Valuing Food Safety and Nutrition

EDITED BY
Julie A. Caswell

Book Originally Published by
Westview Press, Boulder, Colorado, 1995

PART FOUR: Inputs to Valuation Studies

18. Measuring the Food Safety Risk of Pesticides

Kelly A. Day, Betsey A. Kuhn, and
Ann M. Vandeman

Food Marketing Policy Center
Department of Agricultural and Resource Economics
University of Connecticut

Measuring the Food Safety Risk of Pesticides

Kelly A. Day

Betsey A. Kuhn

Ann M. Vandeman

Keywords: Pesticides, food safety, risk weighting, total risk

18

Measuring the Food Safety Risk of Pesticides

Kelly A. Day, Betsey A. Kuhn, and Ann M. Vandeman[1]

Public concern about the use of pesticides and their possible effects on food safety has been increasing in recent years. Lawmakers are seeking to address these concerns, while assuring an abundant and affordable food supply. Whether or not new legislation is enacted, a tradeoff continues to exist between the reduction of pesticide residues and maintaining the current system of agricultural chemical use. The economic benefits of pesticide use are relatively easy to measure, given estimates of yield losses from pest damage. However, the economic costs of pesticide use, in terms of human health effects, are extremely difficult to measure. Even harder to assess are the benefits to human health of incremental reductions in pesticide residues. Were there a clearer measure of these benefits, policy makers might have better information about the food safety effects of pesticides and therefore more easily determine economically and socially rational pesticide policies.

In this chapter, we have attempted a first step towards the valuation of pesticide reduction—the construction of a risk-weighted measure of food safety. While this work is in an infant stage, we hope it will focus attention on the issues underlying pesticide risk and human health concerns.

Consumer surveys indicate that the public is concerned about the risks associated with consuming foods which contain pesticide residues. Horowitz and Carson (1990), for example, report that on a cost per death delayed basis, consumers would rather reduce the risks associated with pesticide residues on foods than those associated with PCBs in drinking water, radiation from the sun from use of CFCs, or automobile exhaust. Another survey (Sachs et al. 1987) compared consumers' concerns about pesticides in 1965 and 1984. The percent of respondents "with a great deal or some concern about the danger of eating fruits and vegetables that have been sprayed or dusted with pesticides" increased from 41.5 percent in 1965 to 71.1 percent in 1984.

In this chapter, we use the level of pesticide residues detected on a market basket of food prepared for home consumption as a proxy for the food safety risk associated with pesticides. We do not include other types of food safety risk, such as microbial contamination or natural toxins associated with food consumption (see, e.g., Roberts and van Ravenswaay 1989, Ames et al. 1987). We also do not offset the nutritional benefits of the food contained in the market basket against the pesticide residues detected on the food. Thus, the level of residues detected in a typical diet is not intended as a measure of general food safety. It is a measure of food safety risks associated with pesticide use and is appropriate for the purpose of investigating changes in these risks. If our purpose was to value the broad benefits of pesticide use, we might wish to include nutritional gains from supply changes attributable to the use of pesticides. The definition of benefits and costs, as well as the approach used to evaluate them, will greatly influence the valuation process.

A Method of Measuring Pesticide-Related Food Safety Risk

The Total Diet Study

A method for estimating food safety risk related to pesticides must be based on the most accurate measure of the chemical residues that humans consume in small quantities over time. The pesticide residue data we chose for this analysis were collected by the Federal Food and Drug Administration (FDA) in their Total Diet Study.[2] The Total Diet Study is the most appropriate source of data for this project because it measures actual residues in a typical diet drawn from a market basket of food. The study is conducted to determine levels of chemical contaminants, including pesticide residues, in foods prepared for consumption, and thus is the closest approximation to the average consumer's exposure to pesticides through the diet. The study has been conducted on a yearly basis since 1961, providing the only continuous time series of data on pesticide residues in the human diet.[3]

In order to determine the residues present in human diets, FDA selects a market basket of food to represent typical consumption patterns. These market baskets are based on nationwide dietary surveys, and have been revised with changes in the typical diet throughout the history of the study.[4] Calorie counts also have been revised to reflect changing consumption patterns. The food is purchased at the retail level and is fully prepared for consumption. It is then tested for the presence of residues at FDA laboratories. The resulting estimates of residue concentrations are weighted according to the food consumption survey and summed to calculate daily intake figures for each chemical.

We used the residue data reported for men 15 to 20 years-old[5] to represent food safety risk. The Total Diet Study assessed other groups, including infants,

children, and the elderly. Infants and children were first included in the study in 1975, with the definition of infants being revised in 1982. Considerable attention has been focused on the effects of pesticide residues on this group. While residues for all age groups fall well below acceptable daily intake levels,[6] infants and children appear to have the greatest level of exposure (Archibald and Winter 1989). However, we did not attempt to assess their food safety risk for two reasons. First, data for this group were not collected during the first fourteen years of the Total Diet Study. Fifteen to 20 year-old males are the only age/sex group for whom residue data are reported consistently over the entire study period. Second, while research suggests that the risks of pesticide exposure may differ for children, how and to what degree risks are different is unclear (National Academy of the Sciences 1993a). Therefore, estimating the relative risks for children as compared to adults was not possible given the available information.

In order to correct for the effect of changes in caloric intake on residue levels, we standardized the data to 2,520 calories, equal to the current estimate of total calories consumed by 15 to 20 year-old males.

A Risk-Weighted Measure of Food Safety

To construct a measure of food safety risk arising from pesticide residues, we needed to take account of a number of types of risk and differences in the level of risk for each of the 78 chemicals reported. Pesticides vary greatly in their toxic effects. For example, chlordane is a known carcinogen, while sulfur pesticides have no known carcinogenic properties. We reviewed the available toxicological data to evaluate the risks associated with each pesticide.

Several measures have been developed to evaluate the potential for adverse physiological reactions from exposure to pesticides. The possible effects fall into two broad categories: acute toxicity, which generally refers to the damage resulting from a single dose or exposure, and chronic toxicological effects, which are health effects caused by repeated exposures to a substance over an extended period.

The most commonly found acute toxicity measure is the LD_{50} (median lethal dose), or the amount of a substance necessary to kill 50 percent of a sample population when exposed in a single dose. The LD_{50} is used to measure both oral and dermal acute toxicity. Expressed as milligrams of chemical per kilogram of body weight, the lower the LD_{50}, the more toxic the chemical. Hammitt used the reciprocal of the LD_{50} multiplied by 100 to form an *acute index* (Hammitt 1986). Acute toxicity is most relevant to populations who come in direct contact with a chemical, such as in the manufacture, mixing, loading, and application of pesticides. Because we sought a measure of the risks of pesticide residues for food consumers, we used indicators of chronic rather than acute risk.

A comprehensive indicator of the chronic health risk associated with exposure to pesticides needs to take into account a number of possible health effects. These include:

1. Oncogenicity: equivocal tumor producing, unknown carcinogenicity,
2. Carcinogenicity: cancer producing,
3. Mutagenicity: causing unnatural changes in cells, in vitro,
4. Teratogenicity: causing birth defects,
5. Neurotoxicity: destructive to nerves or nerve tissue,
6. Immunotoxin: destructive to the immune system,
7. Damage to the reproductive system, and
8. Damage to other organ(s).

There are several chronic risk measurements. However, the primary focus of chronic health risk assessment has been limited to consideration of carcinogenicity and oncogenicity.

The Q* Rating. The Q* is an estimate of the upper bound of extra incidents of tumor formation in humans that can be expected given a 70 year lifetime exposure to a certain dose of a chemical (both from dietary and other exposures). It is equal to the slope of the dose response curve from animal studies, and is expressed in terms of tumors/milligram of pesticide/kilogram of body weight/day. It does not account for the carcinogenicity or severity of the tumors produced. Also, it does not account for health effects other than oncogenicity. It is not peer reviewed, and is not available for all of the chemicals in the Total Diet Study.

The Weight-of-the-Evidence Rankings. There is a chronic risk index created by the U.S. Environmental Protection Agency (EPA) for possible carcinogens, called the weight-of-the-evidence rankings. It is a system of classifications EPA uses to evaluate all potential human carcinogens. The ranking is based on oncogenicity (tumor production) tests, both positive and negative; mutagenicity studies; varieties of tumors induced; the rankings of structurally similar chemicals; and the replication of positive results. There are six possible rankings:

A : Human carcinogen,
B1: Probable human carcinogen (some epidemiological or human data),
B2: Probable human carcinogen (no epidemiological data),
C : Possible human carcinogen,
D : Not classifiable as to carcinogenicity, and
E : Evidence of non-carcinogenicity for humans.

EPA uses a three step process to assign a ranking to each chemical. First, human and animal study evidence is evaluated separately. Then these two

sources of evidence are combined to form an initial overall ranking. Finally, supporting evidence is used to arrive at a final ranking (U.S. EPA 1992).

For our purpose of deriving an index of food safety risk, the weight-of-the-evidence suffers from two major weaknesses. First, it exists for only 32 of the 78 pesticides found in the Total Diet Study. Second, it covers only oncological risks. Other health risks—neurotoxicity, immunotoxicity, nononcogenic reproductive effects, and teratogenicity—are not factored in (National Academy of Sciences 1987). Furthermore, the ranking is based solely on the available evidence, and lack of evidence does not correlate necessarily with low risk. Therefore, a possible human carcinogen may actually pose a greater risk to human health than a probable human carcinogen, despite the lower weight-of-the-evidence ranking, simply because of a greater quantity of accumulated evidence (Harvard Center for Risk Analysis 1992).[7] Because of these limitations, we chose to include additional information to create a more comprehensive risk index.

A number of difficulties accompany any effort to create a risk index: inadequate understanding of pesticide risk; the lack of sufficient testing, particularly of chronic effects; and the problems associated with extrapolation of animal tests to humans (Hammitt 1986). Current toxicological testing generally uses "maximum tolerated doses" (MTD) to determine carcinogenicity, a much debated practice (National Academy of Sciences 1993b). A MTD is the largest quantity of a chemical that laboratory animals can ingest without causing serious health damage (other than cancer). These tests are used to detect carcinogenicity at relatively high levels of exposure. However, they provide limited information about the relationship between other exposure levels and adverse health effects. A linear relationship between dose levels is generally assumed, which may or may not be the case. A pesticide may have a high threshold of exposure that must be crossed in order for any adverse health effects to exist. On the other hand, a pesticide could be more hazardous at lower levels than a linear curve would suggest. This uncertainty is exacerbated by the process of inferring human health risk from the results of animal tests. Generally, scientists assume that human bodies will respond in much the same way as laboratory animals, adjusting for weight differences. However, humans may respond differently than animals (e.g., metabolize a chemical differently).[8] Furthermore, chemicals which are assigned the same risk classification (e.g., probable human carcinogen) may have different probabilities of cancer causation. Therefore, even among experts in the toxicological field, there is extensive debate about methodology and considerable uncertainty surrounding the interpretation of toxicological test results.

Though establishing the relative risks of pesticides is an extremely complex endeavor, it is very important when aggregating pesticide residues to avoid assigning equal weights to residues from substantially different chemicals. Existing toxicological data provide sufficient information to differentiate relative

risks between pesticides. We are not the first to employ toxicological data in this manner. The starting point for developing our index was the work of Hammitt (1986), who constructed a series of food safety risk measures based on acute and chronic effects of pesticides.

Hammitt (1986) used results from five types of toxicological tests—for carcinogenicity, neoplasticity, equivocal tumor agents, mutagenicity, and birth defects—to construct two alternative chronic risk indexes. The results were compiled in the *Registry of Toxic Effects of Chemical Substances* [RTECS]. The indexes considered only whether a test had been performed, not whether the test results were positive or negative. Therefore, Hammitt's indexes are reliable indicators of risk only if the toxicological tests have been performed and reported in RTECS. Three of the indexes are described below.

Chronic risk index: Hammitt's chronic risk index is developed from the results of five types of toxicological tests contained in RTECS: Carcinogenicity, neoplasticity (non-spreading carcinogenic tumors), equivocal tumor promotion (oncogenicity), mutagenicity, and teragenicity. The first four types of chronic effects are weighted according to their relative severity. If a pesticide is considered carcinogenic, it is given a rating of 1.0. Pesticides which are neoplastic are rated at 1/2. Substances that cause equivocal tumor production and mutagenic reaction are rated 1/4 and 1/8, respectively. Teragenicity is considered separately. A positive test for teragenicity contributes 1.0 to the chronic risk index rating. With the exception of a positive teragenicity contribution, the effects are not additive and the chronic risk rating is simply the value of the highest ranking test. This index is of limited value if similar toxicological tests have not been performed and reported for all pesticide residues. The index also does not account for the severity of test reactions.

Chronic sum index: For this index, Hammitt uses the same information considered in the chronic risk index, but sums the assigned weights of each toxic effect to derive a total risk weight for each chemical. In addition, carcinogenicity and mutagenicity are assigned equal weights. Again, missing tests reduce the reliability of the index, and potency is not considered.

Combined risk index: This index combines Hammitt's acute and chronic risk indices (Hammitt 1986). The indices can be weighted according to the discretion of the researcher, although a simple average of the two indices is commonly used.

A New Index. Because we wanted to create an index that accounted for a more comprehensive set of effects, we used three data bases to assemble toxicological information about the pesticides. The data bases were:

Integrated Risk Information System (IRIS): IRIS is a data base maintained by the EPA containing toxicological and regulatory information for approximately 400 chemicals. The data base includes carcinogenic and noncarcinogenic risk information, such as Q* ratings and weight-of-the-evidence rankings, when available. The IRIS data base includes conclusions about chemical toxicity drawn by EPA scientists.

Registry of Toxic Effects of Chemical Substances (RTECS): RTECS is managed by the National Institute for Occupational Safety and Health. This data base contains acute and chronic health risk data on over 90,000 chemicals. We relied on RTECS primarily for information on the carcinogenic, mutagenic, and reproductive effects of chemicals.

Chemical Carcinogenesis Research Information System (CCRIS): CCRIS is a data base created by the National Cancer Institute containing over 2,100 peer-reviewed toxicological records. CCRIS reports the results for carcinogenic, mutagenic, and tumor promotion and inhibition studies.

Our goal was to assign each pesticide to a risk category, either a higher, medium, or lower/unknown level of risk, based on data derived from these sources. We chose carcinogenicity as the first criteria to evaluate a pesticide's risk, based on regulatory precedent. Thus each pesticide in the *higher risk* category was a probable or declared carcinogen. Pesticides in the *medium risk* category were either suspected or possible human carcinogens, or they showed a greater number of positive mutagenic or developmental test results than other pesticides in the Total Diet Study. The *lower risk* category contains pesticides with negative or no carcinogenic test results or with no tests and limited evidence of mutagenic and reproductive effects. Unfortunately, those pesticides for which few toxicity studies have been conducted fall into this category. It is important also to emphasize that the rankings are relative risks, hence the terms higher, medium, or lower. Throughout the period of the Total Diet Study, with few exceptions, the quantities of pesticide residues detected have been well below the legal tolerance levels.[9]

Our first step was to use a step-wise procedure to rank each pesticide according to evidence of carcinogenicity. The criteria we considered, in order, were the following:

1. We began with the weight-of-the-evidence ranking, when available. If a pesticide was classified as a "probable human carcinogen" it automatically fell into the higher risk category. We placed "possible human carcinogens" initially in the medium risk category.
2. We considered other data on suspected carcinogenicity from EPA, such as information accompanying Special Review and cancellation reports.
3. Next we considered the results of carcinogenic studies from RTECS and CCRIS. We assigned greater weight to results from multiple sources.
4. Finally, we used the *Basic Guide to Pesticides*, published by the Rachel Carson Council (Briggs 1992), to verify the conclusions reached from other sources.

While carcinogenicity was the first criteria for rating pesticides, we believed, following Hammitt (1986), that a complete index must incorporate other chronic health effects. Cropper et al. (1990) discussed the difficulties in quantifying

noncarcinogenic effects and concluded that while quantification was infeasible, such effects should be noted and incorporated. Additional reports also support the inclusion of other chronic health effects (Harvard Center for Risk Analysis 1992). To evaluate noncarcinogenic health effects we examined the following sources:

1. First, EPA information from IRIS, press releases, special reviews, and cancellation reports.
2. Next we compiled the results of mutagenic and reproductive studies from RTECS and CCRIS, giving greater weight to positive results from multiple sources.
3. Again, we used the *Basic Guide to Pesticides* (Briggs 1992) as a source for confirmation of other studies.

We deliberately avoided counting the number of toxicity studies because, for a variety of reasons, all pesticides have not been studied equally. Some of the older pesticides that are no longer registered have less toxicological data available. Other pesticides have received more public attention and, consequently, more testing. However, we did assign greater risk to those pesticides with a greater number of diverse, positive tests. Thus, our category assignments (higher, medium, lower) are admittedly and necessarily subjective, based on the risk a pesticide presented, subject to our interpretation of available evidence and relative to the risk of other pesticides in the Total Diet Study. The criteria included the nature and severity of effects, replicability of results, and regulatory actions taken by EPA. Table 18.1 shows the categorization of the 78 chemicals detected in the total diet study by relative risk category.

While somewhat arbitrary, we feel this approach is defensible on two grounds. First, we believe that we could evaluate these data best using ordinal measures, because neither the data nor our level of expertise justified quantitative assessments of risk. Second, the valuation of relative risks is itself subjective. The use of carcinogenicity as a primary (and often the single) criteria for risk evaluation is both a function of the ease of data collection and the subjective value placed on cancer avoidance. Until both toxicological data and economic value assessments are more refined, assessment of relative risks involves subjectivity. For example, to value cancer avoided (either incidence or deaths) higher than the prevention of a birth defect may contradict the conclusion of an exclusively economic valuation process.

We applied integer weights directly to each risk classification, with the higher risk category receiving a weight of 4, the medium risk category a weight of 2, and the lower risk category a weight of 1. We arrived at these weights through our review of the evidence, which, in our opinion, indicated a smaller difference of risk between the lower and medium risk categories than between the higher and medium categories. The weighted residues were then summed

TABLE 18.1 Chemicals Detected in the Total Diet Study, by Relative Risk Category

Higher Risk	Medium Risk	Lower or Unknown Risk
2,4-D	Acephate	Aldoxycarb
2,4,5-T	Aldicarb	Carbophenothion
Aldrin	Azinphos-methyl	Chlorpyrifos
Atrazine	Carbaryl	Chlorpyrifos-methyl
BHC	Carbofuran	DCPA
Captan	Chlorpropham	DEF
Chlordane	Demeton	Diphenylamine
Chlorobenzilate	Demeton-S	Disulfoton
DDE	Deceton-S sulfone	Disulfoton sulfone
DDT	Diazinon	Ethion
Dicofol (Kelthane)	Dicloran (DCNA)	Fonofos
Dieldrin	Endosulfan	Iprodione
Dimethoate	EPN	Leptophos
Endrin	Fenitrothion	Methamidophos
Folpet	Fenvalerate	Methiocarb
Gamma BHC (Lindane)	Linuron	Mevinphos
Heptachlor	Malathion	Omethoate
Hexachlorobenzene	Methidathion	Oxamyl
Methoxychlor	Methomyl	Parathion-methyl
Trans-Nonachlor	Phorate	Phosalone
Nitrofen	Phosmet	Pirimiphos-methyl
Parathion	Phosphamidon	Sulfur
Pentachlorophenol (PCP)	Ronnel	Tecnazene
Permethrin	Thiabendazole	Vinclozolin
Perthane		
o-Phenylphenol		
Quintozene		
Propargite		
TDE		
Toxophene		

to create the risk index. The resulting index measures residues in "risk equivalents." This was an arbitrary weighting, based on our judgement. Other weighting schemes easily could be justified. However, we found that the results of the risk-weighted index were quite insensitive to the weights used.

Results

Figure 18.1 depicts both the risk-weighted and unweighted residue series. The risk-weighted series is equal to:

$$(1) \qquad R_t = \sum_{i=1}^{n} W_i P_{it}$$

where: R_t = risk-weighted index in year t,
W_i = category weight for pesticide i,
P_{it} = residue for pesticide i in year t.

Adjusting for the relative risks of pesticides, Figure 18.1 suggests that food safety risk was greater in the late 1960s than the unweighted residue data indicate. The higher residue levels of several highly toxic pesticides used in the late 1960s account for the greater risk shown by the weighted index. The influence of these residues on the index declined in later years as several higher

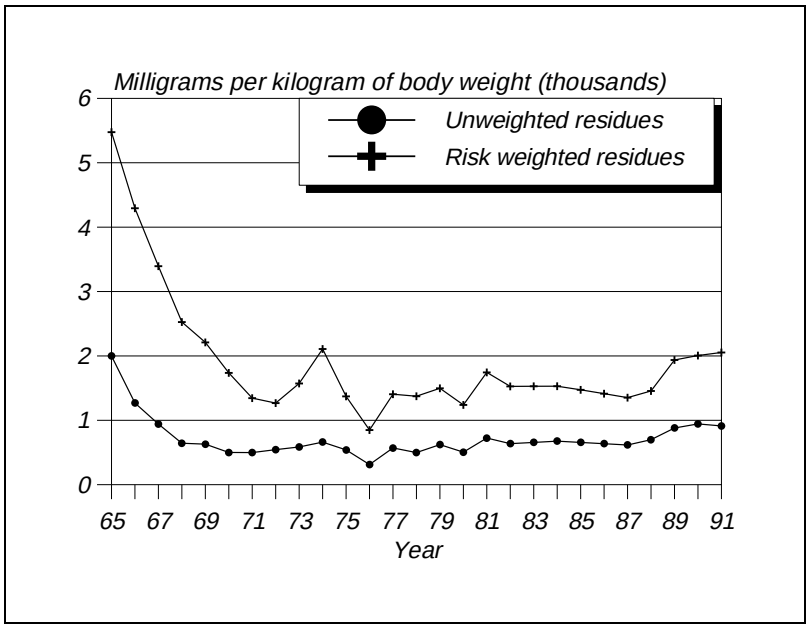

FIGURE 18.1 Pesticide Residues Found in a Market Basket of Food

risk pesticides were cancelled. Among these were DDT and its derivatives, 2,4,5,-T, dieldrin, and endrin. Measured as a risk-weighted index of total food pesticide residues, our study shows that food safety risk, after declining from 1965 to 1972, remained relatively stable and then increased slightly in the late 1980s.

Factors Related to Changes in Food Safety Risk

In an earlier paper (Day et al. 1993), we considered the effect of regulation on food safety, using the risk-weighted index of residues to measure changes in food safety risk from pesticide residues over time. Although the effects of regulation are not the focus of this chapter, it is useful to consider the determinants of the changes in food safety risk associated with pesticide residues. We briefly describe some of these determinants below.

Income

Changes in real per capita disposable income can reflect changes in tastes and preferences. If food safety is a superior good, the demand for food safety should increase with income. One might expect consumers to be more concerned about food safety risk as their incomes increase. Efforts of producers, handlers, and retailers to reduce residues may in part be a response to this increased concern. At the same time, changes in consumption patterns are correlated with income. For example, fresh fruit and vegetable consumption increases with income (Lutz et al. 1992). Larger quantities of chemicals are used in the production of these commodities (USDA 1991 and 1992a). Furthermore, because these commodities are readily perishable, additional pesticides are applied to reduce spoilage. Both factors lead to higher residues on these foods. Therefore, the net effect of income on food safety risk, both increasing demand for food safety and increasing consumption of fruits and vegetables, is uncertain.

Pesticide Use

Pesticide use also influences the quantity of resides found in food. We expect residues to increase with the intensity of pesticide use, not necessarily with total quantities applied. Pesticide use data derived from two sources reveal the trend in intensity of use. First, the total quantities of pesticides applied in agriculture, given in pounds of active ingredients, are reported by EPA and USDA (Aspelin et al. 1992, Osteen and Szmedra 1989). This time series of pesticide quantities goes back to 1964, and is derived from a combination of industry sources and agricultural surveys. The second component, the number

of acres of cropland, was obtained from Agricultural Statistics. By dividing the quantity of pesticides by the area of cropland we obtain a measure of the intensity of pesticide use. This series is depicted in Figure 18.2.

Throughout the late 1960s and 1970s, there was a steady increase in pesticide use per acre. The lower prices of pesticides relative to other inputs encouraged the substitution of pesticides for labor (National Academy of the Sciences 1989). By the end of this period, the percentage of all cropland treated with pesticides, particularly herbicides, reached 95 to 98 percent (Osteen and Szmedra 1989). Thereafter, beginning in 1980, total and per acre pesticide use stabilized. Because herbicides account for much of the increase and because often they are not applied directly to crops, we also show pesticide use excluding herbicides. This measure of use should be more closely related to changes in residues found on food. Without herbicides, total pesticide use per acre has changed little over the period examined. In terms of total pounds applied, insecticide use actually declined in the last decade (Osteen and Szmedra 1989), partly as a result of substituting more potent chemicals which are effective at lower rates of application.

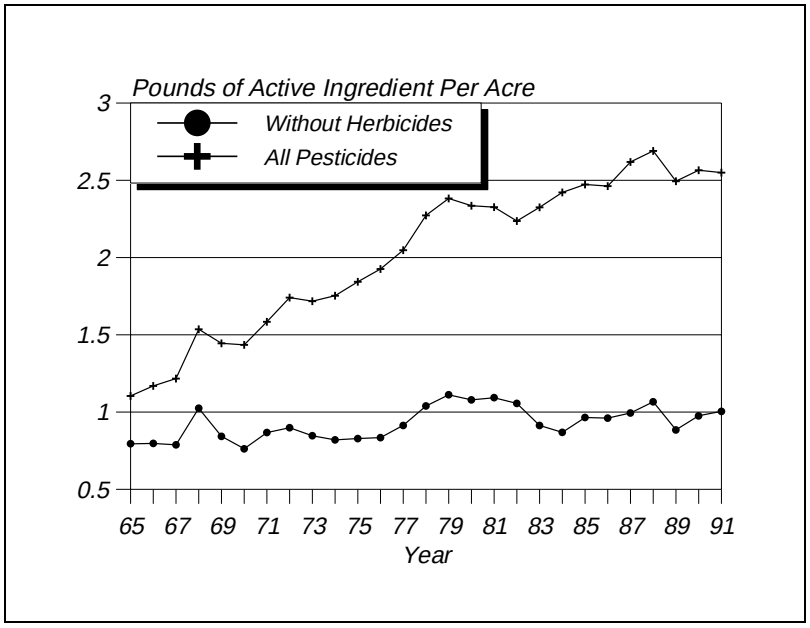

FIGURE 18.2 Pesticide Use Per Acre, 1965-1991

Regulation

Pesticide regulation did not originate in concerns for the safety of the products but because pesticide users were wary of manufacturers' unregulated claims of their effectiveness. The purpose of the first statute governing pesticides, the Insecticide Act of 1910, was to protect farmers and other pesticide users from fraudulent claims. Federal pesticide registration, first introduced in the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) of 1947, also was intended to ensure product efficacy. USDA was given responsibility for administering the registration program. Under FIFRA, tolerances for pesticides used on food crops were set by the Food and Drug Administration on the basis of toxicity information provided by the manufacturer. Manufacturers seeking to register a pesticide needed only to present data demonstrating that, when the product was used as directed on the label, residues left on food did not exceed the established tolerance level.

It was not until 1962 when Rachel Carson, formerly a marine biologist in the U.S. Fish and Wildlife Service, published her account of the safety and environmental risks of pesticide use in *Silent Spring* that protecting the environment and human health gained legitimacy as possible objectives of pesticide regulation. Still, these objectives were not included in federal statutes until ten years later.

In 1969, President Nixon asked the Council on Government Reorganization to design a new environmental regulatory program. The Council recommended consolidating research, standards setting, and pollution control enforcement within a single department. In 1970, EPA was created and the environmental regulatory functions were transferred from USDA, FDA, and the Department of Interior to the new agency.

The creation of EPA was a first step in shifting the focus from product efficacy to emphasizing human health in pesticide regulation. However, while the creation of EPA signalled this change in focus of regulation, it was not accompanied by changes in the law affecting pesticide registration, nor were restrictions imposed on pesticide use at that time.

The 1972 amendments to FIFRA were the first major changes in federal regulation of pesticides since 1947, and the first instance of federal restrictions on pesticide use. Several of the changes included in the amendments were related to food safety; in particular, the requirement that the costs and benefits of pesticide use, including adverse human health effects, be an explicit part of the registration process.

Residue Detection Technology

Residue testing technology has changed dramatically since the early 1960s. The sensitivity of detection methods has improved significantly. In 1965,

pesticide residues could be detected at .003 parts per million (ppm), while in 1982, they were detectable at .001 ppm. Technological advancements also expanded the set of detectable pesticides substantially. When the Total Diet Study began in 1961, less than 24 pesticides were detectable. In 1984, over 200 pesticides and related chemicals could be detected (Pennington and Gunderson 1987). Consequently, later pesticide residue measurements included a greater number of detectable pesticides.[10]

The number of pesticides detected each year has increased substantially since the beginning of the study (Figure 18.3). Unfortunately, it is unclear how much is due to improved residue detection and how much is from increases in the set of available pesticides. We would expect improved residue detection to bias the food safety measure downward early in the series, and upward later in the series.

Conclusion

Several points can be concluded from this study. First, the process of pesticide risk assessment is in its beginning stages for economists, and is preliminary to actual valuation work. No doubt a project such as this one would

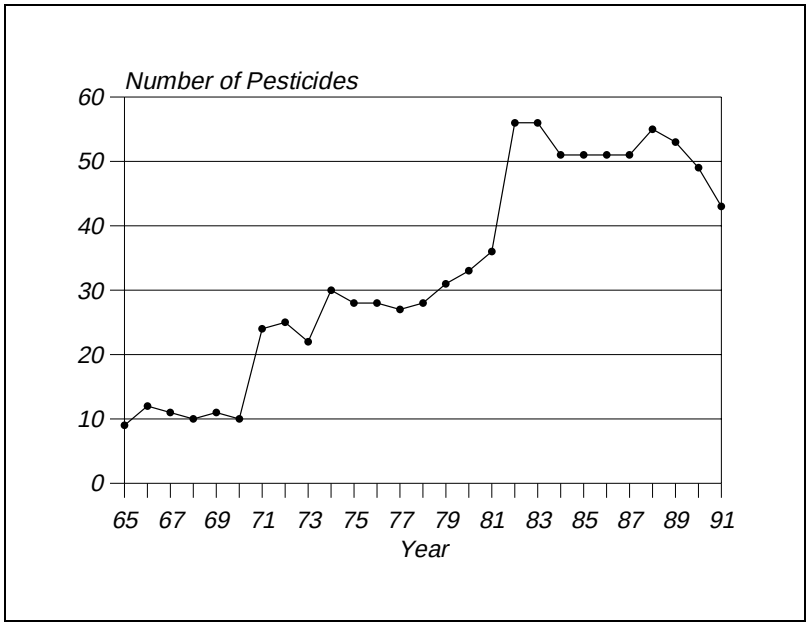

FIGURE 18.3 Number of Pesticides Detected in the Total Diet Study, 1965-1991

benefit from the input of toxicologists. While there is a need to communicate the potential risks associated with pesticide use, the information to assess these risks is limited. Second, if the object of a study is to determine human health risks posed by pesticides, it is important that the researcher assess more than carcinogenicity. If the risk index is limited to a Q* rating, or a weight-of-the-evidence-ranking, then only oncogenic or carcinogenic risks are being measured. Finally, in order for valuation to be meaningful, there must be an accurate determination of risk. Without proper assessment of toxicological data, we will not produce reasonable measures of the cost of current use or the benefits of pesticide use reduction.

Notes

1. The views in this paper are those of the authors and do not necessarily reflect those of the Department of Agriculture. This research has benefitted greatly from the helpful comments and technical expertise of Harry Vroomen, Fred Joutz, and Cynthia Tyler.

2. The Total Diet Study results have been published in some years by the FDA, in other years in the *Journal of the Association of Analytical Chemists*, or published jointly by both. Sources: Corneliussen 1972, Duggan et al. 1971, 1983, U.S. Food and Drug Administration 1988, 1989, 1990, 1991, 1992, Gartrell et al. 1985a, 1985b, Gunderson 1988, Johnson et al. 1984, Podrebarac 1984.

3. Although the Total Diet Study has been conducted annually, the results for some years during the early and mid-1980s were combined. Annual observations were interpolated using the results from these combined years.

4. The basis for the Total Diet Study is a series of food lists from consumer dietary surveys. These food lists have changed over time in accordance with changes in consumption patterns. Detailed below are the studies and number of foods analyzed:

1964-1970: The USDA's 1955 Household Food Consumption Survey was used to determine the typical diet consumed by 16-19 year old males in the U.S. The diet included 82 foods and assumed a large calorie intake (4,200 calories/day) to determine maximum exposure to various chemicals.

1971-1982: The diet and food lists were modified according to the USDA's 1965 Household Consumption Survey. A diet of 120 foods was developed for 15-20 year old males, and the caloric content was reduced to 3,900 calories/day. Four regional diets were constructed from the data, representing the East, Central, South, and Western U.S. In addition to these changes, FDA added an infant and toddler diet.

1982: The diet was revised according to the USDA 1977-1978 Nationwide Food Consumption Survey and the National Center for Health Statistics Second National Health and Nutrition Survey of 1976-1980. The Total Diet Study also returned to a national diet, including 234 foods. The caloric intake for 14-16 year-old males was reduced to 2,677 calories per day.

5. The age-sex group definition varies slightly through the history of the study. Until 1971, the group was 16-19 year-old males. From 1971-1982, the group was 15-20

year-old males. From 1982 onward, the age-sex group was defined as 14-16 year-old males.

6. Acceptable daily intake levels are based on a reference dose. Animal tests are conducted to establish No-Observed-Adverse-Effect-Levels (NOAEL), the maximum dose at which no adverse health effects are observed. NOAELs are adjusted for uncertainty and other factors (by multiplying by a factor of 10 to 100 or more) to arrive at a reference dose that is considered safe for human consumption.

7. An extensive discussion of the drawbacks of qualitative carcinogenic measures can be found in *Recommendation for Improving Cancer Risk Assessment* (Harvard Center for Risk Analysis 1992). Defenses for the qualitative measures can be found in *Regulating Pesticides in Food: The Delaney Paradox* (National Academy of the Sciences 1987) and in "Risk Assessment: Scientists Find Federal Funds are Misguided" (Environmental Policy Alert 1992).

8. See note 6.

9. Tolerances are the maximum legal level of a residue permitted on a particular food product. Tolerances are determined by evaluating the toxicity of a chemical assuming the pesticide is applied at the highest allowable rate and maximum lifetime exposure for the food consumer. Because the Total Diet Study measures actual residues on food prepared for consumption, and aggregates these residues based on the amount of each food consumed in a typical diet, we argue that the Total Diet Study residues are superior to tolerances as a measure of food safety risk.

10. The method of reporting pesticide residues has changed over the period studied. In earlier years, individual chemical metabolites were reported as separate residues. Later, metabolites were combined. We corrected for changes in the number of residues reported by aggregating the individual metabolites to correspond with the later combined lists. However, other unknown reporting procedures still may have biased our measure of the number of residues.

References

Ames, B. N., R. Magraw, and L. S. Gold. 1987. Ranking Possible Carcinogenic Hazards. *Science* 236:271-280.

Archibald, Sandra O. and Carl K. Winter. 1989. Pesticides in Food: Assessing the Risks. Reprint of Chapter 1 in *Chemicals in the Human Food Chain*, ed. C. K. Winter, J. N. Seiber, and C. F. Nuckton. Davis, CA: University of California Agricultural Issues Center.

Aspelin, Arnold L., Arthur Grube, and Robert Torla. 1992. *Pesticide Industry Sales and Usage: 1990 and 1991 Market Estimates*. Washington, D.C.: Environmental Protection Agency.

Briggs, Shirley A. and the Rachel Carson Council. 1992. *Basic Guide to Pesticides*. Washington D.C.: Hemisphere Publishing Corporation.

Carson, Rachel. 1962. *Silent Spring*. Boston, MA: Houghton Mifflin Company.

Corneliussen, P. E. 1972. Residues in Food and Feed: Pesticide Residues in Total Diet Samples (VI). *Pesticide Monitoring Journal* 5:313-341.

Cropper, Maureen L., Bill Evans, Steve Beradi, Maria Soares, and Paul Portney. 1990. *An Analysis of EPA Regulation of Food-Use Pesticides*. Discussion Paper CRM 90-04. Washington, D.C.: Resources for the Future.

Day, Kelly A., Betsey Kuhn, and Ann M. Vandeman. 1993. Pesticide Regulation and Food Safety Risk. Western Economic Association International Conference. Lake Tahoe, NV, June 21-25.

Duggan, R. E., G. Q. Lipscomb, E. L. Cox, R. E. Heatwole, and P. C. Kling. 1971. Residues in Food and Feed: Pesticide Residue Levels in Foods in the United States from July 1, 1963 to June 30, 1969. *Pesticide Monitoring Journal* 5:331-341.

Duggan, Reo E., Paul E. Corneliussen, Mary B. Duggan, Bernadette M. McMahon, and Robert J. Martin. 1983. *Residue Monitoring Data: Pesticide Residue Levels in Foods in the United States from July 1, 1969 to June 30, 1976*. Washington, D.C.: Food and Drug Administration and Association of Official Analytical Chemists.

Environmental Policy Alert. 1992. Risk Assessment: Scientists Find Federal Funds are Misguided. February 5:36.

Gartrell, M. J., J. C. Craun, D. S. Podrebarac, and E. Gunderson. 1985a. Pesticides, Selected Elements, and Other Chemicals in Adult Diet Samples, October 1978-September 1979. *Journal of the Association of Official Analytical Chemists* 60:146-161.

Gartrell, M. J., J. C. Craun, D. S. Podrebarac, and E. L. Gunderson. 1985b. Pesticides, Selected Elements, and Other Chemicals in Adult Diet Samples, October 1980-March 1982. *Journal of the Association of Official Analytical Chemists* 60:146-161.

Gunderson, Ellis L. 1988. Chemical Contaminants Monitoring: FDA Total Diet Study, April 1982-April 1984, Dietary Intake of Pesticides, Selected Elements, and Other Chemicals. *Journal of the Association of Official Analytical Chemists* 71:1200-1209.

Hammitt, James K. 1986. *Estimating Consumer Willingness to Pay for Food-Borne Risk*. R-3347-EPA, October. Santa Monica, CA: The Rand Corporation.

Harvard Center for Risk Analysis. 1992. *Annual Report, 1992*. Cambridge, MA: Harvard School of Public Health.

Horowitz, John K. and Richard T. Carson. 1990. Do Consumers Really Care About the Characteristics of Health Risks? Paper Presented at the American Agricultural Economics Association, Vancouver, British Columbia, August 4-8.

Johnson, Roger D., Dennis D. Manske, Dallas H. New, and David S. Podrebarac. 1984. Pesticide, Metal, and Other Chemical Residues in Adult Total Diet Samples (XIII), August 1976-September 1977. *Journal of the Association of Official Analytical Chemists* 67:154-166.

Lutz, Steven M., David M. Smallwood, James R. Blaylock, and Mary Y. Hama. 1992. *Changes in Food Consumption and Expenditures in American Households During the 1980's*. Statistical Bulletin No. 849. Washington, D.C.: Economic Research Service and Human Nutrition Information Service.

National Academy of Sciences, National Research Council. 1987. *Regulating Pesticides in Food: The Delaney Paradox*. Washington, D.C.: National Academy Press.

National Academy of Sciences, National Research Council. 1989. *Alternative Agriculture*. Washington D.C.: National Academy Press.

National Academy of Sciences, National Research Council. 1993a. *Pesticides in the Diets of Infants and Children*. Washington, D.C.: National Academy Press.

National Academy of Sciences, National Research Council. 1993b. News Release, January 28. Washington, D.C.: National Academy of Sciences.

Osteen, Craig O. and Philip I. Szmedra. 1989. *Agricultural Pesticide Use Trends and Policy Issues*. Agricultural Economic Report No. 622. Washington, D.C.: Economic Research Service, September.

Pennington, Jean A. and Ellis Gunderson. 1987. History of the Food and Drug Administration's Total Diet Study—1961 to 1987. *Journal of the Association of Official Analytical Chemists* 70:772-782.

Podrebarac, David S. 1984. Pesticide, Metal, and Other Chemical Residues in Adult Total Diet Samples (XIII), October 1977-September 1978. *Journal of the Association of Official Analytical Chemists* 67:176-185.

Roberts, Tanya and Eileen van Ravenswaay. 1989. *The Economics of Safeguarding the U.S. Food Supply*. Agriculture Information Bulletin No. 566. Washington, D.C.: Economic Research Service, July.

Sachs, Carolyn, Dorothy Blair, and Carolyn Richter. 1987. Consumer Pesticide Concerns: A 1965 and 1984 Comparison. *Journal of Consumer Affairs* 21:103.

U.S. Department of Agriculture, National Agricultural Statistics Service and Economic Research Service. 1991. *Agricultural Chemical Usage, 1990 Vegetables Summary*. Report No. AgCh1(91). Washington, D.C.: U.S. Department of Agriculture, June.

U.S. Department of Agriculture, National Agricultural Statistics Service and Economic Research Service. 1992a. *Agricultural Chemical Usage, 1991 Fruits and Nuts Summary*. Report No. AgCh1(92). Washington, D.C.: U.S. Department of Agriculture, June.

U.S. Department of Agriculture. 1992b. *Agricultural Statistics*, various years. Washington, D.C.: Government Printing Office.

U.S. Environmental Protection Agency. 1992. *Background Document 2: EPA Approach for Assessing the Risks Associated with Chronic Exposures to Carcinogens*. Washington, D.C.: EPA.

U. S. Food and Drug Administration. 1988. Food and Drug Administration Pesticide Program—Residues in Foods—1987. *Journal of the Association of Official Analytical Chemists* 71:156A-174A.

U. S. Food and Drug Administration. 1989. Food and Drug Administration Pesticide Program—Residues in Foods—1988. *Journal of the Association of Official Analytical Chemists* 72:133A-152A.

U. S. Food and Drug Administration. 1990. Food and Drug Administration Pesticide Program—Residues in Foods—1989. *Journal of the Association of Official Analytical Chemists* 73:127A-146A.

U. S. Food and Drug Administration. 1991. Food and Drug Administration Pesticide Program—Residues in Foods—1990. *Journal of the Association of Official Analytical Chemists* 74:121A-152A.

U. S. Food and Drug Administration. 1992. Food and Drug Administration Pesticide Program—Residues in Foods—1991. *Journal of the Association of Official Analytical Chemists* 75:20-25.