Chapter #18: Pesticide Economics

Contents:

General Overview

There are three major classes of pesticides:

(1) Insecticides
(2) Fungicides
(3) Herbicides

Pesticides are useful in controlling agricultural pests, but must be chemically updated over time as pests build resistance. There are adverse human and animal health effects associated with pesticide use, as well. The adverse human health effects of different types of pesticides tend to depend on the similarity between human biology and the biology of the target pest:

- That is, a "monkeycide" would be worse for human health than a "raticide"
- Fungicides are generally worse for human consumption than insecticides

Our focus in this chapter will be to understand the following concepts:

(1) Economic models of pesticide use.
(2) Health effects of pesticides.
(3) Pesticides policies and regulations.

A Brief History of Pesticide Use

Herbicides

From 1965 to 1980, growth in the relative price of labor increased the use of herbicide as a factor of production. This occurred because herbicide is a substitute for labor-intensive work. During the 1980s, lower agricultural commodity prices and reduced crop acreage led to a reduction in herbicide use.

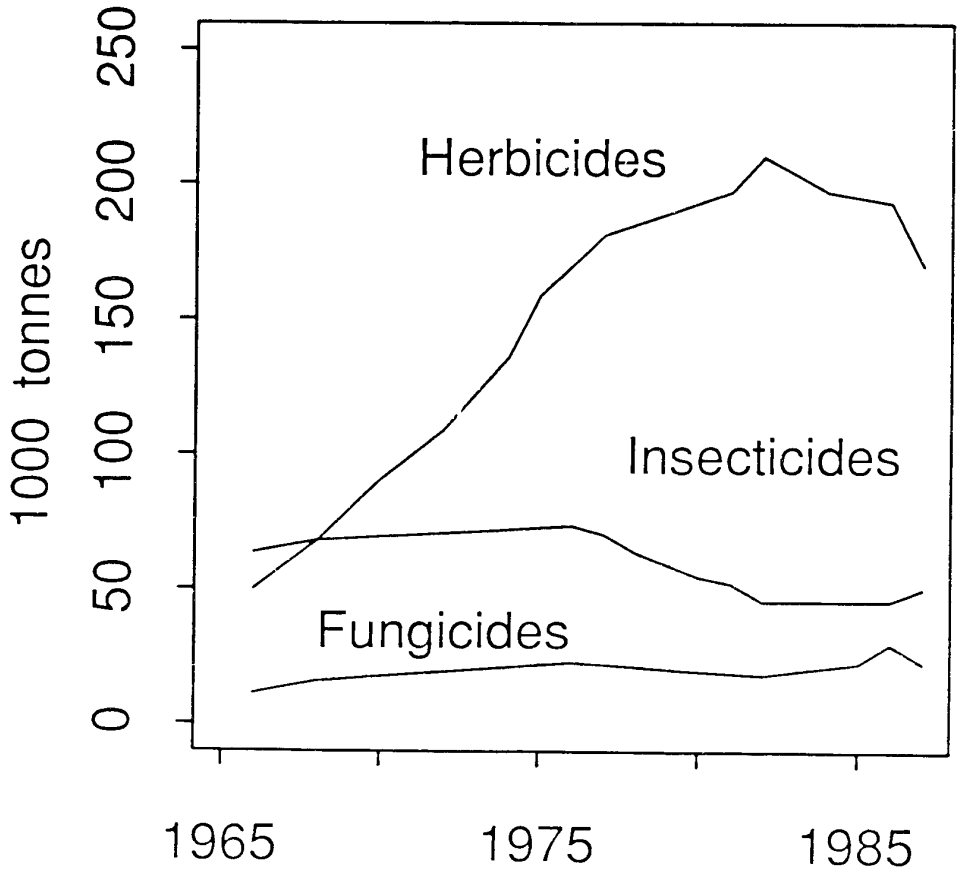
Insecticides

During the 1970's, the creation of the EPA and an increase in energy prices led to a reduction in insecticide use.

Fungicides

Fungicide use has remained relatively stable over the past 30 years, although recent legislation banning the use of carcinogenic chemicals in the Delaney Clause will soon outlaw many fungicides (and several popular insecticides and herbicides).

Figure 18.1: An Overview of Changes in Pesticide Use in the U.S.



Pesticides in a Damage Control Framework

Let's set up the variables for a relatively simple pesticide problem. Pesticides are damage control agents, therefore, we can assess the impact on crop damage from the amount of pesticide used.

Output: $Y = g(Z)[1 - D(n)]$

where

$g(Z)$ potential output (Z are inputs not related to pest control)
$D(n)$ damage function (percent of output lost to pest damage)

Pest Population After Pesticide Use:

$$n_1 = h(n_0, X, A)$$

where

n_0 pest population without pesticide

X level of pesticide applied
A alternative pest control method level, such as IPM

and where: $h_X < 0, h_A < 0$

The Economic Threshold

Obviously, there are costs associated with pesticide application. If the total damage from pests is less than the social cost associated with a single application of a pesticide to a field (including MEC), then the welfare-maximizing level of pesticide use is zero. Note that this implies toleration of some pests in the field as well as toleration of the associated pest damage, such as less aesthetically appealing fruits and vegetables.

When the level of pest damage rises above the social cost of a pesticide application, then it is welfare-maximizing to apply the pesticide. Profit maximization in the private market, however, seeks to maximize profit. The **economic threshold,** $\bar{n}_0$, is the pest population level at which it becomes profit-maximizing to apply the pesticide. The economic threshold is determined by setting total pest damage equal to the total cost of a single pesticide application and solving for $\bar{n}_0$:

$$Pg(Z)D(\bar{n}_0) = w$$

where the value of pest damage to output (at the threshold level) is $Pg(Z)D(\bar{n}_0)$ and w is the cost of applying pesticide. Given function forms for g and D, one could solve the above equation for $\bar{n}_0$.

In the models that follow, we will assume that the pest population is above the economic threshold.

Model of Pesticide Use with Known Pest Population and Pest Control Alternatives

The optimal level of pesticide use is determined by solving:

$$\underset{X,A}{Max}\left\{Pg(Z)\left[1 - D\left(h(n_0, X, A)\right)\right] - VA - WX\right\}$$

where:

P output price
W pesticide price
V price of alternative control methods (i.e., IPM)

The FOC's are:

$$\frac{dL}{dX} = -Pg(Z)D_n h_X - W = 0$$
$$\frac{dL}{dV} = -Pg(Z)D_n h_A - V = 0$$

Thus, one would maximize profits by applying pesticides until the value of marginal product (marginal benefit) of pesticide application equals the marginal cost of pesticide application. The model predicts that the use of pesticides will increase following:

- An increase in initial pest population threshold ($\bar{n}_0$),
- An increase in the output price (P),
- An increase in potential output $g(Z)$,
- An increase in the price of alternative controls, or
- A decrease in the price of pesticides.

Analogous results hold for the alternative pest control method.

A Model with a Secondary Pest

In the model of a secondary pest, we will not consider any direct alternative controls besides the use of pesticides. In this case, the alternative control can be thought of as an indirect control, given the biological relationship between the two pest populations.

Assume that two pests cause damage, and both populations are known, but pest 1 is also a *predator* of pest 2.

Damage = $D(n_1, n_2)$

$n_1 = n(n_0, X_1)$

$n_2 = \Psi(n_1)$ with $\Psi_{n1} < 0$

Using pesticide on pest 1 may lead to an increase in the population of pest 2, since pest 1 is a predator of pest 2. This changes the problem to:

$$Max\left\{ Pg(Z)\left[1 - D\left(h\left(n_0, X, \Psi(n_1)\right)\right)\right] - WX \right\}$$

with F.O.C.:

$$-Pg(Z)\left[D_{n1}h_x + D_{n1}h_{n2}\Psi_{n1}\right] - W = 0$$

Thus, both the direct impact on n_1 and indirect impact on n_2 on crop damage have to be considered on determining X. Lack of recognition of biological predator-prey relationships may lead to economically inefficient over-application of pesticides, since the beneficial effect of the predator pest on reducing pest 1 is ignored.

Pesticide Resistance

Through the biological process of natural selection, pests exposed to pesticides gradually develop genetic resistance to pesticides. Higher levels of pesticide application may accelerate buildup of resistance due to genetic selection of resistant genes. Short run pesticide control problems in a given season will be inefficient if long term resistance effects are not considered. Therefore, the calculation of optimal dosage of pesticide should take into account:

- resistance buildup (pesticide effectiveness is an "exhaustible resource" and should be modeled as such)
- use of alternative chemicals or alternative pest control methods (such as the use of alternative cropping methods, crop rotation, natural diseases and predator-prey relationships).

Unknown Pest Populations and Pest Population Monitoring

When pest populations are unknown, as is usually the case, one can distinguish between *preventive* and *reactive* pesticide application. It turns out that with pesticide application, preventing may be worse (less efficient) than reacting.

With **preventive application**, pesticides are applied without an attempt to determine potential pest populations. Instead, based on experience or historical data, the farmer makes educated guesses about the probabilities of various pest population levels occurring. The farmer then chooses a level of pesticide use to maximize expected profit. For example, suppose there are two possible pest levels, n_1 and n_2:

$$\max_X E(p) = p\{Pg(Z)[1 - D(h(n_1, X))] - wX + (1 - p)\{Pg(Z)[1 - D(h(n_2, X))] - wX$$

where: p is the probability of pest population n1 occurring
$1-p$ is the probability of pest population n2 occurring
$n_1 < n_2$

the FOC is:

$$p\{-Pg(Z)D_h h_X(n_1) - w\} + (1-p)\{-Pg(Z)D_h h_X(n_2) - w\} = 0$$

Given specific g, D, and h functions, one could solve this FOC for X. Plugging this X back into the objective function would then give the level of expected profit associated with preventive pesticide application.

Note that because the pest population is uncertain, X will be the same, regardless of which pest population level, n_1 or n_2 , actually occurs. This is inefficient, because we would like to use less pesticide if n_1 occurs and more if n_2 occurs. To decide between preventive and reactive application methods, we will compare the level of expected profits under preventive pesticide application with the level of expected profits under the following model of reactive application.

With **reactive application**, a fixed monitoring cost is paid to determine the pest population level, and then the optimal X is chosen for the specific pest level. This enables more precise pesticide use.

$$\max_{X_1, X_2} E(p) = p\{Pg(Z)[1 - D(h(n_1, X_1))] - wX_1\} + (1-p)\{Pg(Z)[1 - D(h(n_2, X_2))] - wX_2\} - m$$

where p is the probability of pest population n_1 occurring
1-p is the probability of pest population n_2 occurring
$n_1 < n_2$
m is the fixed cost of monitoring

the FOC's are:

(1) $p\{-Pg(Z)D_h h_{X1}(n_1) - w\} = 0$
(2) $(1-p)\{-Pg(Z)D_h h_{X2}(n_2) - w\} = 0$

Given specific g, D and h functions, one could solve the FOC's for the optimal X_1 and X_2. Plugging X_1 and X_2 back into the objective function gives the level of expected profits under reactive pesticide application. Note that the resulting equation for expected profits will contain monitoring costs, m. With reactive application, there is a tradeoff between monitoring costs, m, and the savings in pesticide costs made possible by monitoring.

If the difference between X_1 and X_2 is large, and m is relatively small, then reactive application will give a higher level of expected profits than would preventive application. Monitoring and reactive application are key components of modern Integrated Pest Management (IPM) programs. However, even if the difference between X_1 and X_2 is large, farmers may still use preventative application, because the price of pesticides is very cheap or the cost of IPM is relatively high.

In order to get align the profit-maximizing decision with social-welfare maximization, an appropriate tax could be placed on pesticides so that prices would reflect MEC.

Regional Cooperation in Pest Control Activities

Obviously, pests do not recognize property rights. When it comes to resistance, either using or not using chemical treatment might lead to externality problems. **Pest control districts** are introduced to overcome these problems, e.g., mosquito control districts. Activities of such organization include joint efforts in monitoring activities, coordinating crop management and rotation, and coordinating pesticide spraying.

Health-risk and Environmental Effects of Pesticide Use

Health-risk is the probability that an individual selected randomly from a population contracts adverse health effects (mortality or morbidity) from a substance. The health risk-generating process contains three stages:

(1) contamination
(2) exposure
(3) dose response:

Contamination is the result of pesticide application. The chemicals are spread through the air and water and are absorbed by the product.

Exposure may result from many activities:

- Exposure may be from eating, breathing, and touching.
- For food safety, the exposure is to the consumer.
- For worker safety, the exposure is to the applicator, mixer, and factory worker.
- For ground water, exposure is to whoever drinks and bathes in the water
- For environmental risk, exposure is to the species who are exposed to the risk.

Dose/response relationship translates exposure to probability of contracting certain diseases.

- Acute risks are immediate risks of poisoning.
- Chronic risks are risk that may depend on accumulated exposure and which may take time to manifest themselves, for example, cancer.

Risk Assessment Models

The processes that determine contamination, exposure, and the dose/response relationship are often characterized by heterogeneity, uncertainty, and random phenomena (weather). Thus, contamination, exposure and the dose/response relationship often exhibit the characteristics of **random variables**. Random variables are variables, which can take on several values, depending on the outcome of some random process, or depending on the outcome of some process which is so complicated that outcomes appear random. When modeling random variables, we often work with models that contain probabilities. For example, r**isk assessment**

models estimate health risks associated with pesticide application by making use of estimated probabilities.

We can look at an example to familiarize ourselves with risk assessment. Let r = the represent individual Health Risk.

$$r = f_3(B_3)\, f_2(B_2)\, f_1(B_1, X)$$

where:

- X = Pollution on Site (i.e., the level of pesticide use)
- B_1 = Damage control activity at the site (i.e. protective clothing; re-entry rules)
- B_2 = Averting Behavior by individual (i.e., washing fruits and vegetables)
- B_3 = Dosage of pollution (i.e., the type of pesticide residual consumed)

The health risk of an average individual is the product of three functions:

(1) **$f_1(B_1,X)$** is the **contamination function**. The function relates contamination of an environmental medium to activities of an economic agent (i.e., relates pesticide residues on apples to pesticides applied by the grower)

(2) **$f_2(B_2)$** is the human **exposure coefficient**, which depends on an individuals' actions to control exposure (i.e., relates ingested pesticide residues to the level of rinsing and degree of food processing an individual engages in)

(3) **$f_3(B_3)$** is the **dose-response function** which relates health risk to the level of exposure of a given substance (i.e., relates the proclivity of contracting cancer to the ingestion of particular levels of a certain pesticide), based on available medical treatment methods, B_3. Dose-Response functions are estimated in epidemiological and toxicological studies of human biology

The product, **$f_2(B_2)\, f_1(B_1,X)$,** is equal to the **overall exposure level** of an individual to a toxic material.

- (amount of pesticide present on an apple)(% *not* removed by rinsing apple)

- the degree of overall exposure can be effected by improved technology and by a greater dissemination of information

Estimating these functions involves much uncertainty

1) **Scientific knowledge of dose-response** relationships of pesticides is incomplete since in some cases, pesticides consumed in small doses over long periods of time have great consequences which may be difficult to estimate.
2) **Contamination Function depends** partly **on assimilation of pollution** by natural systems, which can differ regionally (i.e., wind distributed residues).
3) **Exposure coefficient depends on education** of population (i.e., consumer awareness of pesticide residue averting techniques, such as washing).

Uncertainty is included in the economic model by using a **safety-rule approach**. Such an approach utilizes confidence intervals from toxicology studies in the framework and are beyond the scope of this course.

Policy Goal

The major objective of pesticide policy is to maximize welfare subject to the constraint that the probability of health risk remain below a certain threshold level, R, an acceptable percent of the time, t.

- R = the target level of risk
- t = the safety level (measures the degree of social risk aversion); t might represent the degree of confidence we have in our target risk actually occuring

For any target level of risk and any degree of significance, the model can be solved for the optimal levels of:

- Pesticide Use
- Damage Control Activities
- Averting Behavior by Consumers
- Preventative Medical Treatments

General Implications

The optimal solution involves some combination of pollution control, exposure avoidance, and medical treatment. The cost of reaching the target risk level increases with the safety level.

The shadow price of meeting the risk target depends on the degree of significance we have that the target is being met. the higher the safety level, or the greater the uncertainty we have in our estimate of risk, the higher the shadow value of meeting the constraint

Sample Problems

Now that we have outlined the concepts in pesticide policy, let's look at a numerical example. Say there is no uncertainty regarding the health effects of pesticide use. That is, toxicologists know with certainty a point estimate of the dose-response function for a society or a partiacular area with pesticide use.

Example 1

Let: X = the level of pesticides used on a field
A = the level of alternate pest control activities
P = the value of farm output (i.e., the price of a basket of produce)
Y = the level of farm output
W = the price of pesticide
V = the price of alternative controls (V > W)
r = the level of health risk in society
B_1 = damage control activities by the farm (i.e., pesticide reentry rules)
B_2 = aversion activities by members of the population (i.e., washing residues off)
B_3 = available level of medical treatment
Y = f(X,A) the farm production function (i.e., a pesticide damage function)
$r = f_3(B_3) f_2(B_2) f_1(B_1, X)$ is the Health Risk of pesticide use

The objective of the society is:

$$\underset{X, A r, B_1, B_2, B_3}{Max} \left\{ Pf(X, A) - C(r) - C(B_1, B_2, B_3) - WX - VA \right\}$$

$$\textit{subject to}: \; r = f_3(B_3) f_2(B_2) f_1(B_1, X)$$

which can be written in lagrangian form as:

$$\underset{X, A r, B_1, B_2, B_3}{Max} \left\{ = Pf(X, A) - C(r) - C(B_1, B_2, B_3) - WX - VA + \lambda \left[r - f_3(B_3) f_2(B_2) f_1(B_1, X) \right] \right\}$$

with the FOCs:

(1) $$\frac{d}{dA} = Pf_A - V = 0$$

the MRP of the alternative control equal the MC of the alternative control

(2) $$\frac{d}{dr} = -C'(r) + \lambda = 0$$

the MSC of Health Risk = shadow value of risk (The MC of risk in terms of social damages is equal to the shadow price of reducing societal risk.)

(3) $$\frac{d}{dX} = Pf_X - W - \lambda\left[f_3 f_2 \frac{df_1}{dX}\right] = 0$$

(4) $$\frac{d}{dB_1} = -C_{B1} - \lambda\left[f_3 f_2 \frac{df_1}{dB_1}\right] = 0$$

(5) $$\frac{d}{dB_2} = -C_{B2} - \lambda\left[f_3 f_1 \frac{df_2}{dB_2}\right] = 0$$

(6) $$\frac{d}{dB_3} = -C_{B3} - \lambda\left[f_2 f_1 \frac{df_3}{dB_3}\right] = 0$$

We can re-write equations (3) - (6) using equation (2) as:

$$Pf_X = W + C'(r)\left[f_3 f_2 \frac{df_1}{dX}\right]$$

The MRP of pesticides to the farm is equal to the MPC of pesticides plus the (MC of risk)(marginal contribution of pesticides to Health Risk)

$$C_{B1} = -C'(r)\left[f_3 f_2 \frac{df_1}{dB_1}\right] = 0$$

The MC of damage control equals (avoided MC of risk)(marginal improvement in risk from engaging in damage control activities)

$$C_{B2} = -C'(r)\left[f_3 f_1 \frac{df_2}{dB_2}\right] = 0$$

The MC of averting behavior equals (avoided MC of risk)(marginal improvement in risk from engaging in averting behavior):

$$C_{B3} = -C'(r)\left[f_2 f_1 \frac{df_3}{dB_3}\right] = 0$$

The MC of medical treatment equals (avoided MC of risk)(marginal

improvement in risk from engaging in medical treatment).

The optimal solution involves equating all (6) FOCs. Equations (2)-(6) can be expressed as:

$$\lambda = C'(r) = \frac{Pf_X}{\left(f_3 f_2 \frac{df_1}{dX}\right)} = \frac{-C_{B1}}{\left(f_3 f_2 \frac{df_1}{dB_1}\right)} = \frac{-C_{B2}}{\left(f_3 f_1 \frac{df_2}{dB_2}\right)} = \frac{-C_{B3}}{\left(f_2 f_1 \frac{df_3}{dB_3}\right)}$$

which says that the optimal solution involves equating the shadow price of risk with:

$$\frac{MRP_{pesticides}}{\left(\Delta \text{health risk from } \Delta \text{pesticides}\right)} = \frac{-MC_{damagecontrol}}{\left(\Delta \text{health risk from } \Delta \text{damage control}\right)} = \frac{-MC_{avertingbehavior}}{\left(\Delta \text{health risk from } \Delta \text{averting behavior}\right)} = \frac{-MC_{medicaltreatment}}{\left(\Delta \text{health risk from } \Delta \text{medical treatment}\right)}$$

The denominator of each expression transforms MB and MC of health-related activities into change in health risk.

When parameters are known, the model can be solved for the optimal levels. Some general implications of this type of model are:

(1) If there is no tax on pesticide use and no subsidy on farm-level damage control, then the farm will not recognize the effect of pesticide use on societal health, and operate as if λ=0.
   - An inefficiently high level of pesticides will be used
   - An inefficiently low level of damage control will be applied

(2) The optimal solution may involve a large level of pesticide use, little damage control, little medical treatment, and a high degree of averting behavior
   - Rinsing and washing produce may be the least expensive method of reducing health risk in society.

Example 2 (a model with uncertainty)

Let *r* be the probability of an individual contracting a disease.

$$r = c\ e\ d\ x$$

c = contamination probability
e = exposure probability
d = dose/response probability
x = amount of pesticide applied

Let:

$$c = \begin{cases} 1 \text{ with probability } 1/2 \\ 2 \text{ with probability } 1/2 \end{cases}$$

$$e = \begin{cases} 1 \text{ with probability } 1/2 \\ 3 \text{ with probability } 1/2 \end{cases}$$

$$d = \begin{cases} 10^{-5} \text{ with probability } 1/2 \\ 10^{-6} \text{ with probability } 1/2 \end{cases}.$$

For $x = 1$,

$$r = \begin{cases} 10^{-6} & \text{with probability } 1/8 \\ 2\ 10^{-6} & \text{with probability } 1/8 \\ 3\ 10^{-6} & \text{with probability } 1/8 \\ 6\ 10^{-6} & \text{with probability } 1/8 \\ 1\ 10^{-5} & \text{with probability } 1/8 \\ 2\ 10^{-5} & \text{with probability } 1/8 \\ 3\ 10^{-5} & \text{with probability } 1/8 \\ 6\ 10^{-5} & \text{with probability } 1/8 \end{cases}$$

Note: 10^{-6} means "one person per million people" contracts the disease. 10^{-5} means "one person per hundred thousand people" contracts the disease.

Then, expected risk is $\frac{13.2}{8}\ 10^{-5} = 1.65\ 10^{-5}$, or, one person in 165,000, on average contracts the disease. Yet the variability of this estimate is substantial, which implies that λ is large.

In many cases, the highest value (worst case estimator) of each probability is used when the risk generation processes are broken down to many sub-processes. This creates a "creeping safety" problem, in that the multiplication of many “worst case” estimates may lead to wildly unrealistic risk estimates. Of course, the variability and uncertainty associated with risk estimates can be reduced by expenditures on research and through information-sharing.

Pesticide Policy

Current pesticide policy separates pesticide economics from health considerations. New policy is triggered solely by health considerations: when a chemical is found to be carcinogenic or damaging to the environment, it is banned, or "canceled".

Economic Impacts of Pesticide Cancellation

The impacts of pesticide cancellation depend on the available alternatives. It there are no alternatives, then cancellation causes losses in crop yields due to higher pest damages and to increases in costs, since alternative methods of control are generally more expensive. If chemicals have alternatives, the impact is mostly on cost.

To estimate overall short-term impacts, the impacts on yield per acre and cost per acre are evaluated using one of the following methods:

Delphi method:

The delphi method uses "Guesstimates by experts", which are easy to obtain but are arbitrary and sometimes baseless. (Named after the famous Òracle at DelphiÓ in ancient Greece.)

Experimental studies:

These studies are based on data from agronomical experiments, but experimental plots often do not reflect real farming situations.

Econometric studies:

Statistical methods based (ideally) on data gathered from real farming operations. However, these studies are often not feasible because of data limitations and the difficulty of isolating the specific effects of pesticides.

Cost Budgeting Method:

y_{ij} =output per acre of crop *i* at region *j* with pesticides

P_{ij} = price crop *i*, region *j*

A_{ij} = acreage crop *i*, region *j*.

Δy_{ij} =yield reduction per acre because of cancellation

Δc_{ij} =cost reduction per acre because of cancellation

Under a partial crop budget, impacts on social welfare are estimated as:

$$\sum_{i=1}^{I}\sum_{j=1}^{J}\left(P_{ij}\Delta Y_{ij} + \Delta C_{ij}\right)A_{ij}$$

or, a pesticide cancellation causes losses in revenue from lower yields per acre and increased costs per acre, which is multiplied the total acreage in all regions and across all types of crop affected by the ban.

The cost budgeting approach has several limitations.

(1) It ignores the effect of a change in output on output price. This tends to overestimate producer loss and underestimate consumer loss.

(2) It ignores feedback effects from related markets.

In general, this method does not consider the interaction of supply and demand, and does not attempt to find the new market equilibrium after the application of a pesticide ban.

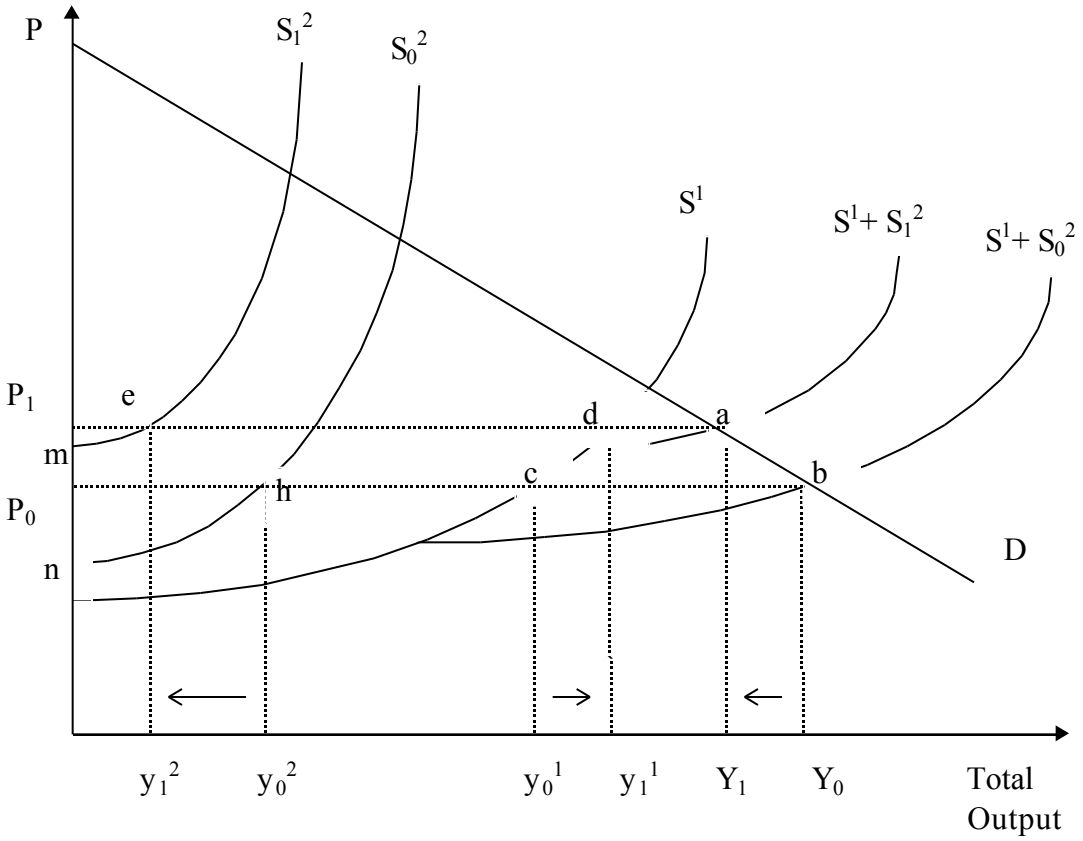
General Equilibrium Method

This method is based on analyzing the impact of a ban on *equilibrium* prices and output, taking into account the interaction of supply and demand and any feedback effects from related markets. In addition, this method offers a better assessment of equity effects by computing welfare changes for various groups.

As a result of a pesticide ban, marginal cost per acre increases, output declines, and output price increases. The magnitude of the change in output price depends on the elasticity of demand and any feedback effects from related markets, such as markets for substitute goods.

General equilibrium analysis recognizes heterogeneity in welfare effects: the welfare of non-pesticide-using farmers increases due to the increase in output price, but the welfare of pesticide-using farmers decreases if demand is elastic (but may increase if demand is inelastic). Consumer welfare decreases due to price increases.

Figure 18.2: If there are two agricultural regions, 1 and 2, and a pesticide is banned only in region 2



Sample Problem

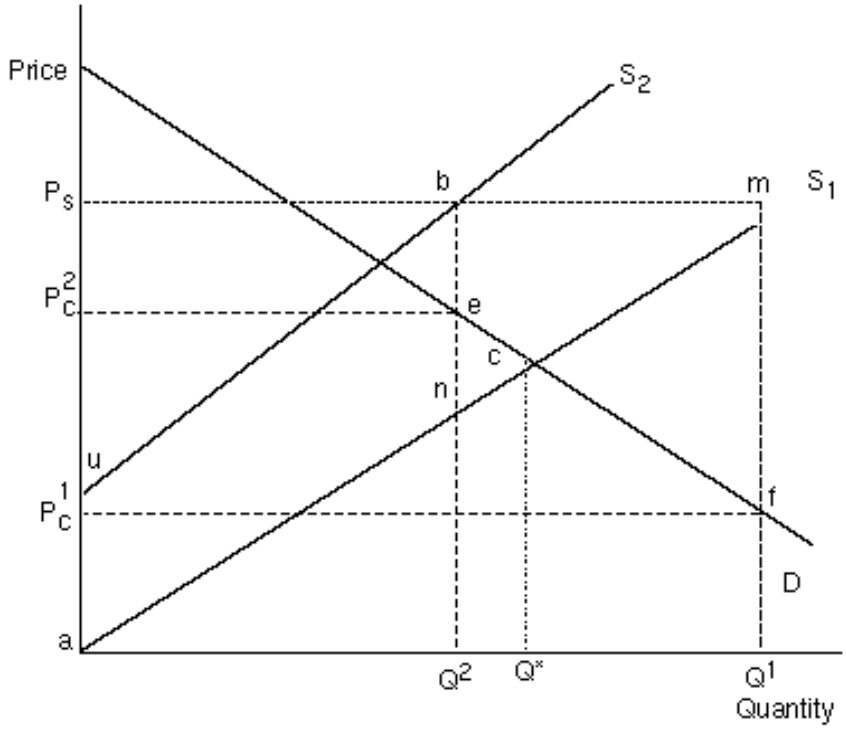
Let:

S^1	=	supply of region 1
S_0^2	=	supply of region 2 before pesticide ban
S_1^2	=	supply of region 2 after pesticide ban
$S^1 + S_0^2$	=	total supply before regulation, regions 1 and 2
$S^1 + S_1^2$	=	total supply after regulation, regions 1 and 2
Y_0	=	total output before regulation
Y_1	=	total output after regulation
y_0^1	=	output of region 1 before regulation
y_1^1	=	output of region 1 after regulation
$y_0^2 = Y_0 - y_0^1$	=	output before regulation, region 2
$y_1^2 = Y_1 - y_1^1$	=	output after regulation, region 2
P_1abP_0	=	consumer loss from cancellation
P_0cdP_1	=	producer gain, region 1
P_0hn-P_1em	=	welfare loss, region 2

We observe in Figure 18.1 that when the ban affects only one of two or more regions, growers in the regions without the ban gain from the pesticide ban. Thus, some farmers may support pesticide bans if they feel the effect of the ban other producers to a greater degree. For example, say farmers in region 2 grow pesticide-free produce.

In this case, agricultural price support policies may lead to oversupply, so that pesticide regulation may increase welfare by reducing excess supply.

Figure 18.3: Pesticide Regulation and Agricultural Policy



S_1 = supply before cancellation
S_2 = supply after regulation
P_S = price support
P_c^1, P_c^2 = output price before and after
Q^1, Q^2 = output before and after
welfare loss because of price support before regulation = mcf
welfare loss after ban = ubecna = ubna (extra cost) + ecn (under supply)

If price support is very high, pesticide cancellation reduces government expenditure substantially. This results in an increase in taxpayer welfare of area $P_s mf P_c^1 - P_s be P_c^2$. Consumer welfare declines by $P_c 1 fe P_c 2$. Producer surplus declines $P_s ma - P_s bu$.

Alternative Policies

Pesticide effects include several related issues:

Food safety
Worker safety
Ground water contamination
Environmental damage

Pesticide bans and taxes address all these issues. However, a pesticide ban can be an inefficient policy, since pesticide uses and impacts vary significantly across regions.

Economic mechanisms (taxes, partial bans) that discriminate across different types of uses may eliminate most of the pesticide damage but retain most pesticide benefits.

Although a pesticide ban may provide the incentive to develop new, less dangerous pest control methods, a pesticide tax may serve the same purpose and also allow a more gradual and efficient transition to the new technology. However, if a pesticide tax is used, policymakers should keep in mind that pesticide use patterns could shift significantly across geographic regions.

Other policy tools can affect different stages of the risk generation process:

-Pollution controls affect contamination
-Protective clothing
-Medical treatment affect dose/response
-Green markets
-Reentry regulations
-Established liability terms