level I) may be defined as the Weibull distribution function, which is constructed by integrating the Weibull density function

$$W(I|a,\lambda) = \int_0^I w(x|a,\lambda)dx$$

$$W(I|a,\lambda) = 1-\exp[-(aI)^{\lambda}] \qquad (2)$$

if $I \geq 0$ Because $\lambda$ is restricted to be greater than 1, W (•) satisfies the desirable properties attributed to dosage-response curves

$$W(0) = 0$$

$$\lim_{I\to\infty} W(I) = 1$$

$$\partial W/\partial I = a\lambda(aI)^{\lambda-1}\exp[-(aI)^{\lambda}] > 0$$

$$\partial^2 W/\partial I^2 > 0 \text{ for } 0 \leq I < ((\lambda-1)/a\lambda)^{1/\lambda}$$

$$\partial^2 W/\partial I^2 < 0 \text{ for } I > ((\lambda-1)/a\lambda)^{1/\lambda}$$

Resistance in a pest population reduces pesticide effectiveness by altering the tolerance density One can characterize the resistance by defining it as a function of the parameters of the Weibull density A convenient definition of a resistance parameter that leads to a plausible transformation of the tolerance density and dosage-response function is $\rho = 1/a$

The corresponding tolerance density and dosage-response functions become

$$w(I|\rho,\lambda) = \lambda\rho^{-1}(I/\rho)^{\lambda-1}\exp[-(I/\rho)^{\lambda}] \qquad (3)$$

$$W(I|\rho,\lambda) = 1-\exp[-(I/\rho)^{\lambda}] \qquad (4)$$

The conditional mean and variance of the redefined Weibull tolerance density are

$$E[I|\rho] = \rho\Gamma(\lambda^{-1}+1)$$

$$V[I|\rho] = \rho^2\Gamma(2\lambda^{-1}+1) - (\Gamma(\lambda^{-1}+1))^2$$

respectively, where $\Gamma(\bullet)$ denotes the gamma distribution

Derivatives of the mean and variance with respect to $\rho$ are

$$\partial E[I|\rho]/\partial\rho = \Gamma(\lambda^{-1}+1) > 0$$

$$\partial V[I|\rho]/\partial\rho = 2\rho[\Gamma(2\lambda^{-1}+1) - (\Gamma(\lambda^{-1}+1))^2] > 0$$

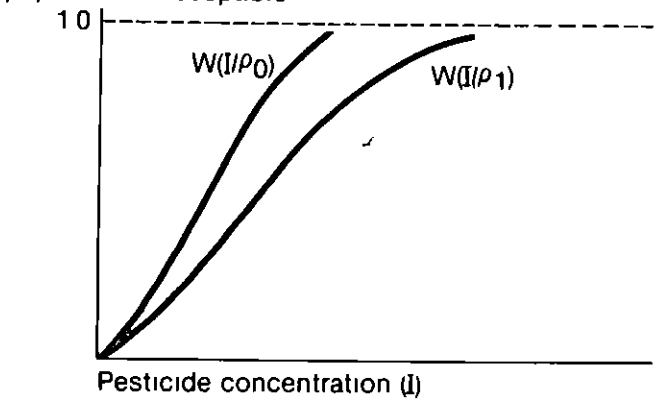
Hence, increasing the resistance parameter shifts the tolerance density to the right and increases the density's dispersion (variance) An increase in resistance alters the dosage-response curve and decreases pesticide effectiveness

$$\partial W(I|\rho)/\partial\rho = -\lambda\rho^{-(\lambda+1)} I^{\lambda} \exp[-(\rho^{-1}I)^{\lambda}] \leq 0$$

For example, an increase in $\rho$ from $\rho_0$ to $\rho_1$ pivots the dosage-response curve to the right and reduces pest mortality at each dosage level (fig 1)

Figure 1

**Dosage-Response Curve.**




Note The dosage response curve is a sigmoidal function which ranges from 0 to 1 It shifts to the right as the resistance parameter $\rho$ increases from $\rho_0$ to $\rho_1$, which indicates that as a pest population develops resistance, more pesticides are required to obtain a specific kill level

## Mechanism and Development of Resistance

We have defined resistance as a function of parameters of the tolerance density and have delineated the ramifications of increasing resistance on pesticide efficacy Now we discuss and model resistance development in a pest population exposed to toxic chemicals The procedure describes resistance as a function of lagged pesticide applications The resistance development model is then substituted into the kill efficiency function in equation (4)

Development of resistance in an insect population is a well-known and logical evolutionary process that allows organisms to evolve and adapt to sudden changes in climate and habitat (2, 5) The rate at which resistance develops in a population is influenced by many factors genetic makeup of the organism, intensity of the selection pressure inducing resistance (for example pesticide dosage and number of applications), ecological conditions behavioral mechanisms which prevent fatal exposure, and biology of the organism, including reproductive rate, inbreeding, dispersal, migration, size, and growth rate Because of their protective mechanisms or other physical factors, some organisms will survive the initial selection pressure The next generation will contain a higher percentage of resistant organisms If selection pressure is applied again, resistant organisms will survive and the next generation will have an even larger percentage of pesticide-resistant organisms Continuous selection pressure eventually results in a population composed largely of resistant organisms

Entomologists have conducted experiments under controlled laboratory conditions to determine implications of resistance development for pesticide dose levels Conclusions are generally stated in terms of the lethal dose (LD) required to kill 50 percent of the pest population over g generations Laboratory results of resistance experiments suggest pesticide dosages necessary to achieve a fixed kill will increase according to a tilted S-shaped pattern over g generations, as figure 2 shows

Available information can be incorporated into the analysis by using the following logistic specification

$$I_k = \tau_k/(1+\exp[\phi + \delta_k(\bar{g}-(t-1))] \qquad (5)$$

where

- k = proportion of the pest population killed,
- $I_k$ = pesticide dose required tokill proportion k of the population,
- $\tau_k$ = a parameter which represents the maximum insecticide dose necessary to kill proportion k of the pest population as generations tend to infinity,
- $\phi$ = a parameter that helps identify the insecticide dose necessary to kill proportion k of the pest population, given the initial level of resistance before pesticides are applied,
- $\delta_k$ = measure of selection pressure at time t that will be associated with accumulated pesticide applications since time t = 0 and

$\bar{g}$ = number of pest generations per time period

This logistic representation is consistent with figure 2, providing both the appropriate shape and a positive vertical axis intercept. Note that equation (5) indicates the dosage level necessary to achieve a constant mortality level as resistance develops. $\delta_t$ represents the accumulated buildup of resistance, reflecting genetic selection pressure of successive generations. To make this concept operational, we define it as a function of accumulated applications of pesticides. We assume $\delta_t$ can be specified as a distributed lag of pesticide levels applied to previous pest generations

$$\delta_t = \sum_{i=1}^{t-1} \sigma_i I_i/(\bar{g}*(t-1))$$

where $\sigma_i$ represents an appropriate lag structure. In our subsequent empirical illustration, $\sigma_i$ is assumed constant ($\sigma_i = \sigma$), which implies

$$\delta_t = \sigma \sum_{i=1}^{t-1} I_i/(\bar{g}*(t-1))$$

When $\delta_t$ is substituted into equation (5), the number of pest generations, g, and time periods drop out. The definition of $I_k$ simplifies to

$$I_k = \tau_k/(1+\exp[\phi+\sigma Z_t]) \quad \text{where } Z_t = \sum_{i=1}^{t-1} I_i \qquad (6)$$

Equation (6) possesses the desired tilted S-shape and describes resistance as a function of cumulative insecticide use. Furthermore note that figure 2 is a special case of equation (6) when dose is held constant over generations. The vertical axis intercept and horizontal asymptote are

$$\lim_{t\to 0} I_k = \tau_k/(1+\exp[\phi])$$

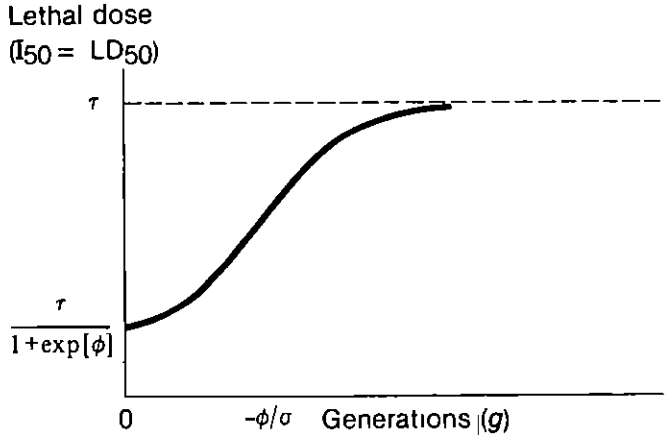
and

$$\lim_{t\to \infty} I_k = \tau_k$$

The positive vertical axis intercept permits initial resistance in the pest population and is consistent with the dosage-response curve which is undefined at a zero level of resistance ($\rho$=0).

Figure 2

## Development of Resistance




Note: Biologists hypothesize one of the effects of increasing resistance is to decrease pesticide effectiveness. As resistance develops over g pest generations, the lethal dose (LD) necessary to kill 50 percent of the population in every generation increases and follows a sigmoidal growth curve. Mathematically, the logistic function in equation (5) has similar characteristics and can be used to represent the sigmoidal curve.

Our next task is to relate $I_k$ to the resistance parameter $\rho$ in equations (3) and (4). From equation (4) the specific proportion $W_k(\bullet)$ of the pest population susceptible to insecticide dose I is a function of $\lambda$ and $\rho_k$. At time t=0, the insecticide dose necessary to achieve $W_k(\bullet)$ is the vertical axis intercept, $\tau_k/(1+\exp[\phi])$, of the resistance development model

$$W_k = 1 - \exp[(-\tau_k/\rho_0(1+\exp[\phi]))^{\lambda}]$$

Solving for $\tau_k$ yields

$$\tau_k = -(1+\exp[\phi])\rho_0[\ln(1-W_k)]^{1/\lambda}$$

Substituting $\tau_k$ into equation (6) yields a resistance development model that is consistent with the dosage-response curve for any time period

$$I_k = -(1+\exp[\phi])\rho_0[\ln(1-W_k)]^{1/\lambda}/(1+\exp[\phi+\sigma Z_t]) \qquad (7)$$

Solving equation (7) for $W_k$ yields

$$W_k = 1 - \exp\left[\frac{-(1+\exp[\phi+\sigma Z_t])I_k}{(1+\exp[\phi])\rho_0}\right]^{\lambda} \qquad (8)$$

Comparison with equation (4) indicates resistance for any time period can be determined from the following equation

$$\rho_t = \frac{(1+\exp[\phi])\rho_{t0}}{1+\exp[\phi+\sigma Z_t]} \qquad (9)$$

Thus, equation (9) relates resistance to parameters which can be estimated from observable data Equation (9) will be used to incorporate pesticide resistance into an analysis of demand by farmers for DDT

## Pesticide Demand Model

The importance of equation (9) is that it allows resistance to be included in a pesticide demand equation Borrowing from Carlson's research we assume the following Cobb-Douglas demand model

$$I = A\rho^{\beta} \prod_{i=1}^{k} X_i^{\alpha_i} + U$$

where I is quantity of pesticide demanded, the $X_i$'s are relevant demand variables, $\rho$ is the resistance parameter which characterizes pesticide effectiveness, A, $\beta$, and the $\alpha_i$'s are unknown parameters, and U is a stochastic disturbance with mean zero and finite variance The resistance parameter $\rho$ is generally unobservable, hence, the pesticide demand model is not directly estimable However, equation (9) can be substituted for the unobserved $\rho$ which yields

$$I = A\left[\frac{(1+\exp[\phi])\rho_{t0}}{1+\exp[\phi+\sigma Z]}\right]^{\beta} \prod X_i^{\alpha_i} + U \qquad (10)$$

Several coefficients of equation (10) excluding A and $\rho_{t0}$ may be consistently estimated by nonlinear ordinary least squares Results provide demand elasticities as well as an estimate of the inflection point $Z^* = -\phi/\sigma$ All parameters contained in equations (3) through (9) can be estimated if minimal extraneous information—that is an estimate of the dosage-response function for any period—is available

## DDT Example

To illustrate the theoretical model, we estimated a demand function for DDT and drew implications DDT was chosen because data were available over a long period Some compromises and assumptions with respect to variable definitions had to be made to account for aggregate data and structural changes in the agricultural community over time For DDT, the model is

$$I_t = B(1/(1+\exp[\phi+\sigma Z_t]))^{\beta}(P_{It}/P_{Rt})^{\alpha}(P_{St}/P_{Rt})^{\alpha} + U_t$$

where

$I_t$ = DDT quantity demand (pounds per acre),
= 0 5$Q_t/A_t$, where 0 5$Q_t$ = DDT domestic disappearance (1,000 pounds) and $A_t$ = cotton acres harvested (1,000 acres),

B = $A((1+\exp[\phi])\rho_0)^{\beta}$ (a constant),
$P_{It}$ = DDT wholesale price per pound (in dollars),
$P_{St}$ = Parathion wholesale price per pound (in dollars),
$P_{Rt}$ = Cotton season-average price per pound received by farmers (in dollars),
$Z_t$ = Measure of selection pressure in previous generations,

$$= \sum_{i=1}^{t-1} I_i \bullet$$

All data employed are contained in *Agricultural Statistics* and *The Pesticide Review* for the 1950-70 period The demand variable, DDT, is expressed in pounds per acre of cotton to account for substantial acreage shifts which have occurred over time We have deflated the price of DDT and the price of a substitute insecticide parathion, by an index of prices received by farmers to incorporate an aspect of derived demand

Finally, selection pressure at time t is defined as cumulative per acre pounds of DDT during the previous t-1 years A subroutine called & Dogleg in the TROLL/1 software package was employed for nonlinear estimation The estimated model is

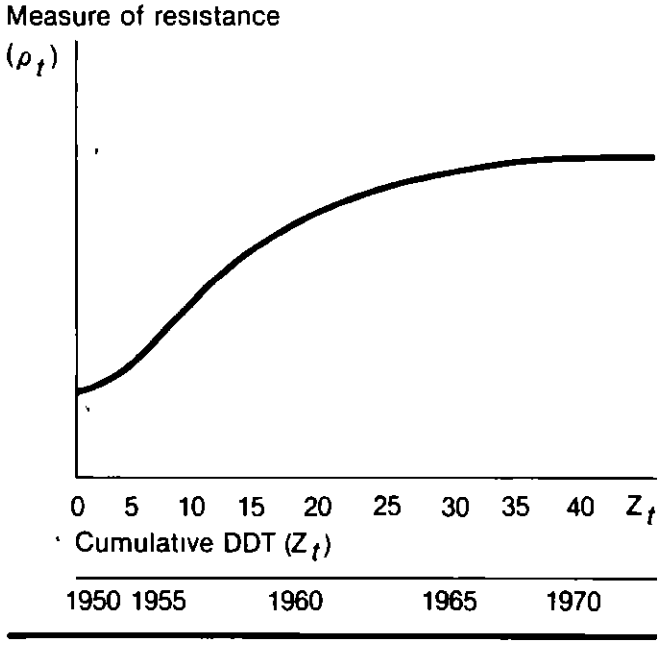
$$I_t = \underset{(0\ 401)}{0\ 9577}(1/(1+\exp[\underset{(1359\ 870)}{29\ 3510} - \underset{(141\ 175)}{3\ 0544}Z_t]))^{\underset{(1\ 703)}{0\ 0366}}$$

$$(P_{It}/P_{Rt})^{\underset{(0\ 351)}{-0\ 3306}} \quad (P_{St}/P_{Rt})^{\underset{(0\ 341)}{0\ 6077}}, \quad R^2 = 0\ 54 \qquad (11)$$

Asymptotic standard errors are reported in parentheses Note the estimated coefficients in equation (11), for which *a priori* expectations exist have the expected sign and are of plausible magnitude That is, the price elasticity of DDT ($P_{It}/P_{Rt}$) is negative, and the price elasticity of the substitute chemical, parathion, ($P_{St}/P_{Rt}$), is positive The resistance elasticity is 0 0366 and suggests that during the sample period, additional DDT was applied to compensate for its declining effectiveness This implies DDT use contributed to real or perceived positive side and cross resistance impacts for other pesticides during much of the sample period As with the previous study by Carlson, a high degree of statistical significance cannot be attached

Figure 3

**Resistance Development for DDT, 1950-70**




to many of the individual parameter estimates This may be attributed to the highly aggregative nature of the data and the relatively low number of degrees of freedom This phenomenon is likely to occur until more reliable data become available

Analysis of the resistance coefficients, $\phi$ and $\sigma$, yields another interesting conclusion Figure 3 represents resistance development over time for DDT using the estimates from equation (11) Complete specification is possible given an estimated dosage-response curve for one of the major pests of cotton during any year of the sample period The inflection point ($Z^* = -\phi/\sigma$) of this curve equals 9 61 This approximately corresponds to the years 1956-57 and coincides with reports of DDT-resistant boll weevils in cotton during the late fifties (*9*) Furthermore, figure 3 suggests that the effectiveness of DDT had been substantially depleted by the early sixties and that the upper bound on the resistance parameter, $\tau$, was essentially reached by 1970 Despite its declining effectiveness, average per acre applications of DDT remained relatively stable after the inflection point, $Z^*$, as indicated by the length of the 5-year intervals The horizontal axis of figure 3 measures accumulated dosages of DDT, the time scale indicates the years in which accumulated dosages were realized

Based on our results, it seems plausible that careful resistance management (derived from information in fig 3) and the extraneous estimates of DDT effectiveness might have prolonged the viability of DDT in insect control and delayed the need to introduce other more environmentally hazardous substitutes This hypothesis might be more clearly indicated if the estimated form of equation (11) were substituted into an optimization framework similar to that suggested by Hueth and Regev

## Conclusion

Sound economic analysis of behavioral relationships in the agricultural sector requires researchers to include relevant economic, institutional, and biological variables All too frequently, some of the important variables are unmeasurable or data are simply not available Demand models for pesticides are an important case in point Researchers know pest resistance to different toxic chemicals significantly reduces pesticide effectiveness which in turn, should affect pesticide demand Pest resistance data are not available and are, therefore generally not included in pesticide demand models The procedure used here replaced the unmeasurable resistance variable in the demand model with a measurable variable based on accumulated applications of pesticides We estimated the demand function for DDT to illustrate the practicality of the model

Several important conclusions can be drawn First, parameters important for resistance management are available as a byproduct of demand analysis and can be estimated without extensive data requirements Second, given the logistic specification for resistance development, resistance management can be important during the early stages of resistance development Third if growers respond to increasing resistance by applying additional pesticides, as they did in the case of DDT, pesticide cancellation will not result in significant additional cost in terms of present patterns of resistance management Fourth, the model can be generalized to alter our current specification of selection pressure or to add additional variables believed to influence resistance development Finally, resistance development has many similarities with diffusion literature (6, 7, 10 12) Further analysis may help us understand resistance development and pesticide demand and also promote sounder policy decisions in this heavily regulated and environmentally important sector

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## In Earlier Issues

Sampling is particularly fruitful in agricultural statistics [An] important reason is the reduction of heavy workloads in the Government agencies that conduct surveys [There have been] efforts to convert one-time censuses and large-scale current enumerations to a sample-survey basis

*Heinrich Strecker*
*Vol 7 No 1 Jan 1955, p 12*