Risk Analysis, Vol. 20, No. 5, 2000

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Risk/Risk Trade-offs in Pesticide Regulation: An Exploratory Analysis of the Public Health Effects of a Ban on Organophosphate and Carbamate Pesticides

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Efforts to reduce pesticide-related risks to consumers and farmworkers often neglect the possibility that measures to reduce the target risk may introduce or enhance countervailing risks. These may arise from substitute pesticides or pest-control practices, from increased levels of pests or pest-related hazards, from increased levels of toxic natural pesticides in plants, from increased costs and decreased consumption of health-enhancing fruits and vegetables, or from direct income effects on consumers and farmers. The effect of the countervailing risks may partially or completely offset the reduction in the target risk. A risk-trade-off analysis was conducted of a potential ban on the use of organophosphate and carbamate (OP/Carbamate) insecticides in U.S. agriculture. Although this scenario is extreme, it has the analytic virtue of dispensing with the infinite number of "next-best" OP/Carbamates that might be substituted for specific combinations of crops and pests should only selected uses be banned. The analysis relies on detailed descriptions of the alternative pesticides and pest-control measures that would be used for each of 14 major crops. The effects of pest-control cost changes on prices and consumption and effects on consumer and producer incomes are projected using a general-equilibrium economic model. Several countervailing risks that may be significant were found, including acute toxicity to farmworkers from substitute pesticides, cancer and noncancer risks from substitute pesticides, and mortality induced by changes in disposable income. Other countervailing risks are more difficult to estimate or weigh. Potential increases in natural plant pesticides following an OP/Carbamate ban are discussed but data are lacking to quantify the effects. Changes in diet following the ban have both positive and negative effects, and the ultimate change is difficult to estimate. Although a net risk cannot be estimated, several approaches were illustrated that would be useful in risk-trade-off analyses. Key factors complicating comprehensive analysis of risk/risk trade-offs for pesticides were also identified, including data gaps and shortcomings of current risk assessment methods.

KEY WORDS: Risk trade-off analysis; pesticides; substitution risk

1. INTRODUCTION

Efforts to reduce pesticide-related risks to consumers and farmworkers may exacerbate other risks and thereby partially or completely offset reductions in the target risk (Gray & Graham, 1995). Countervailing risks may arise from substitute pesticides or pest-control practices, from increased levels of pests or pest-related hazards, from increased levels of toxic natural pesticides in food crops, from increased costs and decreased consumption of health-enhancing fruits and vegetables, or from financial effects on farmers and consumers. Sound decisions about pest-control op-

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0272-4332/00/1000-0665$16.00/1

tions and the production of food and fiber should weigh both target and countervailing risks. This article attempts to compare the effects of a specific pesticide regulation on target and countervailing risks. It identifies a number of data and methodological limitations that preclude a comprehensive and quantitative risk-trade-off analysis (RTA; Graham & Wiener, 1995).

Risk/risk trade-offs occurring as the result of a complete ban on organophosphate and carbamate (OP/Carbamate) insecticides are evaluated. OP/Carbamates are a large and widely used group of chemicals. There are more than 50 OP/Carbamates registered with the U.S. Environmental Protection Agency (EPA), and organophosphates account for almost half of all insecticide sales in the United States (U.S. Environmental Protection Agency, 2000). It is apparent that a ban of OP/Carbamates is an extreme scenario, but it has the analytic virtue of dispensing with the infinite number of "next-best" OP/Carbamates for specific combinations of crops and pests should only some number of uses be banned. In addition, this is a scenario that has been considered in the debate about implementation of the 1996 Food Quality Protection Act (FQPA; Neilsen, 1999). Indeed, restrictions on the use of two organophosphates were announced in late 1999 (U.S. Environmental Protection Agency, 1999).

Although a variety of countervailing risks were identified, a comprehensive and quantitative estimate of the risk trade-offs is not possible under the current study conditions. Significant factors that preclude a more complete analysis include limited data on the concentration of pesticides remaining on food when consumed (especially for those that might substitute for OP/Carbamates) and the concentrations and toxicities of naturally occurring pesticides in food crops, as well as the extent to which both of these might increase from higher pest stress in these crops or substitutions for more resistant cultivars. Potentially more important though are the limitations in conventional risk assessment methods for health effects other than cancer. Because these methods neither identify the specific health effect that may occur, nor provide probabilities of occurrence, they permit no sensible method for comparing changes in risk of cancer and noncancer health effects. Similarly, conventional methods prove inadequate for estimating the risks of changes in dietary intake of essential nutrients and other food constituents. Surprisingly, it was found that the countervailing risk most susceptible to quantification is the effect of increased production costs on family incomes and health—the "richer is safer" effect.

Potential risk trade-offs and populations of concern are identified in section 2. This qualitative evaluation does not allow comparison of the magnitude of the risks; the focus is on risks to public health, however, ecosystem and production risks are also identified. Some methods to quantify the identified risks using available data are demonstrated in section 3. Section 4 discusses how the target and countervailing risks can be compared, identifies additional countervailing risks not addressed in this study, and describes data and methods needed for a more complete RTA. Section 5 concludes with a discussion of the factors impeding comprehensive analysis of risk/risk trade-offs for pesticides, including data gaps and shortcomings of current risk assessment methods.

2. IDENTIFYING TRADE-OFFS

Most members of the OP/Carbamate families of pesticides share a common mode of insecticidal activity: interference with the enzyme acetylcholinesterase. The primary human health concerns—the target risks in the present analysis—are possible neurotoxic effects in exposed humans. This toxicity may manifest acutely (i.e., poisoning of highly exposed workers), or with long-term consumer exposure to residues on food. These insecticides have come under increasing regulatory scrutiny. Since tolerances—legal residue levels—are set with a focus on chronic exposure from food, it was assumed the primary target risk to be addressed is neurotoxicity from chronic consumer exposure to organophosphate or carbamate insecticides. Potential importance of the corollary target risk of acute toxicity to farmworkers is also illustrated. The countervailing risks of substitute pesticides that might be used in crop production, the potential risks of changes in natural plant pesticides, changes in diet and nutrient intake, and health risks due to changes in consumer and farmer income following a ban are also considered.

In an effort to encourage the explicit consideration of risk trade-offs and their consequences, Graham and Wiener (1995) developed a typology of trade-offs and a simple classification system—RTA. In this type of analysis, two questions are considered: First, does the countervailing risk, generated by the risk management activity, fall on the same population as the target risk or on another group? Second, is the countervailing risk of the same type as the target risk (e.g., one cancer risk replacing another cancer risk) or of a different type (e.g., a noncancer risk replacing a cancer risk)? The answers to these two questions

Table I. Risk Trade-off Analysis Classification System

	Compared to the target risk, the countervailing risk is	
	Same type	Different type
Compared to the target risk, the countervailing risk affects		
Same population	Risk offset	Risk substitution
Different population	Risk transfer	Risk transformation

can be used to classify a trade-off either as a "risk offset," a "risk transfer," a "risk substitution" or a "risk transformation," as shown in Table I.

Table II lists some of the qualitative public health risk trade-offs identified for an OP/Carbamate ban. Other risk trade-offs, including those to farmworkers and the environment, are discussed later. The countervailing risks identified in Table II arise from substitute pesticides, changes in levels of natural plant toxins, decreases in income, and changes in diet following an OP/Carbamate ban (see Table II).

In all cases, the primary target risk is assumed to be potential neurotoxicity to consumers from the residues of OP/Carbamates on food. A secondary potential benefit of an OP/Carbamate ban is a reduction in exposure of farmworkers to these compounds. Risk substitutions are the most common trade-off, many arising from the use of alternative pesticides, although all classes of trade-offs are identified. Following is a discussion on methods that might be helpful in quantifying the target and countervailing risks identified in Table II.

Table II. Risk Trade-off Analysis for the OP/Carbamate-Ban Scenario

Risk trade-off type	Target risk	Countervailing risk	Example(s)
Substitution	Neurotoxicity from chronic consumer exposure to organophosphate or carbamate insecticides	Cancer risk from substitute insecticides	Lindane replacing Fonofos for wireworm control on wheat (Peel & Aarke, 1999)
Substitution	Neurotoxicity from chronic consumer exposure to organophosphate or carbamate insecticides	Other (than neurotoxicity) non-cancer effects from substitute pesticides	Imidaclorpid (thyroid toxicity) replacing dimethoate for sharpshooter control on grapes (Peacock, 1999)
Offset	Neurotoxicity from chronic consumer exposure to organophosphate or carbamate insecticides	Neurotoxicity from substitute pesticides	Tefluthrin substituting for ethyl parathion or parathion methyl for control of stinkbugs on soybeans (Wiese, Helmers, & Shaik, 1999)
Substitution	Neurotoxicity from chronic consumer exposure to organophosphate or carbamate insecticides	Decreased nutrient intake and subsequent health risks due to consumer changes in consumption (also has corollary benefits with some food components)	Predicted decreased intake of folate and vitamins C and E, benefit from predicted reduction in cholesterol and fat intake (Taylor & Smith, 1999)
Substitution, offset	Neurotoxicity from chronic consumer exposure to organophosphate or carbamate insecticides	Cancer and other toxic effects due to consumer exposure to higher levels of natural pesticide in foods (due to pest pressure or breeding of resistant cultivars)	Caffeic acid (carcinogen) in apples, carrots, grapes, potatoes; solanine and chaconine (cholinesterase inhibitors, teratogens) in potatoes (Ames *et al.*, 1990)
Substitution	Neurotoxicity from chronic consumer exposure to organophosphate or carbamate insecticides	Adverse health effects on consumers due to decreased income	A ban on OP/Carbamates is estimated to cause an aggregate decrease in personal income of over $5 billion; an increase in household spending on food of approximately $840 million (Taylor & Smith, 1999)
Transformation	Neurotoxicity from chronic consumer exposure to organophosphate or carbamate insecticides	Adverse health effects on farm families due to decreased farm income	A ban on OP/Carbamates is estimated to cause an aggregate $1.6 billion decrease in net income for fruit and vegetable farmers (Taylor & Smith, 1999)
Substitution, transformation	Corollary target risk of acute toxicity to farmworkers from OP/Carbamates	Acute toxicity to farmworkers from substitute pesticides; toxicity to consumers from substitutes	

Note: OP/Carbamate = organophosphate and carbamate.

3. QUANTIFYING RISK TRADE-OFFS

Quantitative risk assessment requires estimates of exposure to OP/Carbamates, substitute pesticides, and other risk agents. To estimate exposure to OP/Carbamates and substitute pesticides, the present analysis relies on studies that estimate changes in pesticide use, yield, and cost for 14 crops in a 4-year period following a complete ban on OP/Carbamate pesticides (Knutson & Smith, 1999). These data were developed through analysis, models, and the expert judgment of farmers, extension agents, and academic researchers around the United States. They represent the crop experts' considered opinions as to the most effective and economical currently available pest-control practices that would be adopted if OP/Carbamates were banned, including the use of substitute pesticides, alternative tilling, biological agents, and other scenarios. The studies have not, to this point, been peer reviewed but follow accepted methods and are fully documented. Data from Knutson and Smith (1999) were also used to propagate production changes through the U.S. economy using AGSIM, a regional econometric simulation model of the agricultural economy (Taylor, 1993) and IMPLAN, a national input/output model of the U.S. economy (Minnesota IMPLAN Group, Inc., 1997). These models predict changes in prices, production levels, demand, and incomes following an OP/Carbamate ban (Taylor & Smith, 1999).

3.1. Substitute Pesticides—Acute Toxicity to Farmworkers

To illustrate a potential approach for addressing farmworker risks, data on the acute effects of pesticides were taken from the California Pesticide Illness Surveillance Program (PISP, California Environmental Protection Agency, 1997). PISP relies on physician reports of any case known or suspected to be due to pesticide exposure, and on California Department of Pesticide Regulation review of worker-compensation claims. This is the most comprehensive database of pesticide-related illness in the country. Data from 1995 were used because they reported illnesses as "definitely," "probably," or "possibly" associated with organophosphates, carbamates, and other insecticides. Data on pesticide use in California for 1995 are from the California Pesticide Reporting System (California Environmental Protection Agency, 1995). A complete analysis would benefit from similar data for different locations and over time.

The California PISP data are useful as a guide to the potential effects of pesticides on farmworkers and others, although there are concerns about attribution of disease and reporting (California Environmental Protection Agency, 1997). Table III summarizes data on acute illnesses/injuries reported to PISP in 1995. Illnesses/injuries are categorized as "systemic," "eye," "skin," and "eye and skin." The table reports totals across the categories. There were no reported fatalities associated with insecticides in California in 1995. The California Department of Pesticide Regulation reports that only 41% of these cases were related to agricultural uses of the pesticides (California Environmental Protection Agency, 1997).

Making a few assumptions, the target risk of acute illness or injury associated with OP/Carbamate applications and the countervailing risk of other insecticides that might be used as substitutes can be estimated. It was first assumed that the 41% share of cases related to agricultural use applies uniformly across classes of pesticides. Another assumption was that "number of applications" is the appropriate measure of toxicity opportunities, rather than, for example, number of pounds applied. Considering only the top 100 most frequently applied pesticides, there were 430,685 applications of OP/Carbamates and 425,529 applications of other named insecticides (excluding oils and soaps) in California in 1994 (California Environmental Protection Agency, 1995).

From these numbers it can be estimated that each application of an OP/Carbamate for agricultural purposes is associated with 0.0011 illness or injury, and each non-OP/Carbamate insecticide is associated with 0.0009 illness or injury. The ratio of illness or injuries per application for non-OP/Carbamates to OP/Carbamates is 0.8. This ratio suggests that a change from OP/Carbamates to other insecticides may lead to a small reduction in acute toxicity to farmworkers and others. If the number of necessary applications increases—either because of lower efficacy or the need for several materials to replace a single OP/

Table III. 1995 California Pesticide Illness Reporting Program Illnesses/Injuries by Insecticide Type

Category	Definite/ probable illness	Possible illness	Total
Organophosphates	103	65	168
Carbamates	20	10	30
Other insecticides	124	30	154

Carbamate—the acute toxicity risk could also increase. Similarly, a decrease in the number of overall applications would make the trade-off more favorable. It appears that any gain from an OP/Carbamate ban would be at least partially offset by the acute toxicity of the pesticides that would be used as alternatives. Other methods of estimating changes in acute toxicity, such as measuring pounds of active ingredient applied, might also be useful in RTA.

3.2. Substitute Pesticides—Chronic Noncancer Toxicity to Consumers

Knutson and Smith (1999) have described the likely changes in farm pest-control practices that would follow a ban on OP/Carbamates, including pesticides that would be substituted. For illustrative purposes, the cancer and noncancer risks from OP/Carbamates and substitutes used on potatoes and tomatoes were compared. These crops were chosen because judgments about current and substitute pesticide use and monitoring data on pesticide residue levels are available. Nine OP/Carbamates can be used on potatoes; five can be used on tomatoes.

The chronic public health risks of OP/Carbamates and substitute pesticides are compared using standard EPA procedures and risk values. Estimates of exposure are compared to a Reference Dose (RfD) or Acceptable Daily Intake (ADI) for noncancer effects by constructing a hazard quotient—exposure/RfD.

Determining exposure estimates for consumers requires data on both residue levels on food and the amount of food consumed. Residue levels are estimated in two ways. First, exposure is assumed to be at the tolerance level (the Tolerance Approach), the highest amount of residue allowed on food by law (Insecticide, Fungicide, and Rodenticide Act, 1948, as amended). It is recognized that actual exposures "on the dinner plate" are often much lower than the tolerance level (Eilrich, 1991). Therefore, data from the U.S. Department of Agriculture's (USDA's) Pesticide Data Program (PDP, U.S. Department of Agriculture 1995, 1996b, 1997) are also used to estimate exposure (the PDP Approach). The PDP collects statistical samples of commodities at distribution or retail establishments and measures the level of specific synthetic pesticides. Reported summary data include the percentage of samples for which residues were detected, the range of levels detected, and the range of levels of detection for the analysis method.

Two methods were used to account for nondetects in the PDP data. First, it is assumed that nondetects are true zeros reflecting different use patterns across farms and regions of the country (the Use Model). In this case, estimated residue concentrations for pesticide/crop combinations with PDP detections are the midpoint of the range detected multiplied by the percentage of samples with detected residues. Concentrations for pesticide/crop combinations tested by the PDP but with no detected residues are treated as zeros. Concentrations for pesticide/crop combinations not tested by the PDP are estimated by multiplying the average of the detection-midpoint/tolerance ratio for pesticides detected on that crop by the tolerance and by the average percentage of samples of that crop with detected residues.

The second approach (the Detect Model) assumes that PDP nondetects reflect the inability to measure low levels of residues. In this case, concentrations for pesticide/crop combinations with PDP detections are the midpoint of the range detected multiplied by the percentage of samples with residues detected, plus one-half the midpoint of the limit of detection for that pesticide times the percentage of nondetects. Concentrations for pesticide/crop combinations tested by PDP but with no detected residues are estimated as one-half of the midpoint of the reported levels of detection for the pesticide. Concentrations for pesticide/crop combinations not tested by PDP are estimated by multiplying the average half-limit of the detection/tolerance ratio for pesticides detected on that crop by the tolerance.

To estimate human exposure, residue levels are combined with average daily consumption. Consumption estimates were taken from the USDA Continuing Survey of Food Intake by Individuals (CSFII, USDA, 1996c).

Table IV shows the cumulative hazard quotient for OP/Carbamates and substitute pesticides for tomatoes (Gilreath, Smith, & Taylor, 1999) and potatoes (Wiese & Guenthner, 1999). The "summed hazard quotient" is the sum of the hazard quotients (exposure/RfD) for the individual pesticides. Several key assumptions are embedded in these estimates: For the estimates based on tolerances, it was assumed that all OP/Carbamates and substitutes identified for a crop are used since there are no data on the substitution pattern that might occur in the event of a ban. Data based on PDP measurements account for use patterns since numbers and levels of detections are influenced by farm practices. For substitute pesticides, it was assumed that frequency of detection and levels of detection will not change as they replace OP/Carbamates. It was also assumed that pesticides not

Table IV. Hazard Indices for Average Consumption of Produce Treated With OP/Carbamates or Substitutes

Crop/model	OP/Carbamate summed hazard quotient	Substitute summed hazard quotient
Potatoes		
Tolerance	6.453	0.041
Use Model	0.011	0.002
Detect Model	0.450	0.003
Tomatoes		
Tolerance	11.448	0.0250
Use Model	0.010	0.000006
Detect Model	0.047	0.001

Note: OP/Carbamate = organophosphate and carbamate.

Table V. Summed Upper-Bound Cancer Risks for Average Consumption of Potatoes and Tomatoes Treated With OP/Carbamates or Substitutes

Crop/model	OP/Carbamate summed cancer risk	Substitute summed cancer risk
Potatoes		
Tolerance	5.23×10^{-5}	1.47×10^{-6}
Use Model	2.74×10^{-8}	2.39×10^{-8}
Detect Model	6.27×10^{-6}	1.20×10^{-7}
Tomatoes		
Tolerance	0	0
Use model	0	0
Detect model	0	0

Note: OP/Carbamate = organophosphate and carbamate.

tested in the PDP will have similar frequencies of detection and ratios of detected levels to tolerance as those that were tested.

An important consideration in RTA is the relative severity of toxic effects. The RfDs (or ADIs) and tolerances for OPs/Carbamates are set primarily on the basis of inhibition of cholinesterase (ChE) in the blood or brain. This is a particularly subtle endpoint that many scientists believe is a marker of exposure rather than toxicity (e.g., Chen, Sheets, Nolan, & Mattsson, 1999). In some cases, other endpoints are the basis for regulatory assessment. Most other pesticides are evaluated on the basis of more traditional, and severe, toxic effects leading to both higher RfDs and more direct links to morbidity. There are also important questions about prediction of actual toxic effects across species, and those that may change from high to low doses.

3.3. Substitute Pesticides—Cancer Risk to Consumers

For a quantitative characterization of cancer risk, exposure estimates are combined with carcinogenic-potency values calculated by the EPA (U.S. Environmental Protection Agency, 1997). For all the pesticides under consideration, concerns about carcinogenic potential are based on results of high-dose animal tests. No direct evidence of carcinogenicity in humans has been found and so the usual concerns about interspecies and low-dose extrapolation arise.

Table V shows the summed cancer risks for OP/Carbamates and substitute pesticides for potatoes and tomatoes. The "summed cancer risk" is the total of individual cancer risks for each pesticide with potential carcinogenicity in each category. The EPA classifies none of the OP/Carbamates or substitutes used on tomatoes as a potential carcinogen. Therefore the risks with and without OP/Carbamates, as conventionally estimated, are zero. It should be noted that the risks may be higher if evaluations of human cancer potential based on animal studies are incorrect. Again, several assumptions are made in interpreting these estimates. The most important is that these compounds are indeed carcinogenic to humans at the levels found on food. Second, the assumption of a linear, no-threshold dose–response relationship, embedded in EPA cancer potency values (Anderson & the Carcinogen Assessment Group of the U.S. Environmental Protection Agency, 1983; U.S. Environmental Protection Agency, 1986) has a strong influence on estimates of risk. Third, these estimates are considered by the EPA to be "plausible upper bounds" on risk and the true risk is expected to be lower, and may be zero (U.S. Environmental Protection Agency, 1986). The same assumptions about exposure (use patterns and PDP data) apply to cancer estimates as to hazard quotients.

3.4. Risk from Natural Toxins in Food

There are insufficient data to quantitatively evaluate the risk trade-offs posed by changes in the levels of natural toxins in plants, although there are reasons to think they could be as large or even larger than pesticide risks (National Research Council, 1996). Plants produce a number of chemical agents that act as natural pesticides to protect the plant against various predators. Many of these natural compounds have been found to be carcinogenic or toxic in other

ways. Some even have natural anti-Che activity; for example, solanine and chaconine are Che inhibitors found in potatoes (Ames, Profet, & Gold, 1990). There could be countervailing risks from plant toxins if farmers react to an OP/Carbamate ban by increasing the use of resistant cultivars, or if pest predation, which stimulates production of natural toxins, increases.

3.5. Risks from Changes in Diet

The OP/Carbamate insecticides are inexpensive relative to many other pest-control options. In many cases, a ban on these compounds would require the substitution of more expensive pest-control methods which would lead to an increase in prices. As price increases, consumers will substitute other foods. Changes in the diet may cause both countervailing risks and corollary benefits. Countervailing risks arise if the substitute foods are less nutritious than the fruits, vegetables, and grains purchased when OP/Carbamates are in use. A number of vitamins and minerals found in these food crops are known to be protective against cancer and other diseases (National Research Council, 1996). Decreases in intake may increase these risks. Corollary benefits of the OP/Carbamate ban come from decreased consumption of food components that are linked to increased risk of disease (e.g., saturated fat) or increased nutrient intake from substitute, for example, organically grown, foods. A series of quantitative models (Taylor & Smith, 1999) are used to estimate changes in food prices, consumer purchasing choices, and nutrient intakes if OP/Carbamates are eliminated.

A simple way to estimate the effects of changes in food consumption in terms of public health is to compare the number of persons ingesting the recommended amount of certain components. For nutrients, this could mean comparing persons achieving the recommended daily intake of a nutrient now and after an OP/Carbamate ban. To address the potential countervailing risks from changes in diet following an OP/Carbamate ban, the change in the number of persons consuming at least the Recommended Daily Intake (RDI) for a wide range of nutrients and other dietary components is evaluated. Estimates of changes in daily consumption of each nutrient following an OP/Carbamate ban are from Taylor and Smith (1999). Data on U.S. population intake of nutrients (mean, median standard deviation of the mean, and sample size) are from the National Center for Health Statistics (Alaimo *et al.*, 1994; McDowell *et al.*, 1994). RDIs are from the Food and Drug Administration (FDA), as published in the *Federal Register* (U.S. Department of Health and Human Services, 1995) or the American Heart Association (AHA) Website (American Heart Association, 1999). Population intake is modeled as a normal distribution. The average (mean-median)/standard deviation value was 0.104 with a standard deviation of 0.023, which suggests that the nutrient intake distributions are reasonably symmetric and the assumption of normality is reasonable. Changes in consumption were assumed to change the mean but not the standard deviation of the intake. U.S. population was assumed to be 271,000,000 as of November 1, 1998 (U.S. Bureau of the Census, 1998).

Table VI shows estimates of the number of Americans that would change intake status, for better or worse, based on the projected changes in food-component consumption (from Taylor & Smith, 1999). The correlation across nutrients cannot be accounted for because of the absence of data on long-term dietary patterns.

3.6. Mortality Induced by Decreases in Income

A significant body of research supports the finding that "richer is safer" (Chapman & Hariharan, 1994, 1996; Keeney, 1990, 1997; Wildavsky, 1979, 1980, 1988). Common sense supports this notion—if health and safety are normal goods, wealthier people will tend to "purchase" more of them. Although wealthier people may also spend some of their money in ways that put them at increased mortality risk (e.g., more airplane travel), on average they are expected to purchase, and to maintain in better condition, safer homes, automobiles, and other products (Wildavsky, 1979, 1980, 1988).

Empirical research finds a correlation between greater income and smaller age-adjusted mortality risk (Lutter & Morrall, 1994). Moreover, this research provides estimates of the rate at which mortality risk increases with decreasing income. Most of the recent evidence comes from cross-sectional studies of people within the United States (Chapman & Hariharan, 1994; Keeney, 1990) supplemented by longitudinal (time-series) studies (Chapman & Hariharan, 1996; Keeney, 1997), which compare changes in mortality rates and changes in income. A limitation of these studies is that it is difficult to control for "reverse causality": the possibility that the observed correlation between lower income and impaired health reflects the effect of poor health in limiting earning potential (Smith, 1999). Chapman & Hariharan (1994)

Table VI. Population Effects of Nutrient Changes Induced by OP/Carbamate Ban

Nutrient	Change in daily consumption (percent)	RDI or AHA recommended (per day)	Number of persons changing to worse health state	Number of persons changing to better health state
Calcium	−0.264	1,000 mg	174,000	
Cholesterol	−0.202	300 mg		115,000
Copper	−0.447	2 mg	399,000	
Dietary Fiber	−0.348	30 g	199,000	
Folate	−0.512	400 μg	288,000	
Iron	−0.226	18 mg	148,000	
Magnesium	−0.205	400 mg	169,000	
Niacin	−0.132	20 mg	132,000	
Phosphorus	−0.201	1,000 mg	212,000	
Riboflavin	−0.126	1.7 mg	105,000	
Saturated fat	−0.414	22 g		243,000
Total fat	−0.390	70 g		298,000
Sodium	−0.651	2,400 mg		540,000
Thiamin	−0.298	1.5 mg	207,000	
Vitamin A	0.059	5,000 IU		24,000
Vitamin B_{12}	0.019	12 μg		7,000
Vitamin B_6	−0.152	2 mg	113,000	
Vitamin C	−1.410	60 mg	520,000	
Vitamin E	−0.522	10 (mg α-TE)	184,000	
Zinc	−0.142	15 mg	96,000	

Note: OP/Carbamate = organophosphate and carbamate; RDI = Recommended Daily Intake; AHA = American Heart Association; IU = international unit; α-TE = alpha-tocopherol.

find that failure to control for individual health overstates the effect of income on mortality risk by about a factor of two. The longitudinal studies, in which time-varying individual mortality risk is modeled as a function of time-varying individual income, provide stronger evidence of a causal relationship.

Results from cross-sectional and longitudinal analyses suggest that the effect of income on mortality risk is smaller at high income levels than at low levels, so the increased mortality risk to a population depends on how income losses are distributed. If wealthy people bear a larger share of the costs, the effect on mortality will be smaller (Chapman & Hariharan, 1996; Keeney, 1990, 1997). Overall, the loss of income in a population that would be expected to induce one premature fatality (the "cost per induced fatality" or CIF) is estimated to be about $5–16 million, with values at the lower range being more appropriate when the loss is concentrated on lower income groups, and values at the higher end being more appropriate when the loss is proportional to income or concentrated on higher income groups (Chapman & Hariharan, 1996; Keeney, 1990, 1997).

An alternative, indirect approach (Viscusi, 1994a, 1994b) uses economic theory to show that CIF is related to the rate at which individuals are willing to trade money for reduced mortality risk, conventionally measured as a "value per statistical life" or VSL. CIF is equal to the VSL divided by the marginal propensity to spend income in ways that reduce mortality risk. For example, assume an individual's marginal propensity to spend on health is 0.2, which means he or she spends an average of 20 cents out of every additional dollar earned in ways that reduce mortality risk (e.g., better diet, improved health care). Using a conventional estimate of the VSL equal to $5 million (Viscusi, 1993), the individual is willing to spend $100 per year in order to reduce his or her annual mortality risk by 2 in 100,000. If income is reduced by $500, the individual will spend $100 less on reducing mortality risk, yielding an increase in mortality risk of 2 in 100,000. Thus, CIF = $500/(2 × 10^{-5}) = $25 million = VSL/0.2. Viscusi (1994a) reviews a body of evidence that suggests the marginal propensity to spend on health is about 0.1–0.3. From this he estimates CIF as between about $30 million and $70 million. Premature fatalities induced by an OP/Carbamate ban were calculated using both direct and indirect estimates of CIF.

Applying estimates of CIF to the effects of a hypothetical ban on use of OP/Carbamate pesticides re-

quires estimates of the resulting loss in disposable income. Taylor and Smith (1999) provide two estimates useful for determining this loss. First, they estimate that annual spending on foods would increase by $8.40 per household. Multiplying this amount by the approximately 100 million U.S. households yields an aggregate increase in food spending of $840 million. Even though this increased spending does not reduce income, it reduces the share of income available for spending on other goods and services (disposable income), and so is anticipated to increase the number of premature fatalities. Accounting for general-equilibrium effects of increased food prices (i.e., changes in the prices of other goods and services due to changes in input costs and demand substitution) yields an aggregate reduction in personal income of $5.5 billion. (This assumes no change in government subsidies.) A substantial share of this loss is due to increased unemployment associated with the loss of 209,000 jobs throughout the economy, which suggests that the total income loss represents large losses to a small share of the population (the newly unemployed) and small losses to most of the population. These job losses are likely to be transient, as displaced workers move to other economic sectors. Even if they are temporary, large changes in income may have significant adverse health effects.

Estimates of the premature fatalities induced per year can be obtained by dividing the estimated income losses by alternative estimates of CIF. The results are summarized in Table VII. The estimated annual number of fatalities induced by the increase in food cost ranges from roughly 10 to 1,000. Considering only the reduction in disposable income directly attributable to increased food costs yields estimated effects of roughly 10 to 170 premature fatalities, depending on the rate at which income loss increases mortality risk (CIF). Considering a more inclusive measure of lost income—the estimated reduction in personal income after accounting for the effects of increased food costs on other economic sectors and employment—yields estimates of about 100 to 1,000 premature fatalities per year.

As noted, estimates of CIF suggest it is smaller for lower income groups, which implies that the aggregate number of induced fatalities depends on how the loss in disposable income is allocated across income groups. Taylor and Smith (1999) provide information showing that the change in food spending increases with income, but less than proportionately. Combining these estimates of increased spending with estimates of CIF by income group (Chapman & Hariharan, 1996) yields results similar to those obtained using the average (all-income) results. Although the estimated effect of reduced disposable income on health is larger for low-income households, higher income households lose more disposable income. If the loss in aggregate personal income aggregates very large losses to a small fraction of the population who become unemployed, accounting for the distributional effects of lost income on mortality risk could be important.

Table VII. Estimates of Annual Premature Fatalities From Loss of Disposable Income

	Estimated loss of disposable income	
Cost per induced fatality	Increased food spending ($840 million)	Aggregate loss in personal income ($5.5 billion)
Direct estimates ($5–16 million)	52–170	340–1,100
Indirect estimates ($30–70 million)	12–28	79–180

4. DISCUSSION—WEIGHING THE RISKS

The quantitative analysis discussed in the previous section provides some indication of the effect of an OP/Carbamate ban on a variety of health risks. The quality of the estimates is hampered, however, by data and methodological limitations, and a method for comparing changes in risks across endpoints is needed. In this section, some of the limitations of the quantitative analysis are highlighted and suggested methods for improvement are given.

4.1. Acute Toxicity

The OP/Carbamates have, as a class, moderate to high acute toxicity. One might expect that this property would make them of greater risk to farmworkers than alternative pesticides. The present evaluation of rates of illness and injury from OP/Carbamates and other insecticides in the California PISP data suggests this may not be so. There are several potential explanations that must be considered. The PISP data include agricultural and nonagricultural uses of pesticides. If the two classes of compounds are used at different rates in these two applications and injury/illness rates are different, simplifying the as-

sumptions, as was done in the present study, may mask a true difference. Another weakness is the inability to account for possible differences in application methods. Aerial application or other dispersive methods could increase the potential for exposure to OP/Carbamates. The California Environmental Protection Agency worries that some pesticide-induced injuries/illnesses are not reported to the PISP program. If reporting, or mention in worker-compensation reports, were differential between OP/Carbamates and other insecticides, it could present a false picture of the rates for each group. Given the high profile and known toxicity of the OP/Carbamates, it is unlikely that their rates are differentially underreported and it is perhaps more likely that health effects due to other pesticides are not fully ascertained. Of course, this would reduce or reverse the apparent differences in the rates of illness between OP/Carbamates and their alternatives. Finally, it may be that farmworkers are aware of the relatively high toxicity of OP/Carbamates and use them more carefully than the substitute pesticides. In this case, one might expect injury risk to be inversely related to toxicity, and for injuries to rise following a ban.

From this evaluation of readily available data, it is apparent that in some situations the countervailing risks of substitute pesticides can significantly offset—and might even outweigh—the benefit from addressing the corollary target risk of acute toxicity. A more thorough assessment of acute toxicity target and countervailing risks would need to include a longer time period and larger geographic area, and would need to ensure all relevant pesticides were evaluated.

4.2. Pesticide Noncancer Effects

The comparison of noncancer risks posed by OP/Carbamates to those of their potential substitutes is complicated by difficulties in interpreting both exposure and endpoint data. Alternative methods for estimating exposure yield quite different conclusions about the relative riskiness of OP/Carbamates and their alternatives (Table IV). For potatoes, the ratio of OP/Carbamate to substitute summed hazard quotients ranges from about 160 at the tolerance level of exposure to about 6 for the Use Model, which assumes PDP nondetects are true 0s. Only the Tolerance Model has a summed hazard quotient above 1, indicating potential risk. In the other cases, the summed hazard quotients are <1, and a strict interpretation of the RfD implies there is no noncancer risk with OP/Carbamates or their substitutes.

Exposure estimates are limited by incomplete data on use patterns of the different pesticides. For the Tolerance Model of residues, it was assumed that all OP/Carbamates reported as used on a crop are used on all acres of that crop. This is clearly an overestimate since many of the OP/Carbamates are substitutes for each other and use varies in accordance with regional differences in pest prevalence. The notion that the PDP data reflect actual use patterns is behind the Use Model of exposure.

The comparison is biased against the OP/Carbamates since for many pests and many crops, there are no alternative pesticides registered and available. In these cases it was implicitly assumed that there is no countervailing risk.

A more thorough evaluation of trade-offs would use full distributions of pesticide residue levels, account for changes in quantities of pesticides used following a ban, and include the complete range of registered pesticides for each relevant crop.

Standard methods for assessing noncancer risks are inadequate for RTA. The hazard quotient does not allow predicting the likelihood of an adverse effect, nor does it directly address how the likelihood changes with changes in exposure, unless the change causes the hazard quotient to fall below (or rise above) 1.

Even if the probabilities of the target and countervailing risks could be estimated, evaluation of the magnitude of noncancer trade-offs is complicated by the relative severity of toxic effects. A hazard quotient of 1 for an OP/Carbamate based on red blood cell Che inhibition presumably has different implications than a hazard quotient of 1 for a substitute pesticide that is based on kidney toxicity, developmental toxicity, or more severe neurotoxic endpoints such as ataxia. The nonsystematic application of the FQPA additional safety factor for developmental effects (the "children's 10×") further complicates the problem. One potential solution might be the use of surrogate values that are explicitly free of endpoint attribution, such as ratios of exposure to LD_{50}, similar to that suggested by Layton, Mallon, Rosenblatt, and Small (1987) and performed by Hammitt (1986, 1993).

Analysis of the noncancer risks that would be reduced by an OP/Carbamate ban, and the risks posed by alternative pesticides, highlights the inability of current risk assessment methods to inform comparisons (Gray, 1996). Use of actual exposure data from the USDA PDP suggests that for most crops, the chronic noncancer risk is 0 if the RfD or ADI is truly a level of exposure without potential for harm, even in sensitive populations (Barnes & Dourson, 1988).

The countervailing risks would also presumably be 0. Of course, the notion of adding hazard indices across crops when pesticides have a common mechanism of action (as called for in the FQPA) could lead to summed hazard quotients above 1, even with the use of measured residue data. There are considerable technical uncertainties in combining hazard quotients across chemicals, and a comprehensive comparison of OP/Carbamates and their substitutes would demand a similar analysis for all pesticides, as well as the natural constituents like the Che inhibiting substances in potatoes.

4.3. Pesticide Cancer Risks

Comparison of the potential cancer risks of OP/Carbamates and their substitutes is more straightforward since risks can theoretically be summed across pesticides. Clearly, for some crops, the decrease in cancer risk from a ban on OP/Carbamates is at least partially offset by potential cancer risks from the substitutes. For others, for example, tomatoes, neither the OP/Carbamates currently used nor the alternative pesticides are suspected of carcinogenicity. In other cases, a carcinogenic substitute could replace an OP/Carbamate classified by the EPA as noncarcinogenic. Regardless of the method used for estimating exposure, the estimated cancer risks from consumption of OP/Carbamate or substitute pesticide residues on food are quite small.

The magnitude of the cancer risk reduction achieved by an OP/Carbamate ban depends on the method of estimating exposure, just as in the noncancer case. For the example of potatoes (Table V), in the tolerance model an OP/Carbamate ban would reduce the upper-bound estimate of risk by 5.08×10^{-5} ($5.23 \times 10^{-5} - 1.47 \times 10^{-6}$). The smallest estimate of benefit is from the Use Model of exposure, which predicts, at most, a 3.5×10^{-9} decrease in cancer risk with an OP/Carbamate ban. Another way to look at these changes is the percentage of the risk reduced by an OP/Carbamate ban taking into account the offsetting risk induced by the alternatives. This calculation—(OP/Carbamate Risk − Substitute Risk)/OP/Carbamate Risk—yields a 97% decrease in risk for the Tolerance Model, but only a 13% decrease for the Use Model.

A complicating factor in the comparison of cancer risks is the assumption of low-dose linearity for all carcinogens. The cancer risk for OP/Carbamates on potatoes is due to the fungicide mancozeb (a carbamate) that is thought to have a mode of action indicating a very nonlinear dose–response relationship, perhaps with a threshold (U.S. Environmental Protection Agency, 1998). If mancozeb truly has safe, nonzero, levels of exposure, applying a conservative risk assessment approach does not provide greater safety because one would be misled about the relative risk of alternative pesticides. Risk could increase if the substitute pesticide has carcinogenic potential while the banned OP/Carbamate poses no cancer risk at actual exposure levels.

Similar to noncancer effects, a more complete analysis would better characterize exposure and would account for model uncertainty in dose response to ensure useful comparisons (Gray, 1996).

4.4. Natural Toxins in Food

Interest in the relative risks of natural and synthetic chemicals in the human food supply prompted a National Research Council (NRC) report entitled *Carcinogens and Anticarcinogens in the Human Diet* (National Research Council, 1996). The NRC found that there are no clear differences in modes of action between natural and synthetic carcinogens, and concluded that carcinogenic potencies of the compounds were very similar. The report also suggested that the natural components of food may be "of greater concern" to public health since human exposures are significantly greater than those to synthetic carcinogens (i.e., pesticides) in the diet. This report, along with research on the relative toxicity of natural compounds in plants compared to pesticides (e.g. Ames *et al.*, 1990) prompted us to evaluate this potential countervailing risk.

In order for the natural pesticides produced by plants to be a countervailing risk, there must be a difference in their levels in plants grown with and without OP/Carbamates. There are two ways in which this could occur: (1) through increased use of resistant varieties of crop plants containing higher levels of natural toxins and (2) if increased predation by pests increases levels of natural pesticides.

There is some reason to think that an OP/Carbamate ban could prompt an increase in the use of resistant cultivars; survey data indicate that 62% of organic farmers, who use no synthetic pesticides, report using insect-resistant plant varieties as a key pest-control practice (U.S. Department of Agriculture, 1996b). Loss of OP/Carbamates could result in increased planting of resistant varieties by conventional farmers. The amount of use of resistant varieties by conventional farmers is unknown. There are examples of human toxicity with resistant-bred vari-

eties of plants. For example, an insect-resistant potato variety had to be withdrawn from the market because it contained levels of chaconine and solanine—two natural Che-inhibiting neurotoxins that are also teratogenic—that were causing toxicity in humans (Beier, 1990). An alternative method for increasing pest resistance is genetic engineering. For example, genetically modified corn, cotton, and potatoes contain a gene from the soil microorganism *Bacillus thuringiensis* that confers resistance to specific insects. If genetically modified crops were thought to be a major alternative, a risk/risk trade-off of an OP/Carbamate ban should explicitly address the risks from this technology.

A general response of plants to stress, the phytoalexin response, can increase the level of natural pesticides in plants (Beier, 1990). Many experts believe that substitutes for OP/Carbamates are less effective in controlling pests (Gilreath *et al.*, 1999; Wiese & Guenthner, 1999). If the stress of increased pest predation results in higher levels of natural toxins, this could be a significant countervailing risk. For example, levels of the natural toxin psoralen, a skin irritant (and potential mutagen and carcinogen), were found to increase 100-fold when celery plants were damaged by mold (Ames, Magaw, & Gold, 1987).

Another class of natural toxins of potential concern are mycotoxins produced by fungal pests. Rodriguez-Kábana and Taylor (1999) suggest that the loss of the OP chlorpyrifos in peanut production could lead to increased levels of aflatoxin, a fungal product that is a known—and very potent—human carcinogen.

Although there are few specific data on changes in the levels of natural toxins that might occur should OP/Carbamates be banned, there is enough general knowledge about plant physiology and the toxicology of natural pesticides and mycotoxins to suggest that this is a countervailing risk worthy of further attention. A comprehensive RTA should consider the differences in the levels of natural toxins with and without the use of OP/Carbamates, along with food consumption patterns, to estimate the magnitude of the countervailing risk.

4.5. Nutrient Changes

Changes in diet and the accompanying changes in consumption of both beneficial and detrimental components of food are an often-overlooked source of countervailing and corollary target risks. Results from Taylor and Smith (1999) predict both positive and negative changes in the average diet following an OP/Carbamate ban. These estimates are derived from predictions of changes in food intake following an OP/Carbamate ban and data on the nutrient content of various foods. The present study follows the results from Taylor and Smith (1999).

Although the percentage changes in daily consumption are small, the potential number of persons affected is fairly great. A large fraction of the U.S. population already falls below RDIs for many nutrients. In many cases, poor or minority persons have even lower nutrient intakes. Decreases in vitamins and nutrients may increase risk in two ways: by reducing the disease-fighting benefits of the nutrients or by inducing damage through deficiencies. For example, 60% of the U.S. population consumes less than the RDI of folate (Alaimo *et al.*, 1994). Folate deficiency is associated with increased risk of chromosome breaks and cancer (Blount *et al.*, 1997), heart disease (Verhoef *et al.*, 1996), and birth defects (Shaw, Lammer, Wasserman, O'Malley, & Tolarova, 1995). Recently introduced supplementation of flour products may reduce folate deficiencies.

There may also be corollary benefits from diet changes induced by an OP/Carbamate ban, including a predicted decrease in consumption of cholesterol, saturated fat, and total fat. These decreases are linked to short-term increases in meat prices as feed for livestock becomes more expensive. Excess cholesterol has been linked to heart disease, and lower fat intake (from a decrease in meat consumption) can reduce risks of heart disease and colon cancer. The increased intake of certain vitamins from dietary changes may also improve general health.

It is difficult to weigh the countervailing and corollary nutritional risks of an OP/Carbamate ban. It is not a matter of just being able to sum the numbers of people changing from a better or worse nutrient intake status because of possible correlations in consumption. As an example, the same people who get less folate may also be taking in less vitamin C. Moreover, a change in status for one food component may have greater health implications than for others. Uncertainty about these relationships means that there are considerable difficulties in translating small changes in consumption of food components into risk. A refinement of this study might use epidemiologic data to estimate the net increase or decrease in sickness and deaths that might result from predicted changes in nutrient consumption.

4.6. Mortality Induced by Changes in Income

The effect of higher food production costs on disposable income and mortality risk is the countervailing risk for which the most comprehensive esti-

mate can be provided. Although there are uncertainties about the magnitude of the effect of reduced income on mortality risk, the existence of such a link correlates with common sense and is well supported by empirical estimates. Intuitively, it seems unlikely that a reduction in income would cause people to spend more (on net) in ways that reduce mortality risk. Empirically, there is a wealth of evidence showing a positive correlation between health, longevity, and income (Keeney, 1990; Smith, 1999; Wildavsky, 1988). Identifying the causal effect of income on health is more difficult, since causality runs both ways, but a significant body of literature using alternative data sets and statistical methods yields roughly comparable estimates of the cost per induced fatality for the U.S. population, on the order of millions to tens of millions of dollars (Chapman & Harriharan, 1994, 1996; Keeney, 1990, 1997; Viscusi, 1994).

Changes in food expenditures and those in disposable income resulting from an OP/Carbamate ban depend on the magnitude of the increased production costs for crops that use OP/Carbamates and the effects of these increases on patterns of food demand, production, and purchase. The changes in production costs are estimated by agricultural economists and scientists familiar with each crop, but are necessarily somewhat speculative as farmers might find superior pest-control alternatives than those the experts predict. It is often claimed that the costs of prospective environmental regulations are overestimated *ex ante*, because analysts fail to foresee the variety of cost-saving innovations that will occur once regulations are adopted (Goodstein & Hodges, 1997; Porter & van der Linde, 1995). Logic and analysis of specific cases, however, suggest that costs are not invariably or even systematically overestimated (Palmer, Oates, & Portney, 1995; Harrington, Morgenstern, & Nelson, 1999; Hammitt, 2000).

Effects of higher production costs on food prices, consumption, and income are estimated using a sophisticated and widely used general-equilibrium economic model, IMPLAN (Minnesota IMPLAN Group Inc., 1997). The estimated increase in household expenditures on food depends on estimates of the supply and demand elasticities (how production and demand for specific crops shifts with price changes), which are reasonably well estimated. The estimated $840 million increase in household expenditure (see Table VII) represents a lower estimate of the economic impact of an OP/Carbamate ban. The estimated $5.5 billion loss in personal income is more uncertain as it depends on modeling the effects of higher food prices on supply and demand of all other market goods as well as sectoral employment.

In summary, the existence of a countervailing health risk due to the higher costs of production is reasonably certain. What is less certain is the magnitude of this risk, which depends on the magnitude of the increase in production costs and how these higher costs influence the food sector and the overall economy.

4.7. Other Countervailing Risks

In addition to health risks associated with agricultural use, several other risks deserve notice. These include pesticide use to control disease vectors, development of resistance to pesticides, and ecotoxicity.

The OP/Carbamate pesticides have public health as well as agricultural value. They are used to control mosquitoes, cockroaches, and other vectors of insect-borne disease. If an OP/Carbamate ban were to limit control of these vectors, rates of encephalitis, malaria, and other deadly diseases could increase.

On the other hand, use of pesticides can act as a selective pressure and may lead to pest populations developing resistance to the pesticidal activity of specific compounds. There are known cases of resistance to certain OP/Carbamates. Resistance is of even greater concern for many of the substitute pesticides. Peacock (1999), for example, suggests that leafhoppers on grapes will develop severe resistance to imidacloprid within 3 to 5 years of the loss of OP/Carbamates. Any weighing of risks should include the changes in efficacy that might occur over time in control of specific pests. In addition, the loss of OP/Carbamates would decrease the number of pesticides that farmers could use, leading to increased likelihood of resistance.

OP/Carbamates are broad-spectrum pesticides, effective against multiple pests. If multiple, narrow-spectrum substitutes are required, pesticide use and potential risk could be increased. More selective pesticides, however, may be less likely to kill nontarget species.

The OP/Carbamate family of pesticides have a mode of toxicity common to many organisms and there are concerns about nontarget species being harmed by routine use of these compounds. Substitute pesticides have their own profiles of nontarget toxicity that will potentially offset some or all of the gain achieved through restriction of the use of OP/Carbamates. For example, Musick (1999) reports that lambda cyhalothrin is a registered and moderately effective alternative to carbofuran for control of rice

water weevil larvae in a water-seeded rice production system. Lambda cyhalothrin, however, is highly toxic to crawfish. In Louisiana, about 120,000 acres of cultured crawfish ponds are in or adjacent to water-seeded rice fields. In this case, use of the substitute pesticide could pose a threat to the approximately $30 million annual production of Louisiana crawfish. A thorough risk/risk analysis should evaluate the toxicity to nontarget species of substitute pesticides or cultivation practices (including the need to bring more land under cultivation) that would result from an OP/Carbamate ban.

5. SUMMARY

The goal of a comprehensive evaluation of risk/risk trade-offs is an estimate of the net change in health that might occur from a regulatory action. This net benefit (or harm) aggregates the reduction in the target risk, and additional benefits that might accrue, with the countervailing risks that would be created. Comparing across different health endpoints requires the use of an integrated measure of risk, such as changes in Quality-Adjusted Life Years (QALYs) (Gold, 1996).

In this analysis, both target and countervailing human health risks of a complete ban on the use of OP/Carbamate pesticides have been identified. Quantification of these risks, in order to find the net change in risk, proves to be quite difficult. Some impediments are amenable to further data collection and analysis; others are rooted in current methods of risk assessment and are more difficult to address.

A key source of difficulty in reliably estimating risks of pesticide use is the lack of data on residue levels on food for many crop/pesticide combinations. The USDA PDP provides very useful data, but does not analyze many foods and many pesticides, especially newer compounds not detected with multiresidue screens. Given the importance of exposure estimates in characterizing risk (Tables IV and V), better residue monitoring would seem necessary to sound pesticide management.

Understanding consumer risk also requires better data on food consumption patterns. Data on long-term average consumption and correlations among foods consumed would be very helpful. Available data (the CSFII) report only 2- to 3-day consumption patterns.

Another source of uncertainty that is, however, amenable to data collection is the illness or injury that farmworkers incur through pesticide use. Current data rely on self-reports of pesticide use and coincidence of use and illness. A more systematic monitoring program would use biomarkers of exposure, such as metabolites in blood or urine, to relate a specific injury or illness to a specific pesticide.

The question of natural toxins in food, and potential changes with alterations in pest-control practices, requires more attention. More systematic evaluation of levels of these plant pesticides in different varieties of crop plants grown under different conditions would determine whether differences among cultivars or between growing regimens lead to significant risk differences.

Changes in nutrient intake could conceivably be balanced through modeling of disease rates at different levels of food consumption. Aggregating these changes would require use of QALYs or a similar method of aggregating across diverse health endpoints.

The greatest challenges to risk/risk comparisons arise from current methods of regulatory risk assessment for pesticides. For noncancer endpoints, available methods do not allow estimation of the probability of harm or even identification of the specific harm that might be expected. Reference doses based on toxicologic endpoints of different severity for different pesticides make even relative comparisons treacherous. Differential conservatism in cancer risk estimates, a result of assumptions being more or less appropriate for different pesticides, makes risk comparisons potentially misleading.

These technical difficulties prevent calculating a net risk change in the current analysis. Qualitatively, it can be said that the benefits of an OP/Carbamate ban will be significantly offset by countervailing risks. It is difficult to imagine that the benefits of a ban would offset the 10–1,000 annual premature fatalities predicted from the income losses that would be caused by elimination of OP/Carbamate use.

In spite of these many concerns, it is apparent that sound management of pesticide risks and benefits demands that we "look before we leap" and examine potential countervailing risks. Understanding foreseeable consequences to health is necessary to ensure that actions do more good than harm (Wiener, 1998). It is also important to look at the distribution of risk across the population. It may influence decision makers to know that, while small benefits accrue to many people, certain groups like farmers or farmworkers may bear a disproportionate share of the countervailing risks. It is hoped that this preliminary exercise helps illustrate the value of explicit identifi-

cation of trade-offs and the need for quantitative analysis to ensure that policies do not inadvertently increase net health risk.

ACKNOWLEDGMENTS

This research was supported by the American Farm Bureau Federation. The authors thank Pamela Williams, John Hoseman, Scott Rawlins, and anonymous reviewers for helpful discussion and suggestions.

REFERENCES

Alaimo, K., McDowell, M. A., Briefel, R. R., Bischof, A. M., Caughman, C. R., Loria, C. M., & Johnson, C. L. (1994). *Dietary intake of vitamins, minerals, and fiber of persons aged 2 months and over in the United States: Third national health and nutrition examination survey, Phase 1, 1988–91.* Centers for Disease Control and Prevention/National Center for Health Statistics. Advance Data Number 255.

American Heart Association. (1999) Dietary Guidelines for Healthy American Adults. www.americanheart.org/Heart and Stroke A Z Guide/dietg.html (10 September 2000).

Ames, B. N., Magaw, R., & Gold, L. S. (1987). Ranking possible carcinogenic hazards. *Science, 236*, 271–280.

Ames, B. N., Profet, M., & Gold, L. S. (1990). Nature's chemicals and synthetic chemicals: Comparative toxicology. *Proceedings of the National Academy of Sciences, USA, 87*, 7782–7786.

Anderson, E. L., and the Carcinogen Assessment Group of the U.S. Environmental Protection Agency. (1983). Quantitative approaches in use to assess cancer risk. *Risk Analysis, 3*, 277–295.

Barnes, D. G., & Dourson, M. (1988). Reference dose (RfD): Description and use in health risk assessments. *Regulatory Toxicology and Pharmacology, 8*, 471–486.

Beier, R. C. (1990). Natural pesticides and bioactive components in food. *Reviews of Environmental Contamination and Toxicology, 113*, 47–137.

Blount, B. C., Mack, M. M., Wehr, C. M., MacGregor, J. T., Hiatt, R. A., Wang, G., Wickramasinghe, S. N., Everson, R. B., & Ames, B. N. (1997). Folate deficiency causes uracil misincorporation into human DNA and chromosome breakage: Implications for cancer and neuronal damage. *Proceedings of the National Academy of Sciences, USA, 94*, 3290–3295.

California Environmental Protection Agency, Information Systems Branch, Department of Pesticide Regulation (1995). *Pesticide Use Reporting: Overview of California's Unique Full Reporting System.* Sacamento CA: Author. http: // www.cdpr.ca.gov/docs/pur/purmenu.htm (29 June 1999).

California Environmental Protection Agency, Department of Pesticide Regulation, Worker Health and Safety Branch. (1997). *Health and safety report HS-1768: Overview of the California Pesticide Illness Surveillance Program 1995.* Sacramento, CA: Author.

Chapman, K. S., & Hariharan, G. (1994). Controlling for causality in the link from income to mortality. *Journal of Risk and Uncertainty, 8*, 85–94.

Chapman, K. S., & Hariharan, G. (1996). Do poor people have a stronger relationship between income and mortality than the rich? Implications of panel data for health-health analysis. *Journal of Risk and Uncertainty, 12*, 51–63.

Chen, W. L., Sheets, J. J., Nolan, R. J., & Mattsson, J. L. (1999). Human red blood cell acetylcholinesterase inhibition as the appropriate and conservative surrogate endpoint for establishing chlorpyrifos reference dose. *Regulatory Toxicology and Pharmacology, 29*, 15–22.

Eilrich, G. L. (1991). Tracking the fate of residues from the farm gate to the table. In B. G. Tweedy, H. J. Dishburger, L. G. Ballantine, & J. McCarthy (eds.), *Pesticides and food safety* (pp. 202–212). Washington, DC: American Chemical Society.

Gilreath, P., Smith, S., & Taylor, T. G. (1999). *Impacts of the elimination of organophosphates and carbamates from tomato production* (AFPC Policy Research Report 99-13). College Station, TX: Agricultural Food and Policy Center, Texas A&M University.

Gold, M. R., Siegel, J. E., Russel, L. B., & Weinstein, M. C. (1996). Cost-effectiveness in health and medicine. Oxford, UK: Oxford University Press.

Goodstein, E., & Hodges, H. (1997). Polluted data: Overestimating environmental costs. *The American Prospect, 35*, 64–69.

Graham, J. D., & Wiener, J. B. (1995). Confronting risk tradeoffs. In J. D. Graham & J. B. Wiener (Eds.), *Risk vs. Risk* (pp. 1–41). Cambridge, MA: Harvard University Press.

Gray, G. M. (1996). *Key issues in environmental risk comparisons: Removing distortions and ensuring fairness* (Reason Foundation Policy Study No. 205). Los Angeles: Reason Foundation, Los Angeles.

Gray, G. M., & Graham, J. D. (1995). Regulating pesticides. In J. D. Graham & J. B. Wiener (Eds.), *Risk vs. Risk* (pp. 173–192). Cambridge, MA: Harvard University Press.

Hammitt, J. K. (1986). *Estimating consumer willingness to pay to reduce food-borne risk* (R-3447-EPA). Santa Monica, CA: RAND Corporation.

Hammitt, J. K. (1993). Consumer willingness to pay to avoid pesticide residues. *Statistica Sinica, 3*(2), 351–366.

Hammitt, J. K. (2000). Are the costs of proposed environmental regulations overestimated? Evidence from the CFC phaseout. *Environmental and Resource Economics, 16*(3), 281–301.

Harrington, W., Morgenstern, R. D., & Nelson, P. (1999). *On the accuracy of regulatory cost estimates* (Working Paper 99-18). Washington, DC: Resources for the Future.

Insecticide, Fungicide, and Rodenticide Act of 1948, as amended, 40 C.F.R. § 180 Subpart C.

Keeney, R. (1990). Mortality risks induced by economic expenditures. *Risk Analysis, 10*, 147–159.

Keeney, R. (1997). Estimating fatalities induced by the economic costs of regulations. *Journal of Risk and Uncertainty, 14*, 5–23.

Knutson, R. D., & Smith, E. G. (1999). *Impacts of the elimination of organophosphates and carbamates from crop production* (AFPC Policy Research Report 99-2). College Station, TX: Agricultural and Food Policy Center, Texas A&M University.

Layton, D. W., Mallon, B. J., Rosenblatt, D. H., & Small, M. J. (1987). Deriving allowable daily intakes for systemic toxicants lacking chronic toxicity data. *Regulatory Toxicology and Pharmacology, 7*, 96–112.

Lutter, R., & Morrall, J. F., III (1994). Health-health analysis: A new way to evaluate health and safety regulation. *Journal of Risk and Uncertainty, 8*, 43–66.

McDowell, M. A., Briefel, R. R., Alaimo, K., Bischof, A. M., Caughman, C. R., Carroll, M. D., Loria, C. M., & Johnson, C. L. (1994). *Energy and macronutrient intakes of persons aged 2 months and over in the United States: Third national health and nutrition examination survey, Phase 1, 1988–91.* Centers for Disease Prevention and Control/National Center for Health Statistics. Advance Data Number 255.

Minnesota IMPLAN Group, Inc. (1997). IMPLAN professional: Social accounting & impact analysis software (2nd ed.) [computer software]. Stillwater, MN: Author.

Musick, J. (1999). *Impacts of the elimination of organophosphates and carbamates from rice production* (AFPC Policy Research Report 99-10), College Station, TX: Agricultural and Food Policy Center, Texas A&M University.

National Research Council. (1996). *Carcinogens and anti-Carcinogens in the Human Diet.* Washington, DC: National Academy of Sciences, National Academy Press.

Nielsen, John (Reporter). (1999, August 3). *Pesticides: All Things Considered.* National Public Radio.

Palmer, K., Oates, W. E., & Portney, P. R. (1995). Tightening environmental standards: The benefit-cost or the no-cost paradigm? *Journal of Economic Perspectives, 9*, 119–132.

Peacock, W. J. (1999). *Impacts of the elimination of organophosphates and carbamates from table grape and raisin grape production* (AFPC Policy Research Report 99-5). College Station, TX: Agricultural and Food Policy Center, Texas A&M University.

Peel, M. D., & Aakre, D. (1999). *Impacts of the elimination of organophosphates and carbamates from wheat production* (AFPC Policy Research Report 99-14). College Station, TX: Agricultural and Food Policy Center, Texas A&M University.

Porter, M. E., & van der Linde, C. (1995). Toward a new conception of the environment–competitiveness relationship. *Journal of Economic Perspectives, 9*, 97–118.

Rodriguez-Kábana, R., & Taylor, C. R. (1999). Impacts of the elimination of organophosphates and carbamates from peanut production (AFPC Policy Research Report 99-8). College Station, TX: Agricultural and Food Policy Center, Texas A&M University.

Shaw, G. M., Lammer, E. J., Wasserman, C. R., O'Malley, C. D., & Tolarova, M. M. (1995). Risks of orofacial clefts in children born to women using multivitamins containing folic acid periconceptionally. *The Lancet, 346*, 393–396.

Smith, J. P. (1999). Health bodies and thick wallets: The direct relations between health and economic status. *Journal of Economic Perspectives, 13*, 145–166.

Taylor, C. R. (1993). AGSIM: An econometric-simulation model of regional crop and national livestock production in the United States. In C. R. Taylor, S. R. Johnson, & K. H. Reichelderfer (Eds.), *Agricultural sector models for the United States: Description and selected policy applications* (pp. 31–59). Ames, IA: Iowa State University Press.

Taylor, C. R., & Smith, H. A. (1999). *Aggregate economic evaluation of banning organophosphate and carbamate pesticides* (AFPC Policy Research Report 99-15). College Station, TX: Agricultural and Food Policy Center, Texas A&M University.

U.S. Bureau of the Census. (1998, December 28). Resident Population of the United States: Estimates by Age and Sex. http://www.census.gov/(10 September 2000).

U.S. Department of Agriculture (USDA). (1995). *Pesticide Data Program 1995.* Washington, DC: USDA Agricultural Marketing Program.

U.S. Department of Agriculture (USDA). (1996a, May). Organic vegetable growers surveyed in 1994. AREI Updates on Agricultural Resources and Environmental Indicators, No. 4. Economic Research Service, Natural Resources and Environmental Division. http://www.ers.usda.gov:80/epubs/pdf/arei/96upd/upd96-4.pdf (10 September 2000).

U.S. Department of Agriculture (USDA). (1996b). *Pesticide Data Program 1996.* Washington, DC: USDA Agricultural Marketing Program.

U.S. Department of Agriculture (USDA). (1996c). *The Continuing Survey of Food Intakes by Individuals (CSFII) and the Diet and Health Knowledge Survey (DHKS), 1994–96.* http://www.barc.usda.gov:80/bhnrc/foodsurvey/Csfii94.html (10 September 2000).

U.S. Department of Agriculture (USDA). (1997). *Pesticide Data Program 1997.* Washington, DC: USDA Agricultural Marketing Program.

U.S. Department of Health and Human Services, Food and Drug Administration, Food Labeling: Reference Daily Intakes, 60 Fed. Reg. 249 (1995, December 28).

U.S. Environmental Protection Agency. (1986). *Guidelines for carcinogen risk assessment* (51 FR33994-33997). Washington, DC: Author.

U.S. Environmental Protection Agency. (1997). *Office of pesticide programs reference dose tracking report.* Washington, DC: Author.

U.S. Environmental Protection Agency. (1998). *Assessment of thyroid follicular cell tumors. Risk assessment forum* (EPA/630/R-97/002). Washington, DC: Author.

U.S. Environmental Protection Agency. (1999, December 22). *Azinphos-methyl; Proposed revocation and lowering of certain tolerances; Tolerance actions* (64FR71708-71712). Washington, DC: Author.

U.S. Environmental Protection Agency, Office of Pesticide Programs. (1999, May). *Organophosphate Pesticides in Food: A Primer on Reassessment of Residue Limits.* http://www.epa.gov/pesticides/op/primer.htm. (24 May 1999).

Verhoef, P., Kok, F. J., Kruyssen, D. A., Schouten, E. G., Witteman, J. C., Grobbee, D. E., Ueland, P. M., & Refsum, H. (1996). Homocysteine metabolism and risk of myocardial infarction: Relation with vitamins B-6, B-12, & folate. *American Journal of Epidemiology, 143*, 845–859.

Viscusi, W. K. (1993). The value of risks to life and health. *Journal of Economic Literature, 31*, 1912–1946.

Viscusi, W. K. (1994a). Mortality effects of regulatory costs and policy evaluation criteria. *RAND Journal of Economics, 225*, 94–109.

Viscusi, W. K. (1994b). Risk-risk analysis. *Journal of Risk and Uncertainty, 8*, 5–18.

Wiener, J. B. (1998). Managing the iatrogenic risks of risk management. *Risk: Health, Safety and Environment, 9*, 39.

Wiese, M. V., & Guenthner, J. F. (1999). *Impacts of the elimination of organophosphates and carbamates from potato production* (AFPC Policy Research Report 99-9). College Station, TX: Agricultural and Food Policy Center, Texas A&M University, AFPC.

Wiese, R., Helmers, G. A., & Shaik, S. (1999). *Impacts of the elimination of organophosphates and carbamates from soybean production* (AFPC Policy Research Report 99-12). College Station, TX: Agricultural and Food Policy Center, Texas A&M University.

Wildavsky, A. (1979). No risk is the highest risk of all. *American Scientist, 67*, 32–37.

Wildavsky, A. (1980). Richer is safer. *The Public Interest, 60*, 23–39.

Wildavsky, A. (1988). *Searching for safety.* New Brunswick, NJ: Transaction Books.