Pesticide Economics

There are three major classes of pesticides:

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

A Brief History of Pesticide Use:

Herbicides:

From 1965 to 1980, growth in the relative price of labor increased the use of herbicide as an factor of production.

- because **herbicide is a substitute for Labor**

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).

Pesticides in a Damage Control framework:

Pesticides are damage control agents.

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

where
$g(Z)$ potential output (Z are inputs unrelated to pest control)
$D(n)$ damage function (% 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 level of alternative pest control, such as IPM.

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

The Economic Threshold

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.

- 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

- $Pg(Z)D(\bar{n}_0)$ is the value of pest damage to output (at threshold level)
- w is the cost of applying pesticide

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:

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

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

The model predicts that the use of pesticides will increase following:

- An increase in initial pest population (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.

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

$$\text{Damage} = D(n_1, n_2)$$
$$n_1 = h(n_0, X_1)$$
$$n_2 = \phi(n_1) \; with \; \phi_{n_1} < 0$$

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.

The new problem:

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

with F.O.C.:

$$-Pg(Z)\left[D_{n_1} h_x + D_{n_1} h_{n_2} \phi_{n_1}\right] - W = 0$$

both the direct impact on n_1 and indirect impact on n_2 on crop damage have to be considered on determining X.

ignoring predator-prey relationships may lead to economically-inefficient over-application of pesticides

Unknown Pest Populations and Pest Population Monitoring:

When pest populations are unknown, one can distinguish between *preventive* and *reactive* pesticide application.

With **preventive application**, pesticides are applied without an attempt to determine potential pest populations. The farmer makes educated guesses about the probabilities of various pest population levels occurring, and chooses a level of pesticide use to maximize expected profit.

Suppose there are 2 possible pest levels, n_1 & n_2 ($n_1 < n_2$):

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

where p is the probability of pest population n_1 occurring
1-p is the probability of pest population n_2 occurring

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 g, D, and h functions, one could solve this FOC for X.

because the pest population is uncertain, X will be the same, regardless of which pest population level, n_1 or n_2 , actually occurs.

inefficient because we would like to use less pesticide if n_1 occurs and more if n_2 occurs.

Unknown Pest Populations and Monitoring (cont.):

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.

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

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 g, D and h, one could solve for the optimal X_1 & X_2.

there is a tradeoff between monitoring costs, m, and the savings in pesticide costs made possible by monitoring.

- If the difference between X_1 & 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.

Health-risk and Environmental Effects of Pesticide Use

Health risk: probability that an individual selected randomly from a population contracts adverse health effects from a substance. The health risk-generating process contains three stages:

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

Contamination is the result of pesticide application. The chemicals are spread through the air and water and become 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, 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.

The **dose-response relationship** translates exposure to probability of contracting certain diseases.

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

A Risk-Assessment Model:

Let r = the represent individual Health Risk

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

- X = Pollution on Site (level of pesticide use)
- B_1 = Damage control activity at the site (protective clothing)
- B_2 =Averting Behavior by individual (wash fruits & veg.)
- B_3 = Is the dosage of pollution (pesticide residual consumed)

Health risk of an avg. individual is the product of 3 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 (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 (relates ingested pesticide residues to the cleaning 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 (relates the proclivity of contracting cancer to the ingestion of pesticides), based on available medical treatment methods, B_3.
    - Dose-Response functions are estimated in epidemiological and toxicological studies of human biology

$f_2(B_2)\, f_1(B_1,X)$ = the **overall exposure level** of an individual
    - (amt. of pesticide on an apple)(% *not* removed by rinsing)
    - the degree of overall exposure can be effected by improved technology and by a greater dissemination of information

Risk-Assessment Model (cont.)

Estimating these functions involves much uncertainty

- **Scientific knowledge of dose-response** relationships of pesticides incomplete
  - pesticides consumed in small doses over long periods of time
- **Contamination Function depends** partly **on assimilation of pollution** by natural systems, which can differ regionally (wind distributed residues)
- **Exposure coefficient depends on education** of population (are consumers aware of pesticide residue averting techniques, such as washing?)

Uncertainty is included in the economic model by using a **safety-rule approach**.

Policy Goal: 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, α.

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

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

Risk-Assessment Model (cont.)

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 α

  - 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 α, or the greater the uncertainty we have in our estimate of risk, the higher the shadow value of meeting the constraint

Risk Assessment Model: An 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.

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 (pesticide reentry rules)
B_2 = aversion activities by members of the population (washing residues off)
B_3 = available level of medical treatment

Y = f(X,A) is the farm production function (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:

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

with the FOCs:

(1) $\frac{d\mathcal{L}}{dA} = Pf_A - V = 0$ (MRP=MC)

(2) $\frac{d\mathcal{L}}{dr} = -C'(r) + \lambda = 0$ (MSC=shadow value)

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

(4) $\frac{d\mathcal{L}}{dB_1} = -C_{B_1} - \lambda\left[f_3 f_2 \frac{df_1}{dB_1}\right] = 0$

(5) $\frac{d\mathcal{L}}{dB_2} = -C_{B_2} - \lambda\left[f_3 f_1 \frac{df_2}{dB_2}\right] = 0$

(6) $\frac{d\mathcal{L}}{dB_3} = -C_{B_3} - \lambda\left[f_2 f_1 \frac{df_3}{dB_3}\right] = 0$

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

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

--MRP of pesticides = MPC of pesticides + (MC of risk)(marginal contribution of pesticides to Health Risk)

$$C_{B_1} = -C'(r)\left[f_3 f_2 \frac{df_1}{dB_1}\right] = 0$$

--MC of damage control = (avoided MC of risk)(marginal improvement in risk from engaging in damage control activities)

$$C_{B_2} = -C'(r)\left[f_3 f_1 \frac{df_2}{dB_2}\right] = 0$$

--MC of averting behavior = (avoided MC of risk)(marginal improvement in risk from engaging in averting behavior)

$$C_{B_3} = -C'(r)\left[f_2 f_1 \frac{df_3}{dB_3}\right] = 0$$

MC of medical treatment = (avoided MC of risk)(marginal improvement in risk from engaging in medical treatment)

Risk-Assessment Model: An Example (cont.)

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

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

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:

- If there is no tax on pesticide use, $t^* = \lambda$, and no subsidy on farm-level damage control, $s^* = \lambda$, then the farm will not recognize the effect of pesticide use on societal health, and operate as if $\lambda = 0$.
  - inefficiently high level of pesticides will be used
  - inefficiently low level of damage control will be applied
- 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.

Risk Assessment: Example 2 (a model with uncertainty)

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

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

$$c = \begin{matrix} 1 \text{ with probability } 1/2 \\ 2 \text{ with probability } 1/2 \end{matrix}; \quad e = \begin{matrix} 1 \text{ with probability } 1/2 \\ 3 \text{ with probability } 1/2 \end{matrix}$$

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

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/million people" contracts the disease.

expected risk is $\frac{13.2}{8}\ 10^{-5} = 1.65\ 10^{-5}$, or, one person in 165,000,

--variability of this estimate is large => α is large.

creeping safety problem: the multiplication of many "worst case" estimates may lead to wildly unrealistic risk estimates

Economic Impacts of Pesticide Cancellation

Delphi method:

The delphi method uses "Guesstimates by experts", which are easy to obtain but are arbitrary and sometimes baseless.

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:

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

a pesticide cancellation causes losses in revenue from lower yields per acre and increased costs per acre

Economic Impacts of Pesticide Cancellation (cont.)

The cost budgeting approach has several limitations

(1) It ignores the effect of a change in output on output price;

- tends to overestimate producer loss and underestimate consumer loss

(2) It ignores feedback effects from related markets.

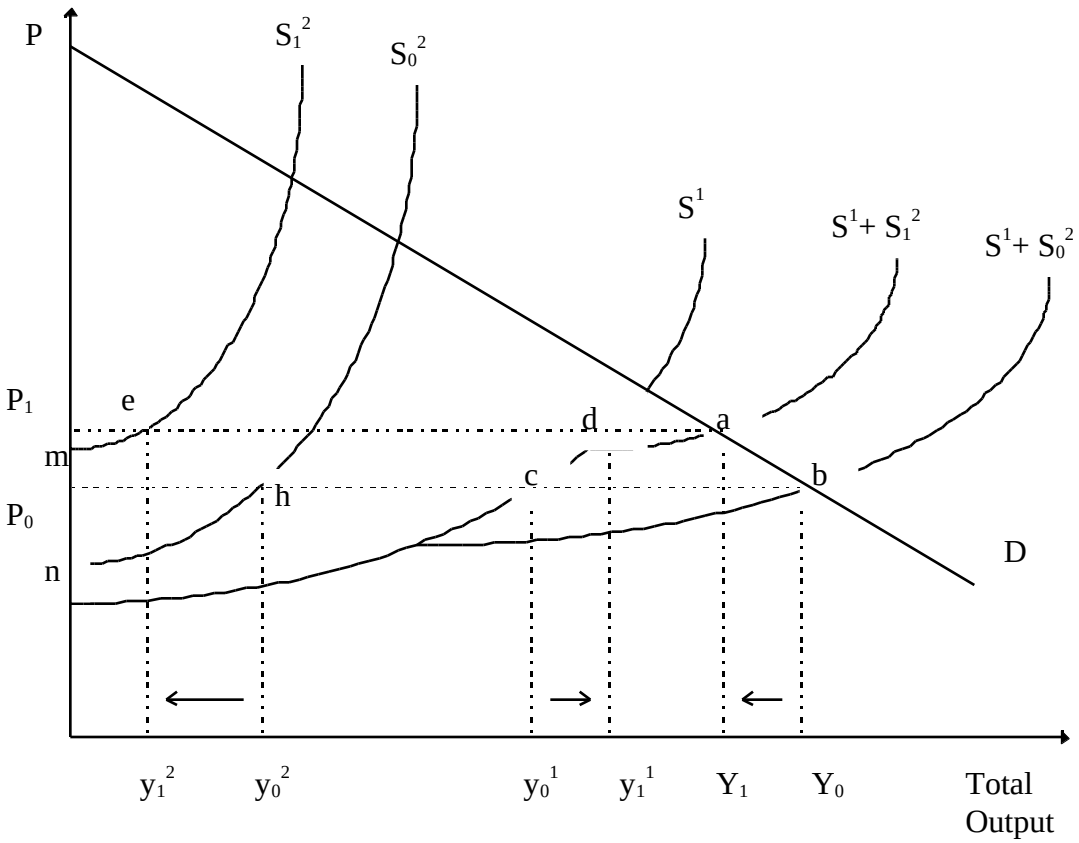
General Equilibrium Method

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.

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.

There are two agricultural regions,1 and 2,and a a pesticide is banned only in region 2.

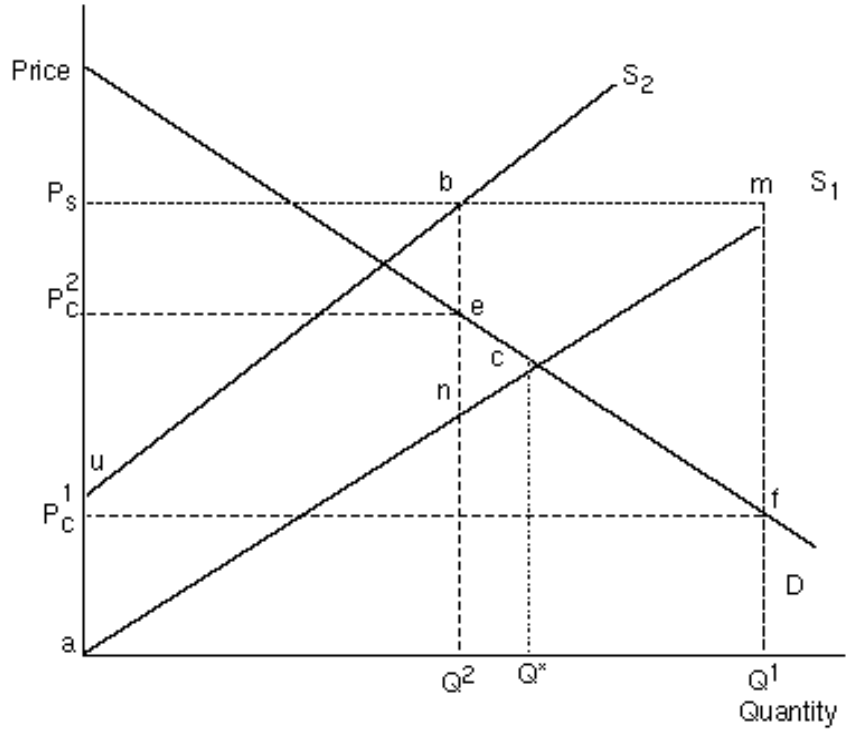


S^1 = supply of region 1; S_0^2 = supply of region 2 before pesticide ban
S_1^2 = supply of region 2 after pesticide ban
$S^1+S_0^2$ = total supply before regulation, regions 1 and 2
$S^1+S_1^2$ = total supply after regulation, regions 1 and 2
Y_0= total output before regulation; Y_1= total output after 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

Result: When the ban affects only one of two or more regions, growers in the regions without the ban gain from the pesticide ban

Pesticide Regulation and Agricultural Policy

Agricultural price support policies may lead to oversupply, so that pesticide regulation may increase welfare by reducing excess supply.



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

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

(1) Pollution controls affect contamination.
(2) Protective clothing affect exposure.
(3) Medical treatment affect dose/response.

Green markets
Tolerance standard? ? address water safety concerns

Reentry regulation
Protective clothing? ? addresses worker safety concerns

Liability
Water disposal regulation? ? affects ground water contamination