

Table 1 Exposure to air pollution and morbidity in adults and children: Odds ratios and 95% confidence intervals

Symptom or disease	Odds Ratio	Lower Confidence	Upper limit
A. Respondents			
Winter cough & cold	1.432202	0.973711	- 2.106581
Permanent cough without cold	1.718250	1.267392	- 2.329494
Winter cough without cold	1.434010	1.151725	- 1.785482
Permanent phlegm & cold	1.572290	0.979119	- 2.524816
Winter phlegm & cold	1.400047	0.977708	- 2.004823
Permanent phlegm without cold	1.347164	1.024256	- 1.771871
Winter phlegm without cold	1.282863	1.040762	- 1.581281
Cough & phlegm	1.566648	1.247142	- 1.968010
Winter phlegm & cold	1.574363	1.252040	- 1.979665
Wheezing & cold	1.358134	1.052843	- 1.751950
Wheezing while breathing	1.352105	1.065619	- 1.715611
Dyspnoea	1.802163	1.530547	- 2.121982
Rhinitis	1.305185	1.043520	- 1.632463
Eye "infection"	1.302482	1.030010	- 1.647031
Headache	1.595975	1.356035	- 1.878371
B. Children			
Cough or phlegm & cold	1.695780	1.209402	- 2.377761
Cough or phlegm without cold	1.922437	1.365540	- 2.706449
Cough or phlegm	1.969765	1.528539	- 2.538353
Wheezing with cold	1.694218	1.150931	- 2.493961
Wheezing	1.466871	1.130402	- 1.903492
Asthma or bronchitis	1.487382	1.139776	- 1.940998
Pneumonia	1.269068	1.008890	- 1.596343
Rhinitis	1.271813	1.013503	- 1.595958
Eye "infection"	1.495645	1.109688	- 2.015841

Table 2. Willingness to pay equations - nonzero bids only

Explanatory variable	Regression coefficients	
	WTP ^c	WTP ^e
<u>Demographic and socioeconomic variables:</u>		
Age (years)	-7.86 (0.29)	-0.73 (0.073)
Sex (1=female)	55.28 (4.76)	
Education (years)	12.18 (0.71)	
Blue collar worker (1=blue collar)	-53.33 (6.80)	
Number of children ages 0-18	-24.64 (2.19)	-6.93 (1.56)
Ethnic origin I (1=born in Africa/Asia)	-26.93 (6.20)	
Ethnic origin II (1=born in Europe)	109.38 (6.38)	
Annual municipal taxes		0.22 (0.006)
<u>Attitudinal variables:</u>		
Perceived exposure to pollution at work (1=yes)	81.29 (5.29)	14.42 (4.31)
Perceived neighborhood air quality (1-6)	-21.14 (1.51)	
Believes budget share allocated to pollution abatment too high	-382.22 (57.85)	101.72 (38.87)
Believes budget share allocated to pollution abatment too low	163.85 (5.90)	
Ready to devote time to public activities concerned with pollution abatement (1=yes)	39.62 (1.64)	5.54 (1.30)
Perception of government influence on pollution abatement (1=yes)	-26.99 (5.90)	
Pollution induces defensive actions by respondent (1=yes)		8.63 (4.48)
<u>Health status</u>		
Perceived health status (1=not healthy)	-67.55 (5.46)	
Family history (exc. respondent) of asthma, pneumonia, or bronchitis (1=yes)	24.60 (4.78)	8.81 (4.29)
Family history exc. respondent) _{**} of respi- ratory system symptoms (1=yes)	55.91 (4.58)	
Adjustment factor	-952.98 (23.63)	*
Intercept	7708.53	*
	Adj. R ²	0.54
		0.64

* Not significant.

** Cough, sputum, wheezing, dyspnoea

Table 3. CVM Experiments: WTP^C (in NIS, per household, excluding protest zero bids, except in binary choice)

Elicitation method	N	Mean	Median
Sample	2,518	34.5	
Standard max. WTP	1,855	37.7	
Repeat bids: <u>One-time payment</u>			
1st bids	343	26.4	
2nd bids	195	67.8 (+22.2)	
<u>Annual payment</u>			
1st bids	343	26.4	
2nd bids	195	67.8 (+22.2)	
Binary choice	360	66.2	65.0

Table 4. CVM Experiments: WTP^e (in NIS, per household, excluding protest zero bids except in binary choice)

Elicitation method	N	Mean	Median
Sample	1,704	68.6	
Standard max. WTP	1,348	70.9	
Repeat bids: <u>One-time payment</u>			
1st bids	199	64.2	
2nd bids	195	89.0	(+24.8)
<u>Annual payment</u>			
1st bids	157	54.5	
2nd bids	163	77.9	(+23.4)
Binary choice	360	69.1	67.2

Table 5. Direct (CVM) valuations of perceived air quality changes
(Includes zero bids)

Present pollution level	Pollution level after change			
	Good	Moderate	Poor	Very poor
	(a) <u>WTP^e</u>			
Good	Mean = 26			
	Median= 15			
	N = 847			
	(b) <u>WTP^c</u>		(c) <u>WTP^e</u>	
Moderate	Mean =37 .9		Mean = 40	
	Median= 28		Median= 28	
	N =750		N =749	
	(d) <u>WTP^c</u>		(e) <u>WTP^e</u>	
Poor	Mean =47 .2		Mean =42 .7	
	Median= 40		Median= 32	
	N = 192		N =192	

* Values in table refer to means and medians of the indicated sample air quality stratum, and stated in NIS per household per year.

Significance Levels:

Nonparametric median test for 2 samples:

$$H_0: WTP^c \text{ (cell b)} = WTP^c \text{ (cell d)} \dots\dots\dots 0.015$$

$$H_0: WTP^e \text{ (cell a)} = WTP^e \text{ (cell c)} \text{ WTP (cell e)} \dots\dots\dots 0.001$$

Paired t-test for means (2 tailed):

$$H_0: WTP^c \text{ (cell b)} = WTP^e \text{ (cell c)} \dots\dots\dots 0.001$$

$$H_0: WTP^c \text{ (cell d)} = WTP^e \text{ (cell e)} \dots\dots\dots 0.049$$

Table 6. Parameter Estimates of the Budget Share Equation*

Parameter	Estimate	Parameter	Estimate
α_1	-0.348 (-110.38)	ϕ_{11}	0.0006 (1.11)
γ_1	-0.721 (-15.74)	ϕ_{12}	-0.0009 (-0.73)
γ_2	-1.404 (-16.42)	ϕ_{13}	0.004 (3.27)
β_{11}	-0.181 (-21.06)	ϕ_{14}	0.00002 (0.04)
β_{12}	0.039 (2.49)	ϕ_{15}	0.0024 (2.12)
β_{22}	-0.159 (-4.90)		
δ_{11}	-0.417 (-12.16)	$R^2 = 0.27$	
δ_{12}	0.001 (0.06)	$N = 2,239$	
δ_{22}	-0.527 (-8.77)		

* Asymptotic t statistics in parentheses.

Table 7. Estimated Logit Regression: Consumption of Medical Care Services (Physician Visits) - Respondents

Explanatory Variable	Regression coefficient	Standard error
Intercept	-4.397	0.217
Health status	0.715	0.097
AV14	0.018	0.005
Sex (1=female)	0.405	0.134
No children 0-18 yrs. (1=none)	0.588	0.134
Birth origin Asia-Africa (1=yes)	0.346	0.154

n = 3,612

$\chi^2 = 125.5$ (5 df).

 Dependent variable: 1 = visited a physician in past 2 weeks

Health status 0 = healthy

1 = suffers from at least one of symptom

2 = suffers from at least 1 disease

Table 8. Estimated Logit Regression: Health Risks and Exposure to Pollution - Respondents

Explanatory Variable	Regression coefficient	Standard error
Intercept (h_1)	0.880	0.134
Intercept (h_2)	-0.732	0.134
AV14	0.011	0.002
Education (1=low level, 0-8 yrs.)	0.248	0.078
Birth origin (1=Europe or America)	0.285	0.072
Sex (1=female)	0.289	0.064
No children 0-18 yrs. (1=none)	0.254	0.088
Age of respondent (<40)	-0.845	0.120
Age of respondent (41-50)	-0.481	0.123
Age of respondent (51-60)	-0.372	0.102

n = 3,612

$\chi^2 = 316.5$ (8 df).

Dependent variable:

h_1 = suffers from at least 1 symptom or disease

h_2 = suffers from at least 1 disease

Table 9. Restricted activity or bed days

Explanatory Variable	Regression coefficient
AV14	0.028 (0.012)
Income (1= "low" income-below NIS 1,300/mo.)	0.80 (0.295)
Intercept	-3.79

$\chi^2 = 24.2$ (18 df).

Dependent variable: 1 = Stayed home at least 1 day during
the past two weeks.

Table 10. Comparisons Between Direct & Indirect Valuations
 (Including zero bids. Mean household values in NIS)

	WTP ^c	WTP ^e
<u>CVM</u>		
Standard bids	37.70	70..90
Repeat bids	67.80	89.00
Binary choice	66.20	69.10
<u>Indirect</u>		
Expenditure function	9.81	73.25
Health production	32.43	
Cost of illness (bed days)	185.0	

• Corresponding to changes in perceived pollution levels.

VALUATION of an ENVIRONMENTAL GOOD

DIRECT vs. INDIRECT APPROACHES

direct approach

indirect (market)
approach

contingent
valuation
(CVM)

↓
wtp

cost
of
illness

▼
house-
hold
production
function

▼
wtp

observed demand for
related market good(s)

▼
hedonic
models

▼
wtp

▼
preferences
system

▼
"exact"
welfare
measures

▼
wtp

FIGURE 1

ODD RATIOS FOR SYMPTOMS & DISEASES

(interviewee, children)

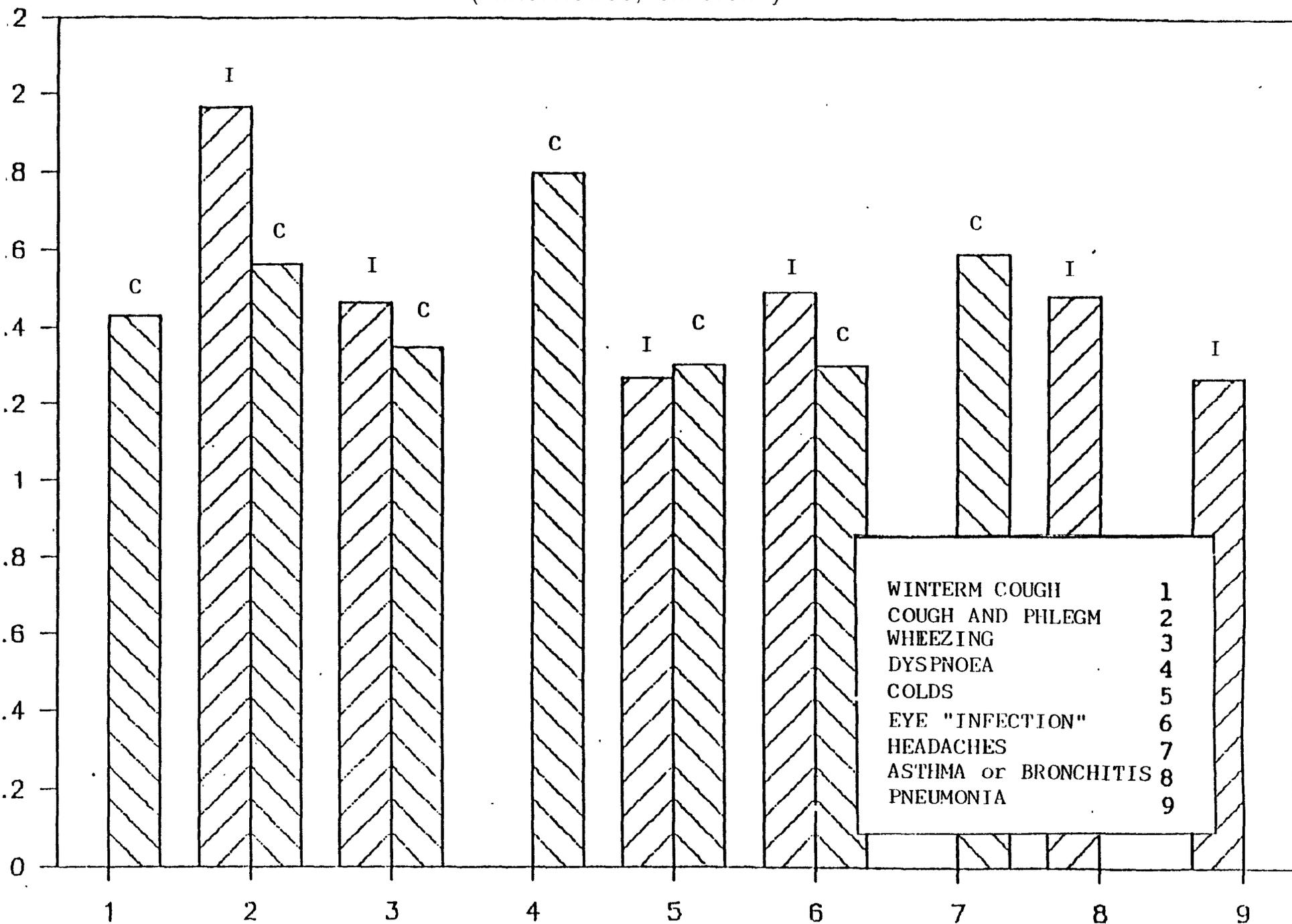


FIGURE 2

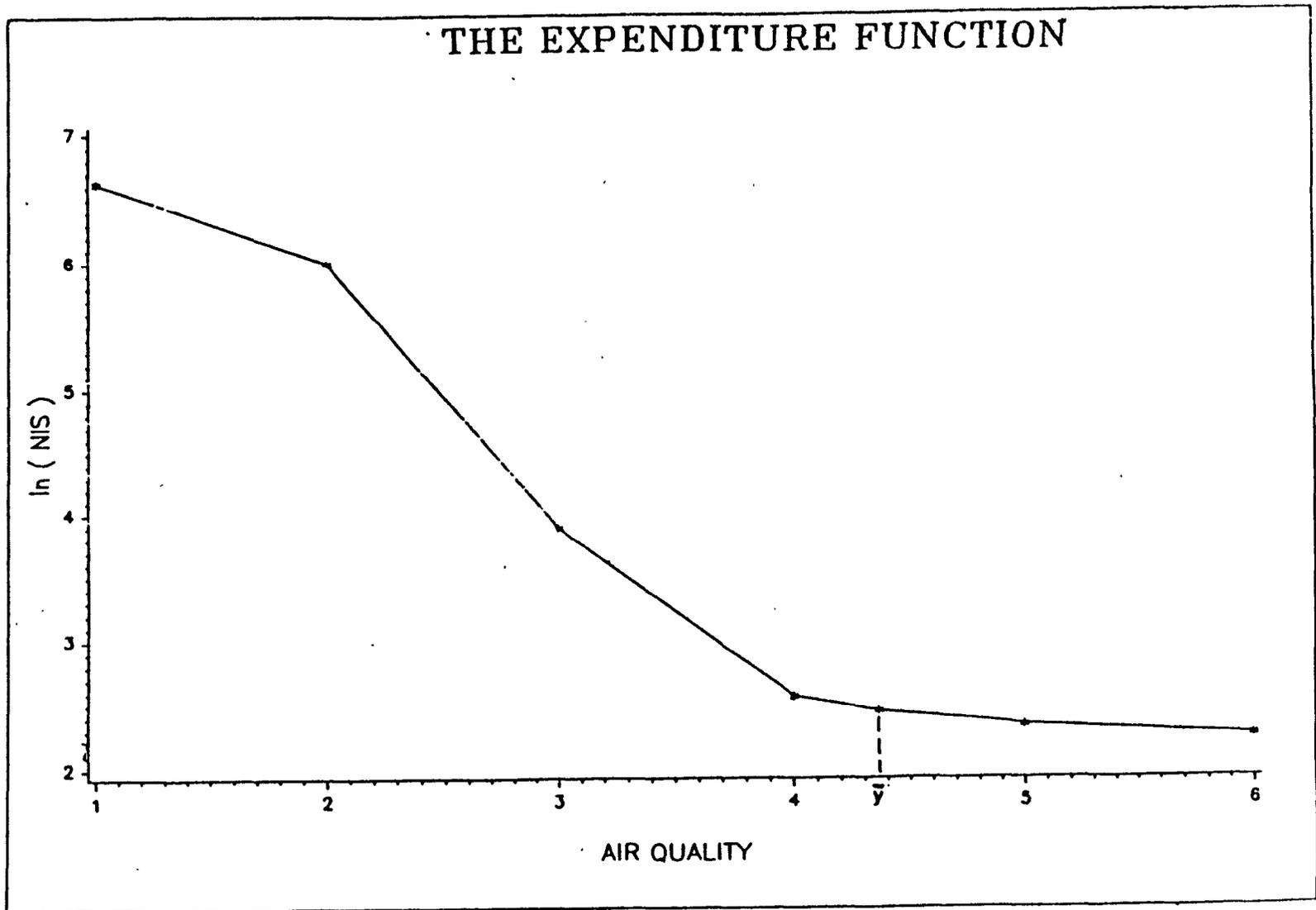


FIGURE 3

BID CURVES FOR AIR QUALITY

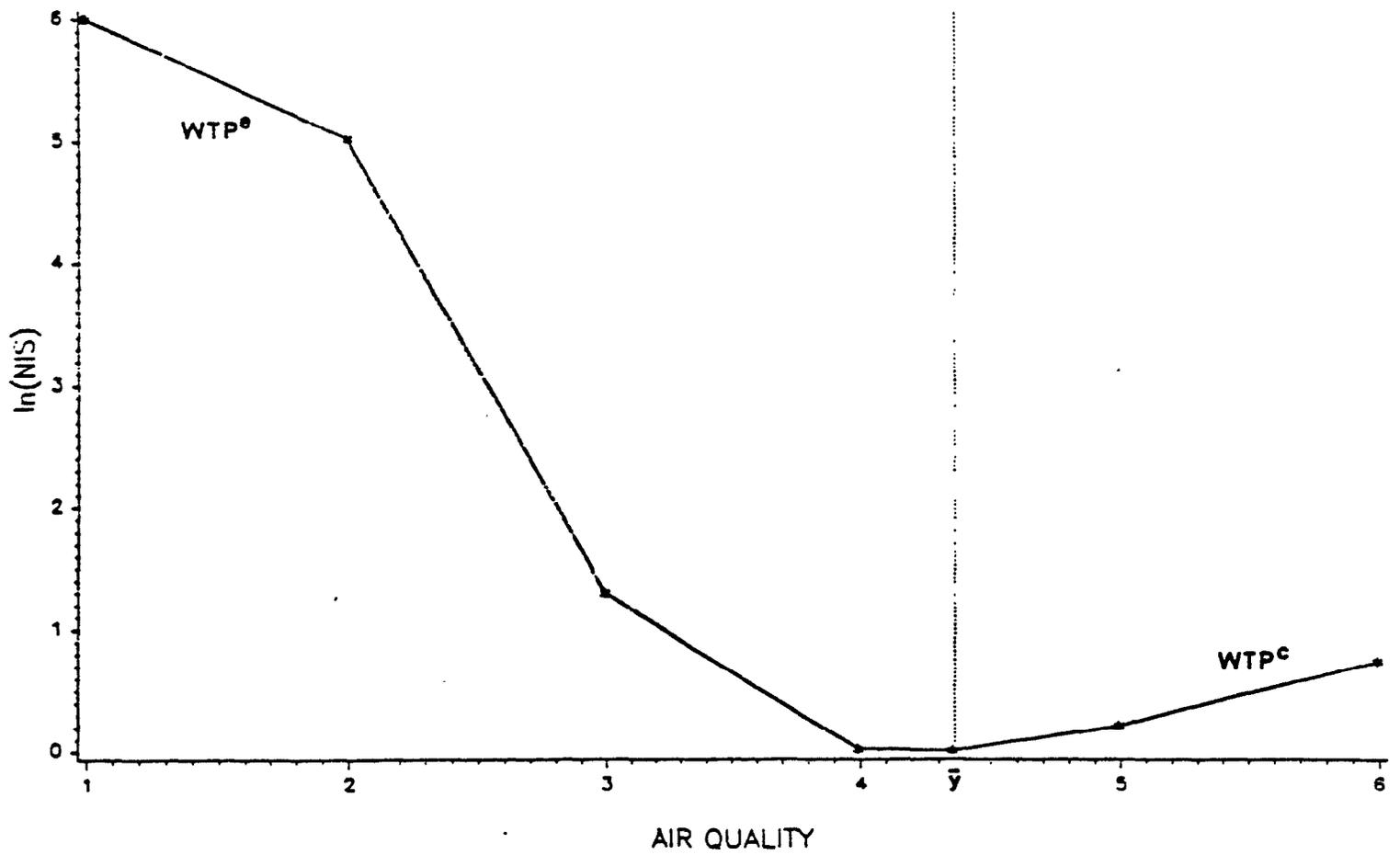


FIGURE 4

Risk,
Self-Protection
and
Ex Ante Economic Value*

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Abstract

We examine the impact of self-protection on the ex ante value of reduced human exposure to an environmental hazard. Assuming a continuous distribution of health outcomes and self-protection that influences both the probability and the severity of an undesired outcome, we develop three propositions:

- 1) If risk is endogenous such that self-protection influences the probability or the severity of an undesirable outcome, then unobservable utility terms cannot be eliminated from the individual's ex ante valuation expression.
- 2) If risk is endogenous, knowledge of the convexity or the nonconvexity of physical dose-response relations is insufficient to sign unambiguously the change in an individual's ex ante marginal valuation of risk, even when consumer cognition is perfect.
- 3) If risk is endogenous, self-protection expenditures will not be a consistent lower bound of the ex ante value that a risk-averse individual attaches to a reduction in risk.

These three statements imply that several propositions originally developed for cases of exogenous risk and which form the analytical basis for most recent empirical work on the value of health risk changes are not immediately transferable to settings where endogenous risks prevail.

I. INTRODUCTION

Any person who might suffer harm from exposure to an undesirable state of nature can reduce expected ex post costs by purchasing market insurance. Moral hazard, however, compels insurers to defray only a fraction of these costs [Arrow (1963), Shaven (1979)].^{1/} Consequently, individuals use self-protection to reduce both the ex ante probability and expected costs of the uninsured event [Ehrlich and Becker (1972)].^{2/} We consider the implications of this for models used to value risks to human health.

In particular, we find that:

- 1) Given moral hazard, when self-protection influences the probability, the severity, or both of an undesirable state, unobservable utility terms cannot be eliminated from the individual's ex ante valuation expression. Consequently, empirical studies that attribute differences across groups in ex ante value estimates solely to unobserved differences in household health production technologies are misplaced.
- 2) with moral hazard and self-protection, knowledge of the convexity or nonconvexity of physical dose-response relations is insufficient to sign unambiguously the change in an individual's ex ante marginal valuation for a reduction in the level of the hazard, even when consumer cognition is perfect. Therefore, we do not support the traditional argument that those individuals exposed to greater risk with greater income must place a higher value on a given risk reduction.
- 3) with moral hazard, an increase in the level of the environmental hazard does not necessarily lead to an increase in the level of self-protection. Therefore, self-protection expenditures are not a

consistent lower bound of the ex ante value a risk averse individual attaches to a reduction in risk.

These three statements imply that several propositions originally developed for cases of exogenous risk and which form the analytical basis for most recent empirical work on the value of health risk changes are not immediately transferable to settings where endogenous risks prevail.^{3/}

Berger, et al. (1987) appear to be among the first to consider endogenous risks in the context of human **health**.^{4/} Our treatment differs from their seminal effort in two significant ways. First, though they state the general continuous distribution case of risks to human health, they examine ex ante value only in a world of two mutually exclusive and independent states of nature: survival or death. We extend the ex ante value concept to the general continuous case. By maintaining continuity throughout, we allow the individual to choose between contractually defining states of nature or making an effort to alter states of nature. Spence and Zeckhauser (1972) demonstrate that the ability to influence states of nature enhances both the ex ante and the ex post gains from adaptation. In particular, we assume that individuals recognize that outcomes are stochastically related to actions, implying that predictions of behavior and the relative values that motivate it depend not only on preference orderings over outcomes, but also on preference orderings of lotteries over outcomes.

Second, Berger, et al. (1987) model only probability-influencing self-protection. They disregard the severity of the health outcome being risked, even though they concede that prior self-protection can influence both probability and severity. As pointed out by Ehrlich and Becker (1972) the distinction between self-protection that influences probability and self-

protection that influences severity is somewhat artificial. The distinction is often said to be made for theoretical convenience [see for example Hiebert (1983)]. In contrast, we model the effects of self-protection that influences both the probability and the severity of the undesired state, and consider the effects on the ex ante value of reduced risk.

2. THE MODEL

Consider an individual who is involuntarily exposed to a health risk under a particular liability regime. Assume the risk is created by exposure to an ambient concentration of an environmental hazard, r , taken from the real interval, R :

$$R = [\underline{r}, \bar{r}] \quad (1)$$

Because of moral hazard, the individual cannot acquire enough market insurance to avoid the risk completely. The individual must decide from a real interval, S how much self-protection, s , to undertake:

$$S = [\underline{s}, \bar{s}] \quad (2)$$

Given exposure to the hazard, the individual is uncertain as to which, i , of N alternative health outcomes will occur. Let

$$H = \{h_1, h_2, \dots, h_N\} \quad (3)$$

denote the outcome space where outcomes are the individual's human health capital returns ordered from smallest to largest, given the individual's genetic and development history.

Let $f(h_i; s, r)$ denote the probability of outcome i occurring given that self-protection, s , is undertaken and that the exposure level to the environmental hazard is r . Assume the following about $f(\cdot)$:

Assumption 1: $f(h_i; s, r) > 0$ for every $i \in [1, \dots, N]$ and every $s \in S$ and $r \in R$.

Let $F(h_i; s, r)$ denote the corresponding distribution function defined over the support $[a, b]$

$$F(h_i; s, r) = \int_a^b f(h_i; s, r) dh \quad (4)$$

where a and b are the minimum and maximum health outcomes. \leq We assume the following about $F(\cdot)$:

Assumption 2: $F(h_i; s, r)$ is twice continuously differentiable in $s \in S$ and $r \in R$ for every $i \in [1, \dots, N]$.

Assumption 3: $F_s(h_i; s, r) \leq 0$ for every $s \in S$ and $r \in R$ and every $i \in [1, \dots, N]$ in the sense of first-order stochastic dominance. \leq

Assumption 4: $F_r(h_i; s, r) \geq 0$ for every $s \in S$ and $r \in R$ and every $i \in [1, \dots, N]$ in the sense of first-order stochastic dominance.

Assumption 5: No restrictions are placed on the convexity of the distribution function in the immediate neighborhood of an optimal level of self-protection, s^* , for all $s \in S$ and $r \in R$ and for every $i \in [1, \dots, N]$.

The individual is risk averse with a von Neumann-Morgenstern utility index over wealth W , $U(W)$. The following assumptions are made about $U(W)$:

Assumption 6: U is defined over the real interval $(\bar{W}, \infty]$ where \bar{W} is 0.

Assumption 7: $\lim_{W \rightarrow \bar{W}} U(W) = -\infty$.

Assumption 8: U is strictly increasing, concave, and thrice continuously differentiable.

For each health outcome the individual might realize, he selects a minimum cost combination of medical care and foregone work and consumption. Let

$$C = C(h_i; s, r) \quad (5)$$

be his ex ante expectation of realized costs which depend on the uncertain health outcome, self-protection, and the exposure level to the hazard. Assume the following about $C(\cdot)$:

Assumption 9: C is strictly decreasing, convex, and thrice continuously differentiable in $s \in S$ for every $i \in [1, \dots, N]$ such that $C_s < 0$ and $c_{ss} > 0$ for all $h \in H$.

Assumption 10: C is strictly increasing and thrice continuously differentiable in $r \in R$ for every $i \in [1, \dots, N]$ such that $C_r > 0$. No restrictions, however, are placed on C_{rr} and C_{sr} for all $h \in H$.

Given incomplete insurance purchases, intertemporally separable utility, and constant expected prices for medical care, the individual's choice problem is then

$$\text{Max}_S \left[\int_a^b U(W - C(h; s, r) - s) dF(h; s, r) \right]. \quad (6)$$

Note that the price of self-protection has been normalized to unity. The subscript i is suppressed to maintain notational simplicity.

Given the model, we are now able to develop the propositions stated in the introduction.

3. EX ANTE VALUE AND WILLINGNESS-TO-PAY

3.1 Endogenous Risk. A few recent refinements to the willingness-to-pay approach to valuing environmental hazards have acknowledged the frequently endogenous form of the problem. For example, Rosen (1981), Berger, et al. (1987), and Viscusi, et al. (1987) note that self-protection affects survival or injury probabilities, while Shibata and Winrich (1983) and Gerking and Stanley (1986) allow self-protection to influence the severity of ex post damages. In a nonstochastic world or in an uncertain world with only two

feasible states, these studies demonstrate that marginal willingness-to-pay can be expressed solely in terms of the marginal rate of technical substitution between hazard concentrations and self-protection. This result cannot be generalized to a continuous world with endogenous risk.

Proposition 1: Given the model assumptions, when self-protection influences either the probability or the severity of health outcomes or both, the individual's marginal willingness-to-pay for reduced risk cannot be expressed solely in terms of the marginal rate of technical substitution between ambient hazard concentrations and self-protection. In particular, unobservable utility terms cannot be eliminated from expressions for the ex ante value of reduced risk. \square

Proof: To show that for a continuous distribution the individual's compensating variation statement of willingness to pay for reduced risk includes the unobservable utility terms, we examine self-protection that influences either the distribution or the severity (costs) of the health outcomes or both.

First, maximize the expected utility index (6) by selecting an optimal level of self-protection $s \in S$ yielding the following first-order condition for an interior solution

$$EU_w = -E[U_w C_s] + \int_a^b U_w C_h F_s dh. \quad (7)$$

The left-hand side of (7) represents the marginal cost of increased self-protection in terms of the utility of foregone wealth. The right-hand side reflects two types of marginal self-protection benefits: the first term is the direct utility effect of enhanced wealth resulting from reduced expected ex post costs; the second term is the indirect utility effect of a stochastically dominating change in the distribution of health outcomes.

The indirect effect was derived by integrating by parts the effect of self-protection on the distribution

$$\int_a^b U(\cdot) dF_s(\cdot) = UF_s \Big|_a^b + \int_a^b U_w C_h F_s dh$$

$$= \int_a^b U_w C_h F_s dh,$$

since $F_s(a; \cdot) = F_s(b; \cdot) = 0$. Assume that improved health outcomes will decrease the ex post costs, $C_h < 0$.

Solve for the compensating variation statement of the willingness-to-pay for reduced risk by totally differentiating the expected utility index (6), and then applying the first-order condition (7). When self-protection influences both the probability and severity of health outcomes such that $F_s < 0$ and $C_s < 0$, the willingness to pay expression is:

$$\frac{dW}{dr} = - \left[\frac{\int U_w C_h F_r dh - \int U_w C_r dF}{\int U_w C_h F_s dh - \int U_w C_s dF} \right] > 0, \quad (8)$$

where all integrals are evaluated over the support $[a, b]$. Obviously, the unobservable utility indexes cannot be removed from the individual's willingness to pay expression (8).

Even the assumption of a simple two state world fails to remove the utility terms from (8). For example, let $\pi(s, r)$ and $(1 - \pi(s, r))$ respectively represent the subjective probabilities of healthy and of sick states. Let $U_0(W - s)$ and $U_1(W - s - C(s, r))$ be the expected utility of being healthy or sick, where $U_0 > U_1$. The individual thus chooses $s \in S$ to maximize

$$EU = \pi(s, r)U_0(W - s) + (1 - \pi(s, r))U_1(W - s - C(s, r)). \quad (9)$$

Following the same steps as before, the willingness to pay expression is

$$\frac{dW}{dr} = - \left[\frac{\pi_r [U_0 - U_1] - (1 - \pi) U_1' C_r}{\pi_s [U_0 - U_1] - (1 - \pi) U_0' C_s} \right] > 0, \quad (10)$$

where $\pi_r < 0$, $\pi_s > 0$, $U_1' = \partial U_1 / \partial W$, and $U_0' = \partial U_0 / \partial W$. Again, utility terms cannot be removed.

Next allow, as do Gerking and Stanley (1986), self-protection to influence the severity, $C_s < 0$, but not the probability, $F_s = 0$, of health outcomes. Further assume that $F_r = 0$ which, with $F_s = 0$, implies that neither collective nor individual actions will influence the probability of a particular health outcome, i.e., hazard concentrations resemble sunspots or the phases of the moon. With these assumptions, expression (8) reduces to:

$$\frac{dW}{dr} = - \frac{E[U_w C_r]}{E[U_w C_s]} = - \left[\frac{EU_w EC_r - \text{cov}(U_w, C_r)}{EU_w EC_s - \text{cov}(U_w, C_s)} \right] > 0. \quad (11)$$

For the unobservable utility terms to be absent from (11), the two covariance expressions must be zero; however, our model assumptions do not allow them to be zero. Therefore the two utility terms cannot be removed.

Finally, assume, as does Rosen (1981), that self-protection affects probability, $F_s < 0$, but not severity, $C_s = 0$. In Rosen's (1981) terms, one cannot be more severely dead. For similar reasons, $C_r = 0$. Under these conditions, expression (8) reduces to:

$$\frac{dW}{dr} = - \frac{\int U_w C_h F_r dh}{\int U_w C_h F_s dh}, \quad (12)$$

and again the willingness-to-pay expression cannot be rid of the unobservable utility terms, which concludes the proof. $\bullet/\$

We could examine additional cases. For example, self-protection might influence only the probability of a health outcome, but hazard concentrations could affect probability and severity, or vice versa. The results would not change: utility terms would loom up in the willingness-to-pay expressions, implying that policy efforts to aggregate across individuals and to account

simultaneously for the reality of probability and severity unavoidably involve interpersonal utility comparisons.

3.2 Nonconvex Dose-Response Relations. Proposition 1 poses hurdles to procedures which would establish a social risk-benefit test by summing unweighted compensating or equivalent variations across individuals. Yet another problem for consistent aggregation is the ambiguous effect that a change in hazard concentrations has on the sign of compensating variation. In a contingent valuation study of the risk valuations attached to hazardous waste exposures, Smith and Desvousges (1986, 1987) report increasing marginal valuations with decreasing risk. This finding is but the latest in a 15-year long series of analytical [Starett (1972), Winrich (1981)] and empirical [Crocker (1985), Repetto (1987)] papers which use prior information on physical dose-response relations, individual abilities to process information about these relations, or individual perceptions of the relations to produce a declining marginal valuation result for more of a desirable commodity. However, when risk is endogenous, no one has yet asked whether convexity of the marginal value of risk follows when cognition is not an issue.

An individual's compensating variation can be shown to be ambiguous in sign even if the strongest possible case for negative effects of increased hazard exposure is imposed. To illustrate, define strong convexity as follows. Definition 1: Strong convexity of risk is defined as: convex ex post cost, $C_{RR} > 0$; convexity of the distribution function, $F_{RR} > 0$; and declining marginal productivity of self-protection, $C_{SR} > 0$, $C_{HR} > 0$, $C_{SH} > 0$ and $F_{SR} > 0$. Strong nonconvexity describes the conditions most favorable for the traditional argument that increased risk requires progressively increasing compensation to maintain a constant level of expected utility. Increased

exposure increases the probability and the expected ex post costs of undesirable health outcomes to the hazard at an increasing rate; moreover, the marginal productivity of self-protection is decreasing across the board.

The opposite case is strong nonconvexity. Strong nonconvexity defines the weakest case for negative effects of increased exposure to the hazard.

Definition 2: Strong nonconvexity of risk is defined as: nonconvex ex post cost, $C_{rr} < 0$; concavity of the distribution function, $F_{rr} < 0$; and increasing marginal productivity of self-protection, $C_{sr} < 0$, $C_{hr} < 0$, $C_{sh} < 0$ and $F_{sr} < 0$.

The following proposition states the result:

Proposition 2: Even in the absence of cognitive illusions or failure to consider all scarcity dimensions of the risk-taking problem, a maintained hypothesis of strong convexity of risk is insufficient to guarantee that increased exposure to a hazard requires progressively increasing compensation to maintain a constant level of expected-utility. Similarly, strong nonconvexity is insufficient to guarantee progressively decreasing compensation.

The proposition is supported by Dehez and **Drèze** (1984, p. 98) who show that the sign of the marginal willingness-to-pay for safety given an increase in the probability of death is generally ambiguous. **Drèze** (1987, p. 172) concludes that any assertions about this sign given a change in safety "...must be carefully justified in terms of underlying assumptions".

Proposition 2 contradicts the argument of Weinstein, et al. (1980) and others that individuals at greater risk must have a greater demand for safety. Consequently, contrary to Rosen (1981), individuals at greater risk with greater wealth cannot necessarily be weighted more heavily when risk reductions

are valued. Similarly, the assertions by Kahneman and Tversky (1979) and Smith and Desvousges (1987) that increasing marginal willingness-to-pay for reduced risk constitutes a lapse from rational economic behavior are not supported. ^{11/}

Proof: To demonstrate that an increase in hazard concentration has an ambiguous effect on an individual's compensating variation, differentiate the compensating variation in expression (8) with respect to the hazard exposure:

$$\begin{aligned} \frac{d(dW/dr)}{dr} = & -\frac{1}{\Omega} \left[E[U_{ww}C_r^2 - U_w C_{rr}] - 2 \int [U_{ww}C_r C_h - U_w C_{hr}] F_r dh \right. \\ & \left. + \int U_w C_h F_{rr} dh \right] \\ & + \frac{\Delta}{\Omega^2} \left[E[U_{ww}C_s C_r - U_w C_{sr}] + \int [U_w C_{hr} - U_{ww}C_h C_r] F_s dh \right. \\ & \left. + \int [U_{ww}C_s C_r - U_w C_{sr}] F_r dh + \int U_w C_h F_{sr} dh \right], \end{aligned} \tag{13}$$

where

$$\Omega = \int U_w C_h F_s dh - \int U_w C_s dF > 0,$$

$$\Delta = \int U_w C_h F_r dh - \int U_w C_r dF < 0,$$

and all integrals are evaluated over the support [a, b].

The terms on the right-hand side of (13) can be defined in terms of direct and indirect utility effects given an increase in exposure to a hazard. $\Omega > 0$ and $\Delta < 0$ represent the combined first-order direct and indirect utility effects of s and r. The first and fourth terms in (13) represent second-order direct utility effects on expected costs with an increase in exposure. Given strong convexity, the sign of the first term is negative. The sign of the fourth term is ambiguous in the sense that alternative parameterizations are conceivable in which either $U_{ww}C_s C_r$ or $U_w C_{sr}$ dominates in absolute magnitude. The second, fifth, and sixth terms are second-order direct and indirect utility effects weighted by the marginal effect on the distribution of either s or r. Given strong convexity, the signs of all three terms are ambiguous in the above

sense. Without prior information on the magnitude of the marginal effects on the expected cost function, there is no reason to expect one term to dominate. The third and seventh terms represent the second-order indirect and cross-indirect utility effects of increased exposure. By the definition of strong convexity, the sign on both terms is negative. Without knowing the relative magnitude of all the direct and indirect utility effects, however, strong convexity is insufficient to sign (13) unambiguously. Likewise, the assumption of strong nonconvexity is also insufficient to sign (13). Whether one imposes strong convexity or strong nonconvexity the sign of (13) is ambiguous. Although sufficient conditions for increasing or decreasing marginal willingness-to-pay can be determined, there is, in the absence of prior information or simple ad hoc assumptions, no reason to expect that one or two terms will dominate expression (13). This concludes the proof.

3.3 Self-Protection Expenditures as a Lower Bound. Consideration of self-protection has not been limited to problems of ex ante valuation under uncertainty. A substantial literature has emerged, e.g., Courant and Porter (1981), and Harrington and Portney (1987), which demonstrates that under perfect certainty the marginal benefit of a reduction in a health threat is equal to the savings in self-protection expenditures necessary to maintain the initial health state. This result cannot be extended to the uncertainty case when self-protection influences both ex ante probability and ex post severity. Proposition 3: Neither strong convexity nor strong nonconvexity of risk is sufficient to sign the effect of a risk change upon self-protection expenditures. Therefore these expenditures cannot be used to determine the welfare effect of a risk change.

Proposition 3 contradicts Berger et al.'s (1987) argument that if increased exposure increases the marginal productivity of self-protection,

$F_{sr} < 0$ then self-protection will increase with exposure. Consequently, Berger, et al.'s (1987 p. 975) sufficient conditions for "plausible" results do not hold when self-protection influences both probability and severity.

Proof: To demonstrate that strong convexity is insufficient to determine the effect increased hazard exposure has on self-protection, take the first-order condition in equation (7) and apply the implicit function theorem. The effect of increased exposure on self-protection is

$$\begin{aligned} \frac{ds}{dr} = & - \left[E[U_{ww}C_r(1 + C_s) - U_wC_{rs}] + \int [U_wC_{sh} - U_{ww}C_h(1 + C_s)]F_r dh \right. \\ & \left. + \int [U_wC_{hr} - U_{ww}C_rC_h]F_s dh + \int U_wC_h F_{sr} dh \right] / D \end{aligned} \quad (14)$$

where

$$\begin{aligned} D = & E[U_{ww}C_s(1 + C_s) - U_wC_{ss}] + 2 \int [U_wC_{sh} - U_{ww}C_hC_s]F_s dh \\ & - \int U_{ww}C_hF_s dh + \int U_wC_hF_{ss} dh < 0 \end{aligned} \quad (15)$$

and all integrals are evaluated over

sufficient condition of the maximization problem (6), and is assumed to hold whenever (7) holds.

Given $D < 0$, the sign of (14) depends on the sign of its right-hand-side numerator. The first term in the numerator of (14) is the direct utility effect of increased exposure on expected costs. Given strong convexity of risk and $(1 + C_s) > 0$ from the first-order condition, the sign of the first term is negative. The second term reflects the indirect utility effect of increased exposure on the distribution. Given strong convexity, its sign is ambiguous in the earlier defined parameterization sense. The third term is a direct utility effect weighted by the marginal effect of self-protection on the distribution ($F_s < 0$), and its sign is also ambiguous. The signs for the second and third effect are ambiguous since there is no a priori reason to believe that any one set of terms dominates the others. The fourth term in the numerator is the

cross-indirect utility effect of increased exposure. Given strong convexity, its sign is negative. Therefore, without prior information on the relative magnitudes of the four direct and indirect utility effects, strong convexity is insufficient to sign (14) unambiguously. Given the conditions most favorable to the traditional argument that increased risk will increase self-protection, we still require prior information on the impact that increased exposure has on the marginal productivity of self-protection to support the argument.

Following the logic above, an assumption of strong nonconvexity of risk leads to a similar conclusion of an ambiguous effect of increased exposure on self-protection. Consequently, since self-protection may decrease as exposure to a hazard increases, self-protection cannot be considered a consistent lower bound on the ex ante value a risk averse individual attaches to a reduction in risk. This concludes the proof.

4. CONCLUSIONS AND IMPLICATIONS

Individuals and policymakers use self-protection activities to influence both their ex ante risks and their expected ex post consequences. The implications of this for models used to value risks to human health are unequivocally negative. We show that unobservable utility terms cannot be eliminated from marginal willingness-to-pay expressions, implying that empirical efforts which identify marginal rates of substitution with willingness-to-pay are misdirected. We also show that even under the most favorable restrictions increased risk need not imply progressively increasing levels of compensation in order to restore initial utility levels. Consequently the traditional argument that those who are exposed to greater risk and have greater wealth must value a given risk reduction more highly does not follow. Finally, we demonstrate that increased risk need not imply

increased self-protection expenditures; thus changes in these expenditures may not bound the value of a risk change.

Some succor for health risk valuation efforts could be obtained by stepping outside professional boundaries to draw upon prior information from psychology, biomedicine, and other disciplines. Insight might therefore be gained into the signs and the relative magnitudes of many terms in expressions (13) and (14). It is odd that the field of economics which explicitly recognizes the policy relevance of incomplete markets has historically been reluctant to use information from other disciplines in order to simulate the valuation results of a complete market. We recognize that there is a growing trend to incorporate restrictions drawn from other disciplines into the behavioral postulates of economic models. ¹² The results of this paper suggest that the incorporation process should be accelerated.

Incorporation will not overcome, however, the aggregation problems posed by the presence of utility terms in individuals' willingness-to-pay expressions. Approaches to aggregate risk-benefit analysis do exist other than the mechanical summation of consumer surpluses calculated from the singular value judgement that social welfare and aggregate total income are synonymous. Given that individual consumer surpluses can be estimated, one possibility is to draw upon the extensive equivalence scale literature, e.g., Deaton and Muellbauer (1986), in order to weight each individual or household. Tradeoffs can then be evaluated using an explicit social welfare function which recognizes that personal health is in part self-produced and inalienable. Alternatively, utilities might be calculated directly.

FOOTNOTES

1. Moral hazard refers to the tendency of insurance to influence an individual's incentive to prevent loss.
2. Self-protection includes everything from installing home water filters in order to reduce pollutant concentrations in drinking water to medical care and the use of tort law. [See Laffont (1980), Crocker (1984)].
3. The empirical human health valuation literature typically assumes that health risks are: (i) independent of individual actions; and (ii) usually for the sake of analytical and empirical tractability, individuals require progressively increasing levels of compensation to maintain constant expected utility when confronted by increasing risk. Jones-Lee, et al. (1985), for example, embodies both conditions. We argue these assumptions are unnecessarily restrictive in the sense that they stretch the ability of economic analysis to cover the domain of risky phenomena.
4. Psychologists agree that individuals perceive that they have substantial control over uncertain events [Perlmutter and Monty (1979)]. Stallen and Tomas (1984) conclude that "... the individual is not so much concerned with estimating uncertain parameters of a physical or material system as he is with estimating the uncertainty involved in his exposure to the threatening event and in opportunities to influence or control his exposure" [emphasis added].
5. The [a, b] interval could also be influenced in subsequent periods by self-protection. We disregard this issue.
6. Subscripts represent partial derivatives.
7. Assumptions of a risk-neutral individual with an identity map of ex post costs would eliminate the unobservable utility expressions. These assumptions seem excessively restrictive.
8. One might eliminate the utility terms by using the pointwise optimization technique that Mirrlees (1974) and **Holmström** (1979) employ. However, pointwise optimization evaluates self-protecting choices individually at each and every health state rather than in terms of lotteries over health states. It thus adopts an ex post rather than an ex ante perspective.
9. See Polemarchakis, et al. (1986) for thinking on aggregation under exogenous risk.
10. Rogerson (1985) assumes that the distribution function must generally satisfy the convexity of the distribution function condition (CDFC). Therefore, the assumption of a concave distribution in r and s is perhaps restrictive. As shown by Jewitt (1988), however, the CDFC assumption is not universally required in that it satisfies very few of the standard distributions set forth in statistics textbooks.
11. Close inspection of the questionnaire formats upon which these assertions are based reveals that respondent opportunities to influence risk and/or severity were not fully controlled.
12. See **Wärneryd** (1986), Weinstein and Quinn (1983) and Smith and Johnson (1988), for example.

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THE ECONOMICS OF QUARANTINES: AN APPLICATION TO PESTICIDE REGULATION

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THE ECONOMICS OF QUARANTINES: AN APPLICATION TO PESTICIDE REGULATION

One of the most common practices for dealing with hazardous situations is simply to remove the hazard from human proximity, either spatially or temporally. Such policies can be termed quarantines. The classic case is that of contagious disease control, where infected individuals are kept apart from vulnerable individuals until the threat of contagion has passed. Other examples include imprisoning dangerous criminals; locating hazardous industries (e.g., military testing grounds, nuclear power plants and other hazardous activities) in remote areas; keeping dangerous chemicals, high voltage equipment, etc. in locked or otherwise inaccessible locations; and keeping workers out of areas recently treated with pesticides.

Any quarantine involves tradeoffs that must be evaluated whether the decision maker is a government agency or an individual concerned with self-protection from self-generated hazards. The benefits of quarantines obviously consist of reductions in hazard. But quarantines typically have costs as well, such as additional discomforts and lost wages of contagious patients or productivity losses from suboptimal siting or scheduling. These tradeoffs must be evaluated in determining the appropriate parameters of a quarantine, that is, the length of time and/or location restriction. This paper develops a framework for optimal quarantine determination and applies it to a widespread form of quarantine, re-entry regulation of pesticide-treated fields. Section I contains a model of optimal quarantine determination. Section II models optimal timing of pesticide application under re-entry regulation. Interestingly, the imposition of re-entry regulation may make it optimal for farmers to switch to

prophylactic treatment of pests, a practice which has been widely criticized as inefficient in the literature on pesticide use. Section III applies this model to the case of pre-harvest intervals in apple production in three major producing states. Section IV develops a model of acute poisoning from exposure to pesticide residues under different re-entry intervals. Section V combines the production and health models into a tradeoff model which is then used to obtain a rough evaluation of current policy.

I. Optimal Quarantine Determination

Generally speaking, quarantine have both a spatial and a temporal dimension: how far away the hazard is sited and how long the quarantine lasts. Contagious disease quarantines have both: one must decide where to locate infectious patients relative to other patients and the general populations well as how long to continue isolation. Penal policy also does: prison location and length of sentence will both depend on how dangerous a criminal is. In other cases, one of these dimensions may be irrelevant. In pesticide regulation, for example, only the temporal dimension may matter: many pesticide residues are absorbed by touch and therefore the hazard affects only those entering a treated field. In siting of military testing grounds, nuclear power plants or other hazardous facilities, on the other hand, only location matters.

Let D represent the spatial dimension of the quarantine and T the temporal dimension. Let Z represent a consumption or production activity affectedly the quarantine. The benefits of consumption or production, $B(Z,D,T)$, depend on Z and on the quarantine parameters D and T , as does the level of hazard, $H(Z,D,T)$. Let $W[B(Z,D,T), H(Z,D,T)]$ denote the utility function of an individual facing a hazardous situation or a social welfare function. The relevant decision

problem is to choose Z , D and T to maximize utility or social welfare. This is typically accomplished in two stages. First, microeconomic theory is used to derive a model of optimal consumptive or productive behavior conditional on the quarantine parameters D and T . The resulting behavioral model is subsequently used to derive the optimal policy parameters.

Formally, letting subscripts denote derivatives, the necessary conditions are

$$(1a) \quad W_B B_Z + W_H H_Z = 0$$

$$(1b) \quad W_B B_D + W_H H_D = 0$$

$$(1c) \quad W_B B_T + W_H H_T = 0.$$

The two-stage procedure described above consists of first solving equation (1a) to get the optimal level of consumption/production activity contingent on the quarantine, $Z^*(D,T)$, and then choosing D and T to maximize $W[B(Z^*(D,T),D,T), H(Z^*(D,T),D,T)]$ according to the necessary conditions

$$(2a) \quad W_B (B_Z Z_D + B_D) + W_H (H_Z Z_D + H_D) = 0$$

$$(2b) \quad W_B (B_Z Z_T + B_T) + W_H (H_Z Z_T + H_T) = 0.$$

The case of pesticide regulation considered below is investigated by first deriving profit-maximizing pesticide use patterns conditional on temporal quarantine restrictions, $Z^*(T)$, and farm profits, $B(Z^*(T))$. The risk of acute organophosphate poisoning of farm workers is modeled as a function of pesticide use, $H(Z^*(T))$. These two components are combined into a tradeoff curve under an assumption of equal welfare weights on farm income, $B(Z^*(T))$, and worker safety, $H(Z^*(T))$, that is $W_B = W_H$. Finally, this tradeoff curve is used to derive the optimal length of the quarantine T^* under different environmental conditions.

One can conceptualize distance-related quarantine problems in the same way.

For example, the size, operating procedures and transmission line requirements of a nuclear power plant may depend on the distance between it and the population and industrial centers it serves, so that one would begin with a relationship between these factors and quarantine distance, $Z^*(D)$: The risks posed by the plant, $H(Z^*(D), D)$ depend on the quarantine distance D and the operating characteristics of the plant, $Z^*(D)$. These two can be combined using the appropriate welfare weights W_B and W_H to obtain a tradeoff relation that can then be used to determine the optimal distance D^* .

In sum, even in regulatory contexts it is typically necessary to solve private optimization problems prior to considering the social decision problem, since the private optimization problems are crucial elements of the tradeoff relations needed. Moreover, close interdisciplinary cooperation is often required to specify the hazard functions H , since they depend in complex ways on combined economic, environmental and biomedical factors.

II. Crop Production Under Re-Entry Regulation

One of the most common measures used to protect farm workers and other rural inhabitants from the health hazards posed by applied pesticides is to forbid entry into treated fields for a specified period of time during which pesticide residue levels (and hence health risks) are thought to be excessive. Similar regulations aim to protect consumers as well by forbidding harvest for a specified interval after application of pesticides. Often, these re-entry regulations lead to reductions in growers' incomes by preventing optimal scheduling of harvest or intraseasonal activities like pruning or irrigation, causing decreases in yield, quality or price received for the crop. Thus, whether the decision maker is a government agency charged with protecting farm

workers or a farmer deciding whether to work in his/her own field, the determination of an appropriate re-entry interval hinges on the choice of a tradeoff between risks to human health and safety, on the one hand, and the economic losses induced by regulation on the other.

For the sake of simplicity, we concentrate on the problem of re-entry regulations affecting an individual farmer's harvest of a perishable crop (fruits, vegetables), the kind of crop to which this form of regulation is applied most often. We assume that benefits B are restricted to farm profits, which are a function of pesticide use Z , itself a function of the re-entry interval T . We assume also that the farmer applies the pesticide at a standard application rate and focus on the determination of the timing of the application.

Assume that there is a time τ_0 representing the earliest date at which the crop can be harvested; prior to τ_0 , the crop will be immature and hence not harvestable. Assume also that after τ_0 , the value of the crop declines because of decreased quality or because of price decreases due to seasonal increases in aggregate production, so that the farmer's revenue is maximized by harvesting at t_0 . Formally, this implies a revenue function $R(t)$ such that $R(\tau_0) = \max (R(t)) = R^*$, and, letting subscripts denote derivatives, $R_t < 0$ and $R_{tt} \leq 0$ for $t > \tau_0$. Production costs, including pesticide materials and application costs, will be assumed to be constant and will thus be ignored.

Now assume that a pest appears at a time τ_a shortly prior to the optimal harvest time t_0 . If left untreated, the pest will damage a proportion of the crop which will then be unsalable. The larger the pest population is, the greater the level of damage will be. This damage can be avoided by treating the crop with a pesticide. To simplify matters, assume that only a single standard treatment is available at a negligible cost. If the farmer treats the crop

immediately upon arrival of the pest, i.e. , chooses a treatment time $t_s - t_a$, the pest will be effectively eradicated and damage will be essentially reduced to zero. If, on the other hand, the farmer treats the crop before the pest arrives ($t_s < t_a$), the pesticide will decay; its effectiveness will be reduced by the time the pest arrives and the farmer will sustain some crop losses. The longer is the interval between treatment and the arrival of the pest, the greater will be the decay of the pesticide and the damage caused by the pest.

These characteristics can be represented formally by letting the proportion of the crop damaged by a pest population of size k be a function $g(k, t_a - t_s)$, where $t_a - t_s$ represents the time elapsed between treatment and the arrival of the pest. The preceding discussion suggests that $g_k > 0$, $g_t > 0$ and $g(k, 0) = 0$. Pesticide decay curves are typically convex, so that one would expect $g_{tt} \geq 0$ as well.

There are two types of treatment strategies available to **farmers**: a reactive strategy of applying pesticides upon the arrival of the pest, and a prophylactic or preventive strategy of applying pesticides in anticipation of a pest problem. The reactive pest management strategy will maximize profits whenever it is feasible, which implies an optimal choice of $t_s - t_a$ whenever $T \leq t_0 - t_a$. If the re-entry period T is sufficiently long, however (specifically $T > t_0 - t_a$), following the reactive treatment plan may force the farmer to delay the harvest and thereby lose revenue. In this case the farmer faces a tradeoff between losing revenue from crop damage and losing revenue from harvesting delays. Under some conditions, it may become optimal for the farmer to adopt a prophylactic treatment strategy. While this practice has been much maligned in the pest management literature, rigidities in scheduling such as those imposed by re-entry regulation may make it desirable for farmers.

Some casual empirical evidence supports the notion that re-entry intervals actually provide a motivation for prophylactic treatment strategies. In Oregon, plum growers expecting to need to use parathion for end-of-season codling moth control typically apply the chemical 14 days -- the length of the pre-harvest interval -- prior to the projected harvest date, regardless of whether the pest is in evidence.

It should be clear that the farmer will never treat any earlier than needed to be able to harvest at time τ_0 , i.e., that $\tau_s \geq \tau_0 - T$; treating any earlier than $\tau_0 - T$ would imply accepting greater damage in return for no gain in revenue and is thus less profitable than treating at $\tau_0 - T$. It should also be evident that the farmer will always harvest the crop as soon as possible, that is, at least as soon as the re-entry period has ended. If the re-entry constraint is non-binding, then the harvest time will be τ_0 . If the re-entry constraint is binding, then the harvest will occur T periods after the treatment time; normalized (without loss of generality) to fit the revenue curve R . This can be written $\tau_s + T - \tau_0$.

The pesticide use patterns adopted and revenues earned by the farmer thus depend critically on whether or not the re-entry interval constitutes a binding constraint. If it does not, then a reactive treatment strategy is always optimal, $\tau_s = \tau_a$, the crop will be harvested at τ_0 and revenue will be R^* . If it does, the farmer will face a tradeoff between crop damage and decreased revenue. The optimal pest management strategy will be determined by the choice of a treatment time τ_s which maximizes realized revenue, given by:

$$(3) [1 - g(k, \tau_a - \tau_s)]R(\tau_s + T - \tau_0)$$

subject to the constraint:

$$(4) \tau_0 - T \leq \tau_s \leq \tau_a.$$

Because the convexity of the pesticide decay function makes the damage function $g(k, \tau_a - \tau_s)$ convex, the realized revenue function (3) will be convex unless R is quite strongly concave. Thus, the optimal treatment plan must be analyzed according to two cases.

Case 1: The most likely case is that realized revenue (3) will be convex, so that the optimal treatment time will be either the maximum or minimum possible time, that is, either τ_a or $\tau_0 - T$. In essence, of course, this constitutes a choice between reactive ($\tau_s = \tau_a$) and prophylactic ($\tau_s = \tau_0 - T$) treatments. The farmer will choose the one which gives the greatest profit. If $\tau_s = \tau_a$, there will be no damage ($g = 0$) but the farmer will have to wait until $\tau_s + T - \tau_0$ to harvest and will thus realize a revenue of $R(\tau_a + T - \tau_0)$. If $\tau_s = \tau_0 - T$, there will be damage $g(k, \tau_a + T - \tau_0)$; the farmer will harvest at τ_0 and thus realize a revenue $[1 - g(k, \tau_a + T - \tau_0)]R^*$. If the difference between these two realized revenues,

$$(5) V = R(\tau_a + T - \tau_0) - [1 - g(k, \tau_a + T - \tau_0)]R^*$$

is positive, the farmer will adopt the reactive strategy and treat at τ_a . If it is negative, the farmer will adopt the prophylactic strategy and treat at $\tau_0 - T$. An increase in the size of the pest population k will increase V and thereby make the farmer more likely to adopt a reactive strategy. An increase in the re-entry interval T , though, will increase V only if the marginal increase in the proportion of the crop damaged by treating earlier (g_t) is less than the marginal increase in the proportion of revenue lost by treating later (R_t/R^*). Thus, if $g_t > R_t/R^*$, an increase in T will make the farmer more likely to adopt

a prophylactic strategy. An increase in the interval between the arrival of the pest and the optimal harvest date, that is, in $t_0 - t_a$, will, of course, have precisely the opposite effect of an increase in the re-entry interval T .

Case 2: If the revenue function $R(\cdot)$ is sufficiently concave to make realized revenue (3) concave, the profit-maximization problem will have an interior solution defined by:

$$(6) \quad g_t R + (1 - g)R_t = 0$$

with sufficiency assured by:

$$(7) \quad Q - g_{tt} R + (1 - g)R_{tt} \leq 0$$

which holds by assumption. It is readily apparent that an increase in the re-entry interval will lead the farmer to treat earlier ($dt_s/dT = -[R_t g_t + (1 - g)R_{tt}]/Q < 0$), thereby accentuating the tendency toward prophylactic treatment. If, as one would expect, the increase in damage from treating earlier is greater for larger pest populations than for smaller ones (i.e., $g_{tk} \geq 0$), an increase in the pest population size will induce the farmer to treat later ($dt_s/dk = -[g_{tk} R - g_k R_t]/Q > 0$), thereby reducing the tendency toward prophylactic treatment. As before, an increase in $t_0 - t_a$ will have the opposite effect of an increase in T .

III. Pesticide Use in Apple Production

Consider the case of re-entry regulation of organophosphate insecticides used to protect apple crops from infestations of codling moth larvae from moth flights shortly prior to harvest. The yield and quality of the apples is assumed to increase up until the maturity date t_0 , which is the earliest date at which

the crops may be harvested. After t_0 , yield and quality will remain constant for a considerable length of time. However, the price the farmer receives for the crop will decline as time passes because the aggregate supply of apples will increase as producers in other regions harvest and market their crops. This price decline will continue until the price of apples for fresh consumption equals the price for processing uses, at which point the price will remain constant. An analysis of the intraseasonal trends in farm-level apple prices in three major producing states (Washington, Michigan, California) indicated that this price decline is convex and could be represented well by an exponential curve. Thus, the price received by a grower harvesting a full crop at time $t \geq t_0$ is $R \cdot \exp(-a(t - t_0))$.

The threat posed by a late-season flight of codling moths consists of an infestation of larvae in the fruit, i.e., of wormy apples. This threat can be alleviated by using organophosphates to kill the moths before they lay eggs. "Standard doses of these pesticides are typically applied; without loss of generality, normalize this standard dose to unity. Pesticide decay rates are typically modeled as exponential curves, so that the proportion of the pest population killed by a treatment applied at t_s is $\exp(-b(t_a - t_s))$ and the proportion surviving is $1 - \exp(-b(t_a - t_s))$. Assume that all infested fruit is unsalable and that the proportion of the crop damaged is proportional to survivorship. Letting k represent the proportion of the crop damaged by a moth population of standard size, the damage function $g(k, t_a - t_s)$ will be in this case $k[1 - \exp(-b(t_a - t_s))]$.

The realized revenue function (3) in this case will thus be:

$$(8) \quad R = R \cdot \exp(-a(t_s + T - t_0)) (1 - k[1 - \exp(-b(t_a - t_s))])$$

which is obviously convex. The difference in profit between treating at τ_a and treating at τ_0 is thus

$$(7) \quad V = R \cdot \exp(-a(\tau_a + T - \tau_0)) - R \cdot (1 - k[1 - \exp(-(\tau_a + T - \tau_0))]).$$

which will be positive whenever

$$k > [1 - \exp(-a(\tau_a + T - \tau_0))] / [1 - \exp(-b(\tau_a + T - \tau_0))] = k_c$$

and negative whenever $k < k_c$. The optimal treatment strategy is thus:

$$(9) \quad \tau_s = \begin{cases} \tau_a, & k > k_c \\ \tau_0 - \tau, & k < k_c \end{cases}$$

In addition to the comparative static results from the general case it is straightforward to show that the faster the price declines over the season, the more likely the farmer is to adopt a prophylactic strategy ($dV/da < 0$) and that the faster the pesticide decays, the more likely the farmer is to adopt a reactive strategy ($dV/db > 0$).

To provide an empirical mechanism for evaluating the impact of re-entry regulation of pre-harvest use of parathion on apples in three main U.S. producing states (Washington, California, Michigan), the model was parameterized as follows. A regression of weekly data on farm-level prices received in Washington, California and Michigan over the period 1971-1980 on a time trend and dummies to control for differences among years and states yielded an estimate of the revenue decay parameter $a = 0.0024$. According to Johannes Joost, California extension specialist on apples, the maximum price received in 1984 was about \$300/ton, which, at a yield of 10 tons/acre, suggests a maximum revenue of \$150,000 for a 50-acre block. The regression analysis suggested that price

levels in Michigan and Washington were about 17 percent and 32 percent above that of California; however, because Michigan harvests about 4 weeks after California and Washington, 2 weeks, the maximum price in these states should be 9.8 percent and 28.2 percent higher than California, respectively, giving estimates of about \$165,000 per 50-acre block in Michigan and \$192,000 per 50-acre block in Washington. An estimate of the parathion decay parameter $b = 0.8$ was taken from Spear et al. 's (1975a) study. of parathion decay in California citrus orchards; examination of parathion decay data on Washington apples (Staiff et al. (1975)) indicated that the decay patterns in the two cases were essentially identical. Conversations with farm advisors indicated that, if left untreated, a codling moth infestation caused by a population of normal size would damage about 10 percent of the crop; thus, k was given a value of 0.10. Calculation of the damage threshold for prophylactic spraying over the range of reasonable re-entry periods, k_c , resulted in values ranging from .009 to .065, all well below k ; thus, it appears that reactive treatment will always be optimal. In fact, apple prices would have to fall 2-10 times more rapidly before prophylactic treatment would become desirable.

IV. Residue Poisoning From Parathion Exposure Among Apple Harvesters

The risk of clinical illness in workers as a result of exposure to residues of parathion applied to apples at various locations was modelled according to the overall scheme laid out by Pependorf and Leffingwell (1982). In essence, the pesticide is applied, a decay process takes place in which some of the parathion is converted to the oxygen analog, paraoxon, and exposure takes place days or weeks later when crews enter the field to harvest the crop. If clinical illness results, it is usually due to a dermally absorbed dose of paraoxon.

There is considerable information available to quantify the various steps in this process but very limited data on climatological effects on the decay process itself.

The characterization of the residue decay process follows that of Spear et al. (1975a) and Popendorf and Leffingwell (1978). In both cases, the dislodgeable foliar residues of parathion and paraoxon are described by linear ordinary differential equations. The parameterization of these models utilized data obtained from citrus crops, but limited data on apples suggests a similar decay pattern (Staiff et al. (1975)). The simplified form of the model used here describes the residue relevant to worker hazard from day three post-application onwards. After day three the parathion residue has decayed to the point where the hazard to workers depends almost entirely on the paraoxon residue (Spear (1975b)).

The form of the model is:

$$(10a) \quad dx/dt = -bx$$

$$(10b) \quad dr/dt = cx - qr$$

where parathion residue is denoted by x and the paraoxon residue by r . The units are in ng/cm^2 . The solution to this set of equations is:

$$(11a) \quad x(t) = x_0 \exp(-bt)$$

$$(11b) \quad r(t) = (cx_0/b + q) [\exp(-qt) - \exp(-bt)]$$

where t is the time post-application in days.

There are, then, four parameters required to solve for $r(t)$, the paraoxon residue, b , c , q , and the initial condition x_0 . The first three parameters are weather dependent whereas the last depends on the application rates and pre-existing levels of foliar dust on the trees. Nigg et al. (1978) have studied the effect of weather variables on the parathion decay process and have concluded that rainfall and leaf wetness from other sources are the primary determinants of the rate of residue disappearance after the period immediately post application. Hence, climatological variability was modeled by assuming that the decay parameters, b , c , and q , are the same for all three regions but that the paraoxon residue is diminished as an exponential function of the cumulative rainfall during the decay period. Under these assumptions the rainfall-modified paraoxon residue at entry time T is given by:

$$(12) \quad r'(T) = r(T) \exp(-.291CR)$$

where CR is the cumulative rainfall during the period $(0,T)$. A one inch rainfall leads to a diminution of the residue by 25 percent and a two inch rainfall a 44 percent decline. These predictions are more or less consistent with the data presented by Gunther et al. (1977).

Estimates of the parameters b , c and d are available from Popendorf and Leffingwell (1978). Also, the initial condition, x_0 was estimated from their data by regressing their parameter a_0 against the applied amount in pounds of active ingredient per acre (AIA). The resulting expression is:

$$(13) \quad x_0 = 1690(AIA)^{.3067} \text{ ng/cm}^2$$

The values used for the other parameters are $b = 0.8$, $c = 0.08$ and $q = 0.05$.

Following the procedure detailed by Popendorf and Leffingwell (1982) the

dermal dose in mg/kg is related to the paraoxon residue by the expression $k_d r'(\tau) \tau_e$, where τ_e is the exposure time in hours and k_d a constant determined empirically and set equal to 9.0 as observed in citrus crops. The exposure time is taken to be an eight hour shift. For a single organophosphate the relation between dermal dose and fractional inhibition of red blood cell cholinesterase (RBCD) is given by:

$$(14) \quad \text{RBCD} = 1 - \exp\{-w_e D / \text{LD}_{50}\}$$

where, for paraoxon, the dermal LD_{50} is 1.0 and w_e equals to 6.0, midway in the reported range of 4.7 to 7.3. All members of a work crew are assumed to be exposed to the same residue environment which is further assumed to result in the same cholinesterase depression. Individual variability is modeled only in the relationship between cholinesterase depression and clinical illness.

The relationship between cholinesterase depression and clinical signs and symptoms of poisoning was modeled by assuming the probability of illness depended on the degree of cholinesterase depression according to the expression:

$$(15) \quad P = 1 / [1 + \exp(w_1 + w_2 \text{RBCD})]$$

where w_1 and w_2 were based on clinical experience and values reported in the medical literature (Midtling et al. (1985), Milby (1988)). Two sets of parameters were used, one relating to mild illness and the other to severe illness. The probability of illness relates to each member of the crew at the end of one eight-hour day and not to exposures cumulated over several days.

V. Profit-Health Tradeoffs in Re-Entry Regulation

The models presented in the two preceding sections can be used to evaluate

the impact of re-entry regulations on apple growers' revenues and apple harvesters' safety. The analysis was conducted under the assumptions that a flight of codling moths arrives four days before the optimal harvest date t_0 (i.e., $t_0 - t_a - 4$), that parathion is applied at a rate of 2.0 pounds of active ingredient per acre, and that, as is typical, the crop produced on a 50-acre block will be harvested in one day by a crew of 500 (10 workers per acre). Losses in growers' revenues were compared to the risk of severe and mild poisoning to each individual worker. Rainfall levels of 0, 0.5, 1, 1.5, and 2 inches during the re-entry period were used to take into account the differences in weather conditions encountered in the different regions under investigation: California receives virtually no rainfall during the harvest period, Washington receives an average of 0.5 inches and Michigan receives an average of 1.5 inches under normal conditions.

Table 1 shows the expected numbers of severe and mild parathion poisoning cases under California, Washington and Michigan conditions, plus the fraction of revenue lost due to harvest delays. The risk of poisoning is clearly non-negligible: With a pre-harvest interval of four days or less, there will be an average of 2.5 severe cases and 43 mild cases under California conditions, 1.6 severe and 29 mild cases under Washington conditions and 0.8 severe and 15 mild cases under Michigan conditions. (At any given time, there will be almost 19 times as many mild as severe cases.) Each additional day entry is prohibited reduces the number of mild and severe cases by about 13 percent, while each additional inch of rainfall reduces them by about 75 percent. Even so, the risk of poisoning remains non-negligible for a relatively lengthy period of time: If re-entry is prohibited for as much as 2 weeks, there will still be an average of one severe poisoning incident for roughly every 2 50-acre blocks harvested

in California, one severe incident for every 3 50-acre blocks harvested in Washington and one severe incident for every 4 50-acre blocks harvested in Michigan.

At the same time, the losses imposed by re-entry regulation can be considerable. Each additional day's delay in harvesting reduces total revenue by about 0.24 percent, corresponding to \$360 per 50-acre block in California, \$460 per 50-acre block in Washington and \$395 per 50-acre block in Michigan. By way of contrast, total harvesting labor costs amount to about \$425 per 50-acre block in Washington (Hinman, Tukey and Hunter). A pre-harvest interval of 2 weeks would result in a revenue loss on the order of 2.5 percent; since profit margins in Washington apple production range from 3 to 10 percent (Hinman, Tukey and Hunter), such a loss would represent a sizable fraction of net income.

The optimal pre-harvest interval in each state (assuming equal social welfare weights on farmers' incomes and workers' health) is determined by equating the marginal cost of additional harvest delays in terms of revenue lost with the marginal benefits associated with reductions in the number of poisoning incidents. For illustrative purposes, we calculated these optimal pre-harvest intervals under the conservative assumptions that benefits were restricted to average avoided costs, that is, to the average costs of hospitalization plus average lost wages. This ignores long-term losses due to chronic neurotoxic effects, the value of the disutility of suffering poisoning, losses caused by additional risks to consumers from residues remaining at the time of ingestion and so on.

A typical severe parathion poisoning case typically requires 3 days of hospitalization, with the first day spent in intensive care, followed by two weeks of recovery, i.e., lost work time. Assuming average costs of \$1200 per

day for intensive care and \$500 per day for a standard hospital bed implies total hospitalization costs of \$2200. Assuming an average wage of \$10 per hour for an 8-hour day implies total lost wages of \$800, for a total cost of \$3000 per severe case (Becker (1988)).

A typical mild case requires no hospitalization; medical care will typically cost about \$40 per case and there will generally be 2 days of lost work time, for a total cost of \$200 per case (Becker (1988)).

Figures 1, 2 and 3 show the respective marginal costs and marginal benefits from severe and all poisoning cases associated with different pre-harvest intervals in California, Washington and Michigan. The optimal pre-harvest intervals are 15 days in California, 12 days in Washington and 9 days in Michigan. Current EPA regulations require 14 days regardless of rainfall conditions for applications of parathion on apples such as the one considered here. Interestingly, the current pre-harvest interval is quite close to the optimal levels calculated here, although our calculations suggest the desirability of greater conservatism under California conditions and less conservatism under Michigan conditions. They also suggest that, as long as local rainfall can be monitored effectively, the same levels of safety implicit in the 14-day pre-harvest interval can be achieved at lower cost by making the pre-harvest interval dependent on rainfall. For example, lowering the pre-harvest interval from 14 to 9 days when there have been 2 inches of rain would cut the losses suffered by Michigan apple growers by \$1944 per 50-acre block, almost 50 percent, while lowering it from 14 days to 12 days when there have been 0.5 inches of rain would cut the losses suffered by Washington growers by \$904 per 50-acre block, almost 20 percent.

VI. Conclusions

Public authorities frequently use quarantines to ensure public safety by removing people from hazardous situations either in time or space. Individuals may pursue similar strategies to enhance their own safety in dealing with hazards. This paper develops a methodology for assessing the tradeoffs between productivity or utility losses from this type of regulation and reductions in risk of disease, accident or illness and applies it to the case of re-entry regulation in pesticides. We show that this form of regulation provides a rational incentive for prophylactic applications of pesticides, a practice that has been much maligned in the pesticide literature. In an empirical evaluation of pre-harvest intervals for parathion used on apples, we demonstrate that the tradeoffs involved are quite substantial, that the optimal pre-harvest intervals implied by rather conservative benefits estimates are quite close to those actually set by the Environmental Protection Agency, and that the same level of worker safety as that implicitly targeted by EPA can be achieved at lower cost by making pre-harvest intervals dependent on rainfall.

In order to focus on the main issues in deriving tradeoffs from quarantine parameter choices, the model used here is partial and rather stylized. Obvious improvements include incorporating considerations such as: pest population dynamics and intraseasonal effects; general equilibrium effects of re-entry regulation on prices and the distribution of production; choice of amounts of pesticides and harvest crew size as well as time of application; the influence of stochastic factors such as weather and size and time of arrival of pest populations; and uncertainties about residue decay, dermal absorption, cholinesterase depression and clinical response. The results we obtain, however, strongly suggest that more elaborate modeling of re-entry regulation and other

forms of quarantine is well worthwhile.

Further research along these lines is especially necessary because environmental and occupational health problems such as the one addressed here are a growing policy concern. While policy advice has been monopolized by natural scientists until recently, recognition of the fact that absolute safety is often unattainable has led to an appreciation of the importance of evaluating tradeoffs between enhanced safety and other social goals. A key problem is that thorough tradeoff assessments require close interdisciplinary cooperation in modeling a full spectrum of economic, physical and biological processes beginning with production and terminating in risks to **health**.¹ While the difficulties of organizing such interdisciplinary cooperation have meant that this sort of modeling has been performed only seldom in the past, hopefully the work reported here will demonstrate the feasibility and importance of pursuing it.

VII . Footnotes

¹ While economists have studied the links between pollution and health (as in the voluminous literature on air pollution and health initiated by Lave and Seskin) and between production and pollution (see for example, Anderson, Opaluch and Sullivan), to our knowledge none have modeled the entire path from production to pollution to health.