HEALTH TRADEOFFS IN PESTICIDE REGULATION

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Abstract

EPA has the authority to ban pesticides to reduce health risks to consumers from food residues. Such bans influence the price of fruits and vegetables, and the resulting consumption shifts impact consumer health. We develop a framework to compare the direct and indirect health effects of pesticide regulation, and investigate the distribution of these effects across social groups. Under some plausible scenarios, the increased incidence of disease from reduced fruit and vegetable consumption outweigh the direct benefits of regulation. Furthermore, high income consumers receive the greatest direct health benefit from pesticide cancellations, whereas low and medium income consumers are most hurt by the resulting dietary changes.

Keywords: health and environment, pesticides, regulation, risk tradeoffs

JEL Codes: I18, I12, Q18

1 Introduction

Pesticide regulation in the United States is focused in large part on the carcinogenic risks posed by pesticide residues on food, as determined by laboratory animal tests. Since these policies are designed to protect human health, it is a worthwhile question to consider whether the implementation of a pesticide ban gives rise to any countervailing risks that partially offset the benefits of the ban. In this study we investigate the health risks that result from producers' and consumers' economic responses to such bans. It is important to note that we are not directly addressing those actions undertaken primarily for protection of agricultural workers or the environment, although this analysis has implications for any policies that may affect food prices.

Regulators have generally paid attention to the direct benefits of banning a pesticide use, which lowers the public's exposure to residue of that target pesticide. Some attention has been given in the regulatory process to the risks posed by those chemicals that may be used in place of a banned pesticide, but the risks that raised by economic responses to the regulations have generally not been considered (Ralston, 1999). This is despite statutory justification for taking these effects into account; changes to the pesticide laws under the 1996 Food Quality Protection Act (FQPA) allow the setting of tolerances that would not otherwise meet the prevailing safety standard if "use of the pesticide prevents even greater health risks to consumers or the lack of the pesticide would result in a significant disruption in domestic production of an adequate, wholesome, and economical food supply" (United States Environmental Protection Agency, Office of Pesticide Programs, 1999).

It is generally true that removing a pesticide from the production process will result in an increase in the price of the treated commodity (Zilberman, Schmitz, Casterline, Lichtenberg and Siebert, 1991; Ralston, 1999). If consumers respond to the increased prices by reducing consumption of the affected fruits and vegetables (and perhaps shifting consumption to less nutritious foods), they will lose the health benefits associated with the change in consumption. This study illustrates the magnitude of some of these tradeoffs. In addition, the people most affected by dietary changes may be different from those who receive the greatest direct health benefit from the pesticide regulation. We therefore also investigate the income distribution of these health effects.

2   Health-based Regulation of Pesticides

Federal involvement in the regulation of pesticides dates back to 1910, when Congress passed the Insecticide Act, prohibiting the sale of fraudulently labelled pesticides (Brown et al., 2000). It was not until 1964, however, that Congress empowered the executive branch to ban a pesticide on the basis of potential hazard to the public. Since 1972, protecting farmworker and consumer safety has been the primary focus of pesticide regulation, as carried out by the Food and Drug Administration (FDA) and the Environmental Protection Agency (EPA). In this study the focus is on consumer safety protections.

Under the Federal Food, Drug, and Cosmetic Act (FFDCA), EPA is responsible for setting a pesticide residue tolerance for food crops and products. For new products, EPA either sets a tolerance and registers the pesticide under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA), or denies the registration request, effectively banning the substance from commercial use. Similarly, EPA can ban substances currently in use if new toxicological data suggests that the pesticide may pose a significant health risk.

The establishment of these product-specific tolerance levels is driven mainly by evidence of carcinogenic and other toxic effects in laboratory animal tests. If these experiments find

that a pesticide causes a statistically significant increase in tumors in the test animals, EPA uses quantitative risk assessment to estimate the potential cancer risk for humans. The risks are expressed as the probability of developing cancer after a lifetime of exposure, e.g., one in 1,000,000 or one in 10,000 (Gray and Hammitt, 2000). Although there is no one acceptable risk level, EPA is required to set tolerances that are "safe," defined as "a reasonable certainty that no harm will result from aggregate exposure to the pesticide chemical residue" (United States Environmental Protection Agency, Office of Pesticide Programs, 1999).[1] In the absence of carcinogenic effects in laboratory animals, the tolerances are based upon the highest dose at which no toxic effects were observed in animal tests, which is then divided by a safety factor (usually 10 or 100) to determine an acceptable daily dose for humans.

There has been considerable controversy over the applicability of laboratory animal tests to determining cancer risks in humans. Detractors argue that the testing method itself, and not simply exposure to the pesticide in question, may often be responsible for observed carcinogenic effects in animals (Ames and Gold, 1997). Similarly, some deem the establishment of tolerances of non-carcinogenic compounds by dividing a dose observed to be harmless in small mammals by a safety factor to be overly cautious. Even if the resulting standards are more protective than are necessary to assure the public health and this comes at an increased cost to consumers, producers, and/or regulators, such an approach can be justified on "safety-first" or "precautionary principle" grounds if the regulatory measure does not give rise to offsetting health risks. Indeed, "when examining the risk and benefits of a pesticide, EPA typically assumes that revoking the registration of the pesticide will eliminate the associated risks to human health and the environment" (Gray and Hammitt, 2000).

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[1]Prior to the 1996 FQPA amendments to the FFDCA and FIFRA, EPA was required to establish tolerances that would "protect the public health." Although the FQPA was to establish a single, health-based standard for all pesticide residues, this refers to eliminating different protection levels based on how the food is processed or when the pesticide was first registered for use; it does not refer to a uniform level of risk tolerance.

3 Trading One Risk for Another

The wisdom of applying the precautionary principle to the evaluation and control of cancer risk from a pesticide becomes less obvious, however, in the presence of countervailing risks. Farmers will typically replace banned pesticides with other compounds or pest control techniques, and these substitutes may carry risks of their own. Additionally, if substitutes are less effective in controlling the target pest than the original substance was, there may be increased human health hazards from the higher pest population. In the case of fungicides, the pest of interest may itself be carcinogenic to humans. There is likewise no guarantee that a standard designed to lower human cancer risk will also be protective of the environment, or vice-versa. A human versus ecological health trade-off may have been made when DDT was banned by the EPA in 1972. Although DDT is persistent in the environment and threatened wildlife, the organophosphate pesticides that were used by many farmers to replace DDT are more acutely toxic and resulted in increased incidents of farmworker poisoning. The unavailability of DDT for use against malaria-bearing insects may have also contributed to the increased incidence of that disease in recent decades (Gray and Hammitt, 2000).

It is also possible that current pesticide regulations are trading one risk to consumers for another. As described in Section 4 below, there is an extensive body of research showing the health benefits of consuming fruits and vegetables. Such findings suggest that a reduction of fruit and vegetable consumption resulting from an increase in commodity prices may result in nontrivial health effects. Although in this paper we are focusing on changes in the incidence of certain outcomes, it is important to keep in mind that there are substantial associated costs as well. The USDA estimates that healthier diets might prevent $71 billion *per annum* in medical costs, productivity losses, and premature deaths (Frazão, 1999).

Economists and political scientists have been writing about risk tradeoffs in public policy for over twenty years. Lave (1981) pointed out that competing policies often differ, and can be compared, by the risks they entail. Wildavsky (1979) is credited with formalizing

the concept that when society attempts to control risks, it diverts resources that could be used for other productive purposes. In recent years, researchers at the AEI-Brookings Joint Center for Regulatory Studies, the Harvard Center for Risk Analysis, and elsewhere have generated a large body of work in the field of "Risk Tradeoff Analysis." A recent book on the topic covers such diverse topics as elderly drivers, automobile fuel-efficiency standards, and the treatment of schizophrenia (Graham and Wiener, 1995b). Such research has in turn contributed to the highly politicized debate on the proper role of government.

Graham and Wiener (1995a) have developed a useful typology for consideration of risk tradeoffs, summarized in Table 1 below. They first distinguish between the "target risk" that a government policy seeks to reduce, and a "countervailing risk" that may arise as an unintended consequence of the policy. They also pay attention to who experiences the risks, noting that sometimes countervailing risks may be faced by the same people who face the target risk, and sometimes these may be different groups. When a risky outcome is shifted from one group to another, this is termed a "risk transfer," and when the same outcome is created in the target population, it is labelled a "risk offset." When one adverse outcome is replaced by another within the same population, this is "risk substitution," and when the countervailing risk gives rise to different negative outcomes in a different population, this is called a "risk transformation." The potential adverse effects of pesticide regulation from decreased fruit and vegetable consumption falls under the categories of "risk substitution" and "risk transformation" in this classification scheme.

Table 1: Typology of Risk Tradeoffs (Graham and Wiener, 1995a)

		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

Other authors have previously noted the possible indirect effects of federal pesticide programs. Ralston (1999) includes pesticide regulation in her review of government policies that influence consumer's dietary choices, noting that restrictions may increase price, while the existence of such programs affect consumer confidence in the food supply. Goddard (2002) discusses the public health benefits of pesticides, arguing that pesticides are necessary tools in the fight against infectious diseases, particularly given the modern capability for rapid travel from one region of the world to another. Gray and Graham (1995) follow the typology described above to discuss the types of risk tradeoffs that may arise in pesticide regulation. Their list of possible tradeoffs includes acute toxicity to farmworkers resulting from substitute pesticides, non-cancer and cancer toxicity to consumers arising from substitutes, risks from natural toxins in foods, risks from changes in diet, and mortality induced by decreases in income. A later study by Gray and Hammitt (2000) extends this analysis to quantify the effects of a hypothetical ban on organophosphate and carbamate insecticides. Although the Gray and Hammitt study reports the possible impact of such a pesticide ban on the population's intake of individual nutrients, they do not relate this to an estimate of disease incidence as we seek to do here. Instead, their findings report the number of people changing to a "worse (or better) health state" for each of twenty nutrients.

4 Benefits of Fruit and Vegetable Consumption

Numerous health benefits have been associated with consuming a diet rich in a wide variety of fruits and vegetables (Van Duyn and Pivonka, 2000). Scientific evidence is accumulating for a protective effect for fruits and vegetables in prevention of cancer (Steinmetz and Potter, 1996; World Cancer Research Fund, 1997), coronary heart disease (Ness and Powles, 1997; Liu, Manson, Lee, Cole, Hennekens, Willett and Buring, 2000; Joshipura, Hu et al., 2001), ischemic stroke (Joshipura, Ascherio et al., 1999; Feldman, 2001), hypertension (Appel et al., 1997), diabetes mellitus (Ford and Mokdad, 2001), chronic obstructive pulmonary

disease (Miedema, Feskens, Heederik and Kromhout, 1993), and diverticulosis (Aldoori, Giovannucci, Wing, Trichopoulos and Willett, 1994; Aldoori, Giovannucci, Rockett, Sampson, Rimm and Willett, 1998). The level of protection suggested by these studies is often quite dramatic. A recent review of several studies found that "the quarter of the population with the lowest dietary intake of fruits and vegetables compared to the quarter with the highest intake has roughly twice the cancer rate for most types of cancer" (Ames, Gold and Willett, 1995).

High consumption of fruits and vegetables (Neumark-Sztainer, Story, Resnick and Blum, 1996; Kahn, Tatham, Rodriguez, Calle, Thun and C.W. Heath, 1997; Müller, Koertringer, Mast, Languix and Frunch, 1999; Epstein, Gordy, Raynor, Beddome, Kilanowski and Paluch, 2001) or consumption of a wide variety of vegetables (McCrory, Fuss, Saltzman and Roberts, 2000) has also been related to a lower prevalence of obesity or reduced weight gain. Further, evidence suggests that focusing on increasing intake of healthy foods, such as fruits and vegetables, may be more effective at reducing weight than focusing on decreasing intake of unhealthy foods, such as high fat and high sugar items (Epstein et al., 2001). Taken together, the evidence on consumption patterns and health benefits supports interventions to increase consumption of a wide variety of fruits and vegetables.

Compared to studies of general fruit and vegetable consumption, the results of studies of the benefits of specific nutrients to reduction of cancer and other health risks have been less uniform and conclusive. In their review of the literature of risk factors for cancer, Ames et al. (1995) explain that although "antioxidants in fruits and vegetables may account for a good part of their beneficial effect ... [it is] difficult to disentagle by epidemiological studies [these effects] from other important vitamins and ingredients in fruits and vegetables." Fruit and vegetable fiber intake have also proven to be important factors for reducing the incidence of certain diseases (Rimm et al., 1996). Such considerations suggest that individuals' aggregate intake of fruits and vegetables is an appropriate level of analysis for investigating health outcomes.

5   Price Impacts of Pesticide Regulation

Banning the use of a pesticide is likely to increase the prices consumers face for fruits and vegetables. Profit-maximizing farmers tend to use the most effective and least costly pesticides. If a used input is no longer available, the productions costs will probably increase. Such increases "are passed on to consumers to whatever degree the market will bear" (Ralston, 1999). Furthermore, if overall yields go down, the resulting supply shift also causes an increase in the market price to consumers.

Table 2: Simulated Impacts of a Pesticide Ban on Five California Crops

Crop	Impact	Output Change (%)	Price Change (%)	Consumer Spending
Almonds	Mean	-15	21	-94
	High	-34	59	-256
Grapes	Mean	-19	29	-358
	High	-52	87	-1031
Lettuce	Mean	-9	57	-321
	High	-28	175	-931
Oranges	Mean	-21	13	-53
	High	-41	29	-114
Strawberries	Mean	-25	18	-57
	High	-53	37	-114

Consumer spending is in hundred thousands of 1990 dollars. The high impact estimate is the value which may be exceeded with a 5% probability (Zilberman et al., 1991).

Several studies have investigated the potential cost to consumers of pesticide regulations. In a 1991 *Science* article, Zilberman et al. (1991) investigated the potential impacts of "Big Green," a failed California ballot initiative that sought to phase out all food-use pesticides known to cause cancer or reproductive damage. Their results for five California crops are presented in Table 2. A similar investigation of the impacts of a complete nationwide pesticide ban on major commodities found price increases could range as high as 83% for rice, 100% for soybeans, and 146% for peanuts (Knutson, Taylor, Pensen and Smith, 1990). Even less dramatic pesticide bans can have significant impacts. A recent study of the effects of banning organophosphate and carbamate pesticides, currently under review by EPA, pre-

dicts price increases of 5.02% for tomatoes, 4.17% for fruit juice, and 6.24% for apples (Taylor and Smith, 1999).

6   Indirect Health Effects of Food Quality Regulation

Consider a regulator that is focusing on only one source of risk to the public health. Such a regulator may adopt or modify an environmental policy Q to reduce cancer risk from intake of pesticide-contaminated crops by a factor of 1 in l exposed individuals, i.e., for every l people in the population consuming fruit and vegetables, one case of cancer will be prevented. In a population of N exposed individuals, this policy change should prevent $\frac{N}{l}$ cases of cancer.

If the policy also raises consumer prices of fruits and vegetables, there may be countervailing risks, such as increased likelihood of ischemic stroke, that affect the target population. Consumer i's relative risk of ischemic stroke is determined by:

$$H_i^s = g(X_i^{fv}, X_i^{of}, Z_i) \tag{1}$$

where Z_i is a vector of other factors influencing stroke risk.

By the chain rule, the increase in this individual's stroke risk resulting from the policy change is:

$$\frac{\partial H_i^s}{\partial Q} = \frac{\partial H_i^s}{\partial X_i^{fv}} \frac{\partial X_i^{fv}}{\partial p^{fv}} \frac{\partial p^{fv}}{\partial Q} \tag{2}$$

The increased incidence of ischemic stroke in the population is therefore given by $\sum_{i=1}^{N} \frac{\partial H_i^s}{\partial Q}$. Summing over all diseases of interest gives us the total mortality offset for the policy change.

Note that the expression given in (2) relies on two further assumptions. The first is that the policy in question does not affect the price of other foods. Perhaps more realistically, we can use the same formulation by standardizing p^{of} to be unity. The other assumption is that the change in policy does not shift consumers' demand for fruits and vegetables. If we are considering marginal changes in pesticide policy and consumers that have already been

conditioned by thirty years of modern pesticide regulation, this does not seem unreasonable. Most consumers are also unaware of specific regulatory actions.[2] Furthermore, one could argue that since there exists a market for foods produced without the use of pesticides, the consumers most likely to respond to a small change in pesticide policy have already sorted themselves out of the conventional produce market.

7   Methodology and Data

This study involves a series of policy simulations designed to quantify some of the countervailing health risks that may result from pesticide regulation. The initial element in each simulation is the hypothetical ban of a pesticide or class of pesticides that results in a broad-based increase in the market price of fruits and vegetables. The change in the intake levels of a sample population of consumers is then calculated. Finally, the dietary changes in the sample are related to a dose-response function to yield the increased health risk and corresponding incidence in the population of coronary heart disease and ischemic stroke.

Coronary heart disease and ischemic stroke were chosen for inclusion in this analysis for two reasons. First, they are two of the major causes of death in the United States. They were also the subject of two extensive studies (Joshipura et al., 2001; Joshipura et al., 1999) conducted by Harvard researchers and published in major medical journals in the last three years. These studies were based upon large panel surveys of over 110,000 medical professionals, with 8 years of follow-up for men and 14 years of follow-up for women. These studies divide the sample populations into quintiles of fruit and vegetable consumption and then calculate the relative risk of the disease of interest for members of each quintile, controlling for factors such as age, smoking status, alcohol intake, family history, weight,

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[2]For example, on October 31, 2001, EPA announced the cancellation of several uses of two organophosphate pesticides, phosmet and azinphos-methyl. Three crop uses of phosmet are being cancelled voluntarily. Twenty-eight uses of azinphos-methyl are being cancelled, and seven more uses are being phased out over the next four years (Breen, 2001). The authors learned of these cancellations through a trade publication with a very limited audience, and could not find reference to EPA's action in any major news source, including CNN.com, the *New York Times*, the *Washington Post*, or the Associated Press newswire service.

supplement use, and exercise level. These studies provide strong evidence that relative risk decreases as fruit and vegetable consumption increases. For example, men in the highest quintile had 20% less risk of coronary heart disease, and 39% less risk of ischemic stroke, than men in the lowest quintile. Joshipura et al. also break their findings down by broad categories of fruit and vegetable type. The findings for differences in consumption of all fruits and vegetables are further summarized in Table 3 below.

Table 3: Relative Risk of Ischemic Stroke and Coronary Heart Disease, by Quintile of Fruit and Vegetable Intake and per Serving per Day

	1st Quintile	3rd Quintile	5th Quintile	1 Serving/Day
Ischemic Stroke				
Women	1.0	0.75	0.74	0.93
Men	1.0	0.70	0.61	0.96
Pooled	1.0	0.73	0.69	0.94
Coronary Heart Disease				
Women	1.0	0.88	0.80	0.97
Men	1.0	0.95	0.80	0.96
Pooled	1.0	0.92	0.80	0.96

Risks by quintile of intake are relative to the risk for the lowest quintile of intake, and are adjusted for age, smoking status, alcohol intake, family history of myocardial infarction, body mass index, vitamin supplement use, vitamin E use, physical activity, aspirin use, hypertension, hypercholesterolemia, total energy intake, and postmenopausal hormone use (among women). One serving per day is risk reduction per one-serving increment, using median values for the quintile of intake (Joshipura et al., 1999; Joshipura et al., 2001).

As noted above, aggregate intake of fruit and vegetables is an appropriate level of analysis for considering health outcomes. These studies allow for the calculation of dose-response functions describing the increase in specific health risks resulting from reduced consumption of broad categories of fruits and vegetables, including all fruits and vegetables, all fruits, all vegetables, total citrus fruits, citrus fruit juices, cruciferous vegetables, green leafy vegetables, etc. In contrast, the literature relating intake of specific nutrients to the incidence of diseases is neither complete nor uniform enough to apply it to overall dietary patterns.

The reported results from these studies suggest that the relative risk curves generally follows a log-linear shape. Using these reported results, we estimated parameterized curves for use in our simulations. When calibrated to an appropriate set of baseline risks for the

control group (here, the observed incidence for the quintile with the lowest consumption of fruits and vegetables), the relative risk curves yield dose-response functions for the protective benefits of fruit and vegetable consumption. The results that we report in Section 8 below are to be considered preliminary because of the limited number of observations that were used to calculate these curves.[3]

In order to quantify the health outcomes from dietary changes resulting from federal policies, it is necessary to have information on the consumption habits of a representative sample of the population. The sample population used in our study is the 18,081 individuals over the age of two included in the U.S. Department of Agriculture's Continuing Study of Food Intakes by Individuals (CSFII) for 1994-1996 and 1998 (United States Department of Agriculture, 2000a). The CSFII comes with a set of sampling weights that allows extrapolation of our analysis to the entire U.S. population, i.e., 253.9 million people over two years of age. CSFII data include detailed information on individual food and nutrient intake, dietary knowledge, attitude, and behavior, and household demographics. A set of "cookbooks" available from USDA was used to convert the consumption data in the CSFII to food pyramid servings (United States Department of Agriculture, 2000b).

The consumption response of the individuals in the sample to the simulated changes in the price of fruits and vegetables is described by demand elasticities, which give the percentage change in quantity demanded for a one-percent change in price. Here we used recent elasticities for fruit and vegetable consumption calculated by researchers at the U.S. Department of Agriculture, shown in Table 4 below (Huang and Lin, 2000). These elasticities are particularly appropriate for this study as they are segmented by income level, and were calculated from a earlier survey similar in format to the CSFII.

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[3]It is our intention to more accurately estimate the dose-response functions by obtaining the complete underlying data used in these medical studies. Nevertheless, we do not anticipate that the final results will change drastically. The main advantage of a formal estimation based upon the underlying data is that it will allow for simulation of meaningful confidence intervals based upon the likely variation in individuals' dose-response relationships.

Table 4: Own-Price Demand Elasticities by Income Group

Commodity	All Incomes	Low Income	Medium Income	High Income
Fruit	-0.7196	-0.6472	-0.6614	- 0.7523
	(0.0282)	(0.0693)	(0.0469)	(0.0409)
Vegetables	-0.7238	-0.6965	-0.7436	-0.7087
	(0.0179)	(0.0391)	(0.0301)	(0.0272)
Juice	-1.0109	-1.0498	-0.8997	-1.0387
	(0.0364)	(0.0837)	(0.0591)	(0.0563)

Low income refers to families below 130% of the poverty income guidelines, and high income households are above 300 percent of this level. Numbers in parentheses are standard errors. (Huang and Lin, 2000)

8   Preliminary Results

In order to be able to calculate health outcomes, we are constrained by the methodology and scope of the medical literature. As a result, our simulations are limited to changes in the price of broad categories of produce. This means we can not estimate the health outcomes resulting from a pesticide ban that, for example, causes a three percent change in the price of certain tomato varieties but impacts no other crops. Nonetheless, there are pesticide regulations under consideration by EPA, such as the current review of all organophosphate and carbamate chemicals, that would impact the price of fruits and vegetables generally, as noted in section 5 above. Moreover, it should be noted that the pesticide programs keep many new substances from being introduced to agricultural production. The exercise conducted here would apply equally to any price-reducing innovation that is not adopted because of regulator concerns regarding human cancer risk.

The results reported in Table 5 below describe the health outcomes of a pesticide cancellation that causes a lasting average one percent increase in the price of all fruits, all vegetables, or all fruits and vegetables. By "lasting" we mean a change in price that persists at least as long as the study period of the medical research used in the simulations. The number of induced diseases reported is the mean from an extensive series of Monte Carlo trials. Standard errors reflect the likely variations in individuals' economic responses to the price change. In each trial, every individual in the sample was assigned a different elastic-

ity drawn from the distribution implied by Huang and Lin's findings. Health outcomes are shown for the entire population, as well as by income group.

Table 5: Cases of Coronary Heart Disease and Ischemic Stroke Induced in the U.S. Population by a 1% Price Increase in All Fruits, All Vegetables, or All Fruits and Vegetables

Disease	All Incomes	Low Income	Medium Income	High Income
	All Fruits			
Coronary Heart Disease	1,442	231	422	789
	(51.06)	(23.80)	(26.02)	(36.65)
Ischemic Stroke	744	132	225	386
	(29.90)	(14.30)	(16.42)	(20.39)
Total	2,186	363	648	1,175
	(72.11)	(33.98)	(37.93)	(50.65)
	All Vegetables			
Coronary Heart Disease	2,950	528	1,008	1,414
	(53.62)	(23.01)	(29.70)	(38.20)
Ischemic Stroke	1,482	285	507	690
	(32.04)	(13.56)	(18.07)	(22.73)
Total	4,433	813	1,516	2,104
	(82.60)	(35.23)	(46.14)	(58.72)
	All Fruits and Vegetables			
Coronary Heart Disease	6,904	1,152	2,260	3,492
	(119.64)	(53.28)	(63.63)	(85.58)
Ischemic Stroke	3,022	568	997	1,457
	(58.78)	(26.24)	(32.81)	(41.06)
Total	9,926	1,720	3,257	4,949
	(160.44)	(71.50)	(86.80)	(113.68)

Results reported are the simulation means and standard errors (in parentheses) from a series of Monte Carlo trials (n=100,000). Low income refers to families below 130% of the poverty income guidelines, and high income households are above 300 percent of this level. Number of cases across income groups may not sum perfectly to amount shown in "all income" column because of rounding.

For a one percent increase in the average price of all fruits and vegetables, the simulations indicate an increase of 6,904 cases of coronary heart disease and 3,022 ischemic strokes. In order to offset these 9,926 cases in a population of 253.9 million people, a pesticide action would have to prevent 1 in 25,580 cancers. This is almost four times as protective as the mean risk of pesticide uses that were banned between 1975 and 1989 (Van Houtven and Cropper, 1996).

As described above, EPA bases its regulatory decisions on increases in cancer risk over

a *lifetime* of exposure. Here we have used the number of incidences of ischemic stroke and coronary heart disease observed during the duration of the Joshipura studies as our baseline. It is certain that a substantial number of the participants in these studies experienced ischemic stroke or coronary heart disease since the study period ended, or will do so in the future. Since our baseline risk is not calculated on a lifetime basis, the quantities calculated in these simulations are likely to be under-estimates. These numbers also do not reflect a complete accounting of all negative health outcomes, but just those from two major causes of death. Furthermore, recall that EPA extrapolates risk estimates from animals studies by incorporating an arbitrary safety factor. Taken together, it seems quite likely that some pesticide regulatory actions will harm more people than they protect.

It is also of interest to examine the income distribution of these induced morbidities and mortalities. This focus is not just of theoretical interest but is also supported by language in the FQPA, which charges federal regulators with considering "dietary consumption patterns and variations in the sensitivities of major identifiable subpopulations" (United States Environmental Protection Agency, Office of Pesticide Programs, 1999). If we assume that the linear dose-response function used by EPA for calculating dietary cancer risk are correct, we find that the people saved from reducing dietary exposure to carcinogens tend to have higher incomes (as they are consuming more fresh fruits and vegetables, and therefore have greater exposure). For example, if we assumed that the simulated policy involves no net loss of lives – i.e., total cancer deaths exactly equal the number of induced negative health outcomes – we find that for a one percent change in the price of all fruits and vegetables, approximately 275 high income cancer cases are shifted to low and medium income consumers. Other distributional effects of such a "break-even" policy are shown in Table 6 below.

Table 6: Distribution of Avoided Cancers in a Break-Even Scenario for a 1% Price Increase in All Fruits, All Vegetables, or All Fruits and Vegetables

Health Outcome	All Incomes	Low Income	Medium Income	High Income
		All Fruits		
Cancers Avoided	2,186	354	682	1,151
Cases Induced	2,186	363	648	1,175
Change	0	+ 9	- 34	+ 24
		All Vegetables		
Cancers Avoided	4,433	717	1,383	2,333
Cases Induced	4,433	813	1,516	2,104
Change	0	+ 96	+ 133	- 229
		All Fruits and Vegetables		
Cancers Avoided	9,926	1,606	3,096	5,223
Cases Induced	9,926	1,720	3,257	4,949
Change	0	+ 114	+ 161	- 274

"Cancers Avoided" calculated assuming a linear dose-response function for cancer. "Cases Induced" is the total of ischemic stroke and coronary heart disease from Table 5. Columns may not sum perfectly due to rounding.

9 Conclusions

The regulation of pesticides to prevent cancer from dietary exposure to carcinogens involves risk tradeoffs which should be taken into consideration. We have illustrated that certain plausible regulatory scenarios will lead to increased incidence of common health problems resulting from decreased fruit and vegetable consumption. Some regulations may result in a shifting of health risks from certain classes of consumers to others. Furthermore, the overall effect of some pesticide bans is that more lives may be lost than saved.

The argument presented here should not be taken to be anti-organic, or even pro-pesticide. The authors are among those who, everything being equal, would prefer to consume fewer pesticide residues in their own diets. Yet an overly protective focus on cancer risk from pesticide residues that ignores countervailing health risks is misguided, as the net effect on public health may be negative. This is even more poignant when you consider that certain pesticide uses have been cancelled by the EPA on the basis of consumer risks that were less than one in a million over a *lifetime* of exposure – the same amount of incremental *annual* risk you incur of suffering a fatal accident from spending six minutes in canoe or travelling

150 miles by car (Wilson, 1979). It is also worth noting that cancer risk is a poor proxy for other concerns involved in agricultural pesticide use. Banning uses on the basis of over-estimates of cancer risk does little to protect consumers from possible endocrine disruption or neurological effects, prevent human health risks from contamination of water supplies, or guarantee ecological health. In fact, banning a pesticide on the basis of human cancer risk may even increase the damage to animals, drinking water systems, etc., as the replacement pesticides may be hazardous in those respects but exhibit minimal human carcinogenicity.

We also believe there are other applications of this analysis in considering the health implications of a wide variety of government policies. Any policy that affects the price of fruits and vegetables is likely to influence consumption behavior and, ultimately, public health. These simulations could be used to model the indirect impacts of other food quality regulations, trade restrictions, and farm price supports.

It is interesting to consider the existing policy debate surrounding the framework adopted here. In recent years a highly politicized debate over "risk tradeoff analysis" has emerged. The recent controversy over the appointment of John Graham, one of the authors cited above, as Administrator of the Federal Office of Information and Regulatory Affairs highlighted some of these disagreements. On one extreme are anti-regulatory ideologues who cite any example of risk tradeoffs as a general indictment of government intervention in the areas of health and environment. At the other pole are those who will not accept that the existence of any level of harm or risk can be socially optimal.

The objective analyst presumably operates between these two extremes. Regulatory tradeoffs are perhaps inevitable and their mere existence should not paralyze regulation; the use of pesticides involve significant externalities that justify government involvement. Yet a social optimum can not be achieved if countervailing risks posed by regulations designed to protect human health are ignored. It is certainly possible to conceive of some policies that are more effective than others. Our goal here has been to illustrate how great the tradeoffs in current pesticide policy may be. We also hope that, as the role that diet plays in

human health becomes better understood, EPA will apply models similar to ours to provide guidance in future regulatory decisions.

As mentioned above, we consider these results to be preliminary. We wish to expand this work by revisiting the data underlying the medical studies used here. This will allow for the estimation of more accurate dose-response curves and confidence intervals that can be incorporated into the Monte Carlo trials performed here to better account for uncertainty. We would also like to extend the sensitivity analyses to cover measurement error in consumption or income. Finally, it would be of interest to investigate the distribution of health impacts over region and ethnic group as well as income level.

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