

US EPA ARCHIVE DOCUMENT

# APPENDIX I

BENEFITS ANALYSIS  
SUPPORTING INFORMATION

## **I.1 Particulate Matter Health and Welfare Effects Estimation**

### **I.1.1 Table I.1 PM Health and Welfare Effects Estimation (also used for RH analysis)**

**Table I.1 PM Health and Welfare Effects Estimation (also used for RH analysis)**

Endpoint	Concentration-Response Function		PM Averaging Time		Population <sup>a</sup>	Annual Baseline Incidence (per 100,000 of indicated population) <sup>b</sup>	Pollutant Coefficient <sup>c</sup>
	Source	Functional Form	Studied	Applied			
<b>Mortality</b>							
Mortality (long-term exposure)	Pope et al., 1995	log-linear	annual median	annual median <sup>d</sup>	ages 30+	759 (nonaccidental deaths in general pop.)	0.006408
Mortality (short-term exposure) using PM10 indicator	Schwartz et al., 1996a (Boston, Knoxville, St. Louis, Steubenville, Portage & Topeka)	log-linear	2-day average	1-day average <sup>e</sup>	all	803 (nonaccidental deaths in general pop.)	0.001433
Mortality (short-term exposure) using PM2.5 indicator	Ito & Thurston, 1996 (Chicago)	log-linear	2-day average	1-day average <sup>e</sup>	all	803 (nonaccidental deaths in general pop.)	0.000782
	Kinney et al., 1995 (Los Angeles)	log-linear	1-day average		all		
	Pope et al., 1992 (Utah)	log-linear	5-day average		all		
	Schwartz, 1993 (Birmingham)	log-linear	3-day average		all		
	Schwartz et al., 1996a (Boston)	log-linear	2-day average		all		
	Schwartz et al., 1996a (Knoxville)	log-linear	2-day average		all		
	Schwartz et al., 1996a (St. Louis)	log-linear	2-day average		all		
	Schwartz et al., 1996a (Steubenville)	log-linear	2-day average		all		
	Schwartz et al., 1996a (Portage)	log-linear	2-day average		all		
	Schwartz et al., 1996a (Topeka)	log-linear	2-day average		all		
<b>Hospital Admissions</b>							

Endpoint	Concentration-Response Function		PM Averaging Time		Population <sup>a</sup>	Annual Baseline Incidence (per 100,000 of indicated population) <sup>b</sup>	Pollutant Coefficient <sup>c</sup>
	Source	Functional Form	Studied	Applied			
All respiratory illnesses, using PM2.5 indicator	Thurston et al., 1994 (Toronto)	linear	1-day average	1-day average	all	n/a	$3.45 \times 10^{-8}$
All respiratory illnesses, using PM10 indicator	Schwartz et al., 1995 (Tacoma)	log-linear	1-day average	1-day average	age 65+	504 (general pop.)	0.00170
	Schwartz et al., 1995 (New Haven)	log-linear	1-day average		age 65+		
	Schwartz, 1996 (Spokane)	log-linear	1-day average		age 65+		
COPD, using PM10 indicator	Schwartz, 1994a (Birmingham)	log-linear	1-day average	1-day average	age 65+	103 (general pop.)	0.002533
	Schwartz, 1994b (Detroit)	log-linear	1-day average		age 65+		
	Schwartz, 1996 (Spokane)	log-linear	1-day average		age 65+		
Pneumonia, using PM10 indicator	Schwartz, 1994a (Birmingham)	log-linear	1-day average	1-day average	age 65+	229 (general pop.)	0.0013345
	Schwartz, 1994b (Detroit)	log-linear	1-day average		age 65+		
	Schwartz, 1994c (Minneapolis)	log-linear	1-day average		age 65+		
	Schwartz, 1996 (Spokane)	log-linear	1-day average		age 65+		
Congestive heart failure, using PM10 indicator	Schwartz and Morris, 1995 (Detroit)	log-linear	2-day average	1-day average	age 65+	231 (general pop.)	0.00098
Ischemic heart disease, using PM10 indicator	Schwartz & Morris, 1995 (Detroit)	log-linear	1-day average	1-day average	age 65+	450 (general pop.)	0.00056
<b>Respiratory Symptoms/Illnesses not requiring hospitalization</b>							

Endpoint	Concentration-Response Function		PM Averaging Time		Population <sup>a</sup>	Annual Baseline Incidence (per 100,000 of indicated population) <sup>b</sup>	Pollutant Coefficient <sup>c</sup>
	Source	Functional Form	Studied	Applied			
Development of chronic bronchitis, using PM10 indicator	Schwartz, 1993		annual mean	annual mean	all	n/a	0.012
Acute bronchitis, using PM2.5 indicator	Dockery et al., 1989	logistic	annual mean	annual mean <sup>d</sup>	ages 10-12	n/a	0.0298
Upper respiratory symptoms (URS), using PM10 indicator	Pope et al., 1991	log-linear	1-day average	1-day average	asthmatics, ages 9-11	38,187 (applied pop.)	0.0036
Lower respiratory symptoms (LRS), using PM10 indicator	Schwartz et al., 1994	logistic	1-day average	1-day average	ages 8-12	n/a	0.0142
Lower respiratory symptoms (LRS), using PM2.5 indicator	Schwartz et al., 1994	logistic	1-day average	1-day average	ages 8-12	n/a	0.01823
Asthma (moderate or worse), using PM2.5 indicator	Ostro et al., 1991	linear (with log pollutant)	daily 8-hour average (9:00 am-4:00 pm)	1-day average	asthmatics, ages 9-11	n/a	0.0006
MRADs, using PM2.5 indicator	Ostro and Rothschild, 1989	log-linear	2-week average	1-day average	ages 18-65	780,000 days/year (applied pop.)	0.00741
RADs, using PM2.5 indicator	Ostro, 1987	log-linear	2-week average	1-day average	ages 18-65	400,531 days/year (applied pop.)	0.00475

Endpoint	Concentration-Response Function		PM Averaging Time		Population <sup>a</sup>	Annual Baseline Incidence (per 100,000 of indicated population) <sup>b</sup>	Pollutant Coefficient <sup>c</sup>
	Source	Functional Form	Studied	Applied			
Acute respiratory symptoms (any of 19), using PM10 indicator	Krupnick et al., 1990	logistic	1-day average COH	1-day average	ages 18-65 (study examined "adults")	n/a	0.00046
Shortness of breath (days), using PM10 indicator	Ostro et al., 1995	logistic	1-day average	1-day average <sup>d</sup>	African-American asthmatics, ages 7-12	n/a	0.00841
Work loss days (WLDs), using PM10 indicator	Ostro, 1987	log-linear	2-week average	1-day average	ages 18-65	150,750 days/year (applied pop.)	0.0046
<b>Welfare Endpoints</b>							
Household soiling and damage, using PM2.5 indicator	ESEERCO, 1994	linear	annual mean	annual mean	all households	n/a	2.52 (dollars per µg/m <sup>3</sup> PM10 per household)

NOTES:

<sup>a</sup> The population examined in the study and to which this analysis applies the reported concentration-response relationship. In general, epidemiological studies analyzed the concentration-response relationship for a specific age group (e.g., ages 65+) in a specific geographical area. This analysis applies the reported pollutant coefficient to all individuals in the age group nationwide.

<sup>b</sup> annual baseline incidence in the applied population per 100,000 individuals in the indicated population.

<sup>c</sup> a single pollutant coefficient reported for several studies indicates a pooled analysis; see text for discussion of pooling concentration-response relationships across studies.

<sup>d</sup> The following studies report a lowest observed pollution level:

Pope et al., 1995	Mortality (long-term exposure)	9 µg/m <sup>3</sup> PM <sub>2.5</sub>
Dockery et al., 1995	Acute Bronchitis	11.8 µg/m <sup>3</sup> PM <sub>2.5</sub> (20.1 µg/m <sup>3</sup> PM <sub>10</sub> )
Ostro et al., 1995	Shortness of Breath, days	19.63 µg/m <sup>3</sup> PM <sub>10</sub>

Since these studies did not examine the concentration-response relationship for concentrations below the reported levels, this analysis does not estimate benefits for ambient concentration reductions below these concentrations. The remaining studies did not report lowest observed concentrations.

<sup>e</sup> All 1-day averages are 24-hour averages, 2-day averages are 48-hour averages, etc.

\* See U.S. EPA 1997 for citations



## **I.2 Ozone Health and Welfare Effects Estimation**

### **I.2.1 Table I.2 Ozone Health and Welfare Estimation**

**Table I.2 Ozone Health and Welfare Effects Estimation**

Endpoint	Concentration-Response Function		Ozone Averaging Time		Population <sup>a</sup>	Annual Baseline Incidence (per 100,000 of indicated population) <sup>b</sup>	Pollutant Coefficient <sup>c</sup>
	Source	Functional Form	Studied	Applied			
<b>Mortality (Short-Term Exposure)</b>							
	Anderson et al., 1996	log-linear	1-day average <sup>d</sup>	1-day average	all	803 (nonaccidental deaths in general pop.)	0.001126
	Hoek et al., 1997 (in press)	log-linear	1-day average	1-day average	all		0.001705
	Ito & Thurston, 1996	log-linear	1-day average	1-day average	all		0.000677
	Kinney et al., 1995	log-linear	daily 1-hour max.	daily 1-hour max	all		0.00
	Loomis et al., 1996 (HEI)	log-linear	daily 1-hour max	daily 1-hour max	all		0.000182
	Moolgavkar et al., 1995	log-linear	1-day average	1-day average	all		0.000611
	Ostro et al., 1996	log-linear	daily 1-hour max	daily 1-hour max	all		0.00019
	Samet et al., 1996, 1997 (HEI)	log-linear	1-day average	1-day average	all		0.000936
	Verhoeff et al., 1996	log-linear	daily 1-hour max	daily 1-hour max	all		0.000956
<b>Hospital Admissions</b>							
All respiratory Illnesses	Schwartz, 1996 (Spokane)	log-linear	daily 1-hour max	daily 1-hour max	age 65+	504 (general pop.)	0.008562
All respiratory Illnesses	Schwartz, 1995 (New Haven)	log-linear	1-day average	1-day average	age 65+	504 (general pop.)	0.0014

Endpoint	Concentration-Response Function		Ozone Averaging Time		Population <sup>a</sup>	Annual Baseline Incidence (per 100,000 of indicated population) <sup>b</sup>	Pollutant Coefficient <sup>c</sup>
	Source	Functional Form	Studied	Applied			
All respiratory Illnesses	Schwartz, 1995 (Tacoma)	log-linear	1-day average	1-day average	age 65+	504 (general pop.)	0.0036
All respiratory Illnesses	Thurston et al., 1994 (Toronto)	linear	daily 1-hour max.	daily 1-hour max.	all	n/a	$1.62 \times 10^{-8}$
All respiratory Illnesses	Thurston et al., 1992 (New York City)	linear	daily 1-hour max.	daily 1-hour max	all	n/a	$1.37 \times 10^{-8}$
COPD	Schwartz, 1994a	log-linear	1-day average	1-day average	age 65+	103 (general pop.)	0.00314
COPD	Schwartz, 1994b	log-linear	1-day average	1-day average	age 65+	103 (general pop.)	0.00549
COPD	Schwartz, 1996 (Spokane)	log-linear	daily 1-hour max.	daily 1-hour max	age 65+	103 (general pop.)	0.004619
Pneumonia	Schwartz, 1994a	log-linear	1-day average	1-day average	age 65+	229 (general pop.)	0.00262
Pneumonia	Schwartz, 1994b	log-linear	1-day average	1-day average	age 65+	229 (general pop.)	0.00521
Pneumonia	Schwartz, 1994c	log-linear	1-day average	1-day average	age 65+	229 (general pop.)	0.002795
Pneumonia	Schwartz, 1996 (Spokane)	log-linear	daily 1-hour max.	daily 1-hour max.	age 65+	229 (general pop.)	0.00965

Endpoint	Concentration-Response Function		Ozone Averaging Time		Population <sup>a</sup>	Annual Baseline Incidence (per 100,000 of indicated population) <sup>b</sup>	Pollutant Coefficient <sup>c</sup>
	Source	Functional Form	Studied	Applied			
<b>Respiratory Symptoms not Requiring Hospitalization</b>							
Acute respiratory symptoms (any of 19)	Krupnick et al., 1990	logistic	daily 1-hour max.	daily 1-hour max.	ages 18-65	n/a	0.00014
Asthma attacks	Whittemore and Korn, 1980 and US EPA, 1993	logistic	daily 1-hour max..	daily 1-hour max..	asthmatics	n/a	0.0019
MRADs	Ostro and Rothschild, 1989	log-linear	daily 1-hr max. (avg. over 2 weeks)	daily 1-hr max. (avg. over 2 weeks)	ages 18-65	780,000 days/year (applied pop.)	0.0022
RRADs	Ostro and Rothschild, 1989	log-linear	daily 1-hr max. (avg. over 2 weeks)	daily 1-hr max. (avg. over 2 weeks)	ages 18-65	310,000 days/year (applied pop.)	0.0054
<b>Welfare Endpoints</b>							
Decreased worker productivity	Crocker and Horst, 1981 and US EPA, 1994	percent change	1-day average	1-day average	laborers	n/a	n/a

NOTES:

<sup>a</sup> The population examined in the study and to which this analysis applies the reported concentration-response relationship. In general, epidemiological studies analyzed the concentration-response relationship for a specific age group (e.g., ages 65+) in a specific geographical area. This analysis applies the reported pollutant coefficient to all individuals in the age group nationwide.

<sup>b</sup> annual baseline incidence in the applied population per 100,000 individuals in the indicated population.

<sup>c</sup> a single pollutant coefficient reported for several studies indicates a pooled analysis; see text for discussion of pooling concentration-response relationships across studies.

<sup>d</sup> All 1-day averages are 24-hour averages, 2-day averages are 48-hour averages, etc.

<sup>e</sup> units on linear pollutant coefficient: hospital admissions per ppb O<sub>3</sub> per exposed individual

\* See U. S. EPA 1997 for citations

## **I.3 Valuation and Aggregation**

### **I.3.1 Introduction**

The purpose of this section is to summarize the valuation estimates used to monetize many of the health and welfare benefits categories included in this analysis. In addition, this section describes the procedure this analysis employs to estimate the monetized benefits associated with reductions in premature mortality. For a more detailed description of the procedure used to monetize all other benefits categories, refer to the Benefits Technical Support Document (TSD). (U.S. EPA, 1997a)

Table I.3 presents point estimates for economic values associated with each health and welfare category, by pollutant. Note that there is uncertainty surrounding any estimate of the monetized benefit associated with a unit change in health or welfare effect (e.g., an additional hospital admission avoided). Point estimates are often a central tendency estimate taken from a distribution of possible values. The descriptions of the derivations of the distributions and point estimates of the monetized values (unit dollar values) are presented in the Benefits TSD. (U.S. EPA, 1997a)

#### **Premature Mortality**

Reductions in mortality risk are valued in this monetized benefit analysis using two different approaches, as outlined in the Office of Management and Budget's guidance. The high-end estimate uses a value of statistical life saved approach, and the low-end estimated is based on the value of statistical life year extended approach. Individual WTPs for small reductions in mortality risk are summed over enough individuals to infer the value of a statistical life saved or statistical life-year extended. This is different from the value of a particular, identified life saved. The "value of a premature death avoided" then should be understood as shorthand for the "value of a statistical premature death avoided".

The value of a premature death avoided is based on an analysis of 26 policy-relevant

value of life studies. A summary of these studies is provided in Table I.4. Five of the 26 studies are contingent valuation (CV) studies, which directly solicit WTP information from subjects; the rest are wage-risk studies, which base WTP estimates on estimates of the additional compensation demanded in the labor market for riskier jobs. Each of the 26 studies provides an estimate of the mean WTP to avoid a statistical premature death. Several plausible standard distributions were fit to the 26 estimates of mean WTP. A Weibull distribution, with a mean of \$4.8 million and standard deviation of \$3.24 million, provided the best fit to the 26 estimates. The central tendency estimate of the WTP to avoid a statistical premature death is the mean of this distribution, \$4.8 million. The value of statistical life-year extended was derived from a number of studies, including Moore and Viscusi (1988) and Miller, Calhoun, Arthur (1990)--summarized in Tolley, et. al. (1994). Tolley, et. al. report a range for the value of a statistical life-year of \$70,000, \$120,000, and \$175,000. This analysis uses the midpoint of that range, \$120,000 per life-year extended.

The transferability of estimates of the value of a statistical life from the 26 studies to these benefits analyses rests on the assumption that, within a reasonable range, WTP for reductions in mortality risk is linear in risk reduction. In addition, the characteristics of the study subjects and the nature of the mortality risk being valued in the study could affect the transferability of the value of statistical life to this assessment.

Compared with the subjects in wage-risk studies, the population believed to be most affected by PM (i.e., the population that would receive the greatest mortality risk reduction associated with a given reduction in PM concentrations) is, on average, older and probably more risk averse. Citing Schwartz and Dockery (1992) and Ostro et al. (unpublished), Chestnut estimates that approximately 85 percent of those who die prematurely from PM-related causes are over 65 years of age. The average age of subjects in wage-risk studies, in contrast, would be well under 65. At this time, there is insufficient information in the current ozone-related mortality literature to conclude that premature mortality related to ozone exposure is age-dependent.

**Table I.3 Economic Valuation of Health and Welfare Effects of PM, Ozone, and Regional Haze  
1990 \$**

Health or Welfare Effect	Pollutant(s) <sup>a</sup>	Valuation Measure <sup>b</sup>	Unit Value (Point Estimate)	Comments
<b>Mortality:</b>				
Statistical Lives Saved	PM <sub>10</sub> /PM <sub>2.5</sub> /O <sub>3</sub>	\$ per case	\$4.8 million	
Life-Years Saved	PM <sub>10</sub> /PM <sub>2.5</sub>	\$ per life-year	\$120,000	
<b>Hospital Admissions:</b>				
All Respiratory Illnesses, all ages	PM <sub>10</sub> /PM <sub>2.5</sub>	\$ per hospital admission	\$12,700	The PM value is smaller than for ozone because opportunity cost is excluded from the PM value to avoid double-counting (see the next section). Also, the study estimating a concentration-response function for PM defines "all respiratory illnesses" slightly differently from the corresponding ozone study.
	O <sub>3</sub>	\$ per hospital admission	\$13,400	
Pneumonia, age ≥ 65	PM <sub>10</sub> /O <sub>3</sub>	\$ per hospital admission	\$15,900	
COPD, age ≥ 65	PM <sub>10</sub> /O <sub>3</sub>	\$ per hospital admission	\$15,700	
Ischemic Heart Disease, age ≥ 65	PM <sub>10</sub>	\$ per hospital admission	\$20,600	
Congestive Heart Failure, age ≥ 65	PM <sub>10</sub>	\$ per hospital admission	\$16,600	
Emergency Department Visits for Asthma	O <sub>3</sub>	\$ per hospital admission	\$9,000	
<b>Respiratory Ailments Not Requiring a Hospital Admission:</b>				
Chronic Bronchitis	PM <sub>10</sub>	\$ per case	\$260,000	

Health or Welfare Effect	Pollutant(s) <sup>a</sup>	Valuation Measure <sup>b</sup>	Unit Value (Point Estimate)	Comments
Upper Respiratory Symptoms (URS)	PM <sub>10</sub>	\$ per symptom-day	\$19	
Lower Respiratory Symptoms (LRS)	PM <sub>10</sub> /PM <sub>2.5</sub>	\$ per symptom-day	\$12	
Acute Bronchitis	PM <sub>10</sub> /PM <sub>2.5</sub>	\$ per case	\$45	
Acute Respiratory Symptoms: Any of 19	PM <sub>10</sub> /O <sub>3</sub>	\$ per symptom-day	\$18	
Asthma <sup>c</sup>	O <sub>3</sub> /PM <sub>2.5</sub>	\$ per symptom-day	\$32	
Shortness of Breath	PM <sub>10</sub>	\$ per symptom-day	\$5.30	
Sinusitis and Hay Fever	O <sub>3</sub>	-----	quantified but not monetized	
<b>Restricted Activity:</b>				
Work Loss Day (WLD)	PM <sub>2.5</sub>	\$ per day	\$83	
Restricted Activity Day (RAD)	PM <sub>2.5</sub>	\$ per day	quantified but not monetized	
Minor Restricted Activity Day (MRAD)	O <sub>3</sub> /PM <sub>2.5</sub>	\$ per day	\$38	
Respiratory Restricted Activity Day (RRAD)	O <sub>3</sub> /PM <sub>2.5</sub>	-----	quantified but not monetized	
<b>Welfare Effects:</b>				
Worker Productivity (resulting in changes in daily wages)	O <sub>3</sub>	change in daily wages	\$1 per worker per 10% change in O <sub>3</sub> <sup>d</sup>	



Health or Welfare Effect	Pollutant(s) <sup>a</sup>	Valuation Measure <sup>b</sup>	Unit Value (Point Estimate)	Comments
Visibility (residential)	deciview	annual household WTP	WTP per unit decrease in deciview = \$14	
Visibility (recreational)	deciview	annual household WTP		see U.S. EPA 1997 for valuations
Household Soiling Damage	TSP	\$ per household per $\mu\text{g}/\text{m}^3$ $\text{PM}_{10}$ (annual cost)	\$2.50	

NOTES:

<sup>a</sup> Attainment for which epidemiological evidence quantifies a concentration-response relationship for the given endpoint

<sup>b</sup> Most unit values quantify the willingness to pay (WTP) to avoid a case of the given effect. However, for those effects measured in terms of symptom-days, the unit value reflects the WTP to avoid one day of the given respiratory symptoms

<sup>c</sup> Asthma is either self-reported asthma or moderate or worse asthma status

<sup>d</sup> Deciview (DV) is a common visibility measure useful for characterizing visibility in terms of perceptible changes independent of baseline conditions. A decrease in deciview corresponds to an increase in visibility. It is related to another common visibility measure, visual range (VR):  $DV = 10 \ln[391 \text{ km}/VR]$  where DV is unitless and VR is measured in kilometers

\* See U.S. EPA 1997 for citations

**Table I.4 Summary of Mortality Valuation Estimates**  
(millions of 1990 \$)

<b>Study</b>	<b>Type of Estimate</b>	<b>Valuation per Statistical Life</b>
Kneisner and Leeth (1991) (U.S.)	Labor Market	0.6
Smith and Gilbert (1984)	Labor Market	0.7
Dillingham (1985)	Labor Market	0.9
Butler (1983)	Labor Market	1.1
Miller and Guria (1991)	Contingent Valuation	1.2
Moore and Viscusi (1988a)	Labor Market	2.5
Viscusi, Magat, and Huber (1991b)	Contingent Valuation	2.7
Gegax et al. (1985)	Contingent Valuation	3.3
Marin and Psacharopoulos (1982)	Labor Market	2.8
Kneisner and Leeth (1991) (Australia)	Labor Market	3.3
Gerking, de Haan, and Schulze (1988)	Contingent Valuation	3.4
Cousineau, Lacroix, and Girard (1988)	Labor Market	3.6
Jones-Lee (1989)	Contingent Valuation	3.8
Dillingham (1985)	Labor Market	3.9
Viscusi (1978, 1979)	Labor Market	4.1
R.S Smith (1976)	Labor Market	4.6
V.K. Smith (1976)	Labor Market	4.7
Olson (1981)	Labor Market	5.2
Viscusi (1981)	Labor Market	6.5
R.S. Smith (1974)	Labor Market	7.2
Moore and Viscusi (1988a)	Labor Market	7.3
Kneisner and Leeth (1991) (Japan)	Labor Market	7.6
Herzog and Schlottman (1987)	Labor Market	9.1
Leigh and Folson (1984)	Labor Market	9.7
Leigh (1987)	Labor Market	10.4
Gaten (1988)	Labor Market	13.5

\* Source: Viscusi, 1992

There is also reason to believe that those over 65 are, in general, more risk averse than the general population while workers in wage-risk studies are likely to be less risk averse than the general population. Although Viscusi's list of recommended studies excludes studies that consider only much-higher-than-average occupational risks, there is nevertheless likely to be some selection bias in the remaining studies, i.e., these studies are likely to be based on samples of workers who are, on average, more willing to accept higher risks than the general population. In contrast, older people as a group exhibit more risk averse behavior.

In addition, it might be argued that because the elderly have greater average wealth than those younger, the affected population is also wealthier, on average, than wage-risk study subjects, who tend to be blue collar workers. It is possible, however, that among the elderly, it is largely the poor elderly who are most vulnerable to PM-related mortality (e.g., because of generally poorer health care). If this is the case, the average wealth of those affected by a reduction in PM concentrations relative to that of subjects in wage-risk studies is uncertain.

The direction of bias resulting from the age difference is unclear, particularly because age is confounded by risk aversion (relative to the general population). It could be argued that, because an older person has fewer expected years left to lose, his/her WTP to reduce mortality risk would be less than that of a younger person. This hypothesis is supported by one empirical study, Jones-Lee et al. (1985), that found the value of a statistical life at age 65 to be approximately 90 percent of what it is at age 40. Citing the evidence provided by Jones-Lee et al., Chestnut (1995) estimates a weighted average value of a statistical life based on the approximate age distribution for the U.S. population age 65 and older. This results in an adjustment to the value of a statistical life for those 65 and over of 75 percent of what it is for those under 65.

The greater risk aversion of older people, however, implies just the opposite. Citing Ehrlich and Chuma (1990), IEc (1992) notes that "older persons, who as a group tend to avoid health risks associated with drinking, smoking, and reckless driving, reveal a greater demand for reducing mortality risks and hence have a greater implicit value of a life year." That is, the more

risk averse behavior of older individuals suggests a greater WTP to reduce mortality risk.

There is substantial evidence that the income elasticity of WTP for health risk reductions is positive, although there is uncertainty about the exact value of this elasticity. Individuals with higher incomes (or greater wealth) should be willing to pay more to reduce risk, all else equal, than individuals with lower incomes or wealth. Whether the average income or level of wealth of the population affected by PM reductions is likely to be significantly different from that of subjects in wage-risk studies, however, is unclear, as discussed above.

Finally, there is some evidence (see, for example, Violette and Chestnut, 1983) that people will pay more to reduce involuntarily incurred risks than risks incurred voluntarily. If this is the case, WTP estimates based on wage-risk studies may be downward-biased estimates of WTP to reduce involuntarily incurred PM-related mortality risks.

### **Hospital Admissions**

The value to an individual of avoiding a hospital admission is measured by the individual's WTP to avoid the hospital admission. This value is the amount of money such that the individual would be indifferent between having the money and avoiding the hospital admission. An individual's WTP will include, at a minimum the amount of money he pays for medical expenses and the loss in earnings. In addition, an individual is likely to be willing to pay some amount to avoid the pain and suffering associated with the illness itself.

The total value to society of an individual's avoiding a hospital admission, then, might be thought of as having two components: (1) the cost of illness (COI) to society, including the total medical costs plus the value of the lost productivity, as well as (2) the individual's WTP to avoid the disutility of the illness itself (e.g., the pain and suffering associated with the illness). It is useful to note that although medical expenditures are to a significant extent shared by society, via medical insurance, Medicare, etc. However, the limited evidence comparing individual WTPs to social COI suggests that individual WTPs to avoid morbidity effects generally do in fact exceed the total COIs associated with those effects.

In the absence of estimates of social WTP to avoid hospital admissions for specific illnesses (components 1 plus 2 above), estimates of total COI (component 1) are typically used as lower-bound estimates. Because these estimates do not include the value of avoiding the disutility of the illness itself (component 2), they are biased downward. This analysis adjusts the COI estimate upward by multiplying an estimate of the ratio of WTP to COI to better approximate total WTP to avoid a hospital admission.

The average physician charges of the first day of hospital care for asthma or COPD is estimated as \$94; average physician charges for subsequent days of hospital care are estimated to be \$35. Average physician charges associated with hospital care for asthma or COPD are assumed to provide reasonably good estimates of average physician charges associated with hospital stays for the other illness categories considered here.

To estimate the opportunity cost of a day spent in the hospital for an individual aged 65 or older, it is assumed that such an individual is not in the workforce. As an approximation, it is assumed that, for the young, the elderly, and any other unemployed individuals the opportunity cost of a day spent in the hospital is one-half the median daily wage, or \$41.50. Thus, the opportunity cost associated with a hospital admission is simply equal to \$41.50 times the average number of days of the hospital stay.

To derive unit dollar values for hospital admissions for respiratory illness based on the Thurston study, which considered individuals of all ages, it is assumed that half of the PM-related hospital admissions are among individuals who are not employed, including the young and the elderly. Because the value of work loss days for those in the labor force is considered as a separate endpoint, only the opportunity cost for those outside of the workforce is included.

Since COI estimates do not measure values associated with pain and suffering, as well as other reductions in well-being from illness, they significantly understate the true WTP to avoid illness. For this reason, an adjustment factor is employed to scale the hospital admission COI estimate upward to estimate WTP. Using evidence from a range of estimates that examine WTP

to COI ratios (Rowe and Chestnut, 1986; Rowe et al., 1984; and Rowe and Neither cut, 1987), the hospital admissions COI estimate is multiplied by a factor of 2. This factor is based on results from three studies providing evidence on WTP/COI ratios for the same study population addressing the same change in the same health effect. While this adjustment approach is based on limited evidence, the resulting hospital admissions valuation estimate is not clearly biased.

There is substantial uncertainty associated with the adjustment factor of 2. Acknowledging that the adjustment factor may vary from one endpoint to another, the factor is taken to have a continuous uniform distribution from 1.5 to 2.5, with a mean of 2. This distribution is both simple and consistent with the point estimate of 2.

The hospital charge component of COI is generally an order of magnitude greater than the other two components (physician charge and opportunity cost). Sample mean hospital charges, as well as standard errors of the means, are provided by Elixhauser et al., 1993. An asymptotic normality of the sample mean can be invoked because these sample means are generally based on very large samples.

The physician charge and opportunity cost are relatively small components of the COI associated with a hospital admission. Including estimates of uncertainty surrounding these two small components of WTP to avoid a hospital admission is therefore largely “fine tuning.” These components are omitted from the uncertainty analysis because information concerning their distributions is lacking. The following distributional form is used for the COI associated with each of the hospital admission classifications: a normal distribution with mean = the point estimate (i.e., the mean hospital charge + physician charge + opportunity cost) and standard deviation = the standard error of the mean hospital charge reported in the Elixhauser et al. study.

#### Chronic Bronchitis

Chronic bronchitis is one of the only morbidity endpoints that may be expected to last from the initial onset of the illness throughout the rest of the individual’s life. WTP to avoid chronic bronchitis would therefore be expected to incorporate the present discounted value of a

potentially long stream of costs (e.g., medical expenditures and lost earnings) associated with the illness. Two studies, Viscusi et al. (1991) and Krupnick and Cropper (1992) provide estimates of WTP to avoid a case of chronic bronchitis. The study by Viscusi et al., however, uses a sample that is larger and more representative of the general population than the study by Krupnick and Cropper (which selects people who have a relative with the disease). The valuation of chronic bronchitis in this analysis is therefore based on the distribution of WTP responses from Viscusi et al. (1991).

Both Viscusi et al. and Krupnick and Cropper, however, defined a case of severe chronic bronchitis. It is unclear what proportion of the cases of chronic bronchitis predicted to be associated with exposure to pollution would turn out to be severe cases. The incidence of pollution-related chronic bronchitis was based on Abbey et al. (1993), which considered only new cases of illness. While a new case may not start out being severe, chronic bronchitis is a chronic illness which may progress in severity from onset throughout the rest of the individual's life. It is the chronic illness which is being valued, rather than the illness at onset.

The WTP to avoid a case of pollution-related chronic bronchitis is derived by starting with the WTP to avoid a severe case of chronic bronchitis, as described by Viscusi et al. (1991), and adjusting it downward to reflect (1) the decrease in severity of a case of pollution-related chronic bronchitis relative to the severe case in the Viscusi study, and (2) the elasticity of WTP with respect to severity. Because elasticity is a marginal concept and because it is a function of severity (as estimated from Krupnick and Cropper), WTP adjustments were made incrementally, in one percent steps. At each step, given a WTP to avoid a case of CB of severity level  $sev$ , the WTP to avoid a case of severity of  $0.99*sev$  was derived. This procedure is iterated until the desired severity level was reached and the corresponding WTP estimate is derived. Because the elasticity of WTP with respect to severity is a function of severity, this elasticity changes at each iteration. If for example, it is believed that a pollution-related case of chronic bronchitis is of average severity, that is 50 percent reduction in severity from the case described in the Viscusi study, then the iterative procedure would proceed until the severity level was half of what it started out to be.

The derivation of the WTP to avoid a case of pollution-related chronic bronchitis is based on three components, each of which is uncertain: (1) the WTP to avoid a case of severe chronic bronchitis, as described in the Viscusi study, (2) the severity level of an average pollution-related case of chronic bronchitis (relative to that of the case described by Viscusi), and (3) the elasticity of WTP with respect to severity of the illness. These three sources of uncertainty make the WTP estimate uncertain. Based on assumptions about the distributions of each of the three uncertain components, a distribution of WTP to avoid a pollution-related case of chronic bronchitis is derived by Monte Carlo methods. The mean of this distribution, which is \$260,000, is taken as the central tendency estimate of WTP to avoid a pollution-related case of chronic bronchitis.

The distribution of WTP to avoid a case of pollution-related chronic bronchitis is generated by Monte Carlo methods, drawing on distribution estimates related to: (a) the distribution to avoid a severe case of chronic bronchitis (mean = \$720,000); (b) the distribution of the severity level of an average case of pollution-related chronic bronchitis (represents a 50 percent reduction in severity from a severe case); and (c) the elasticity of WTP to avoid a case of chronic bronchitis (mean = 0.18 and standard deviation = 0.0669). On each of 16,000 iterations, (1) a value is selected from each distribution, and (2) a value for WTP is generated by the iterative procedure, in which the severity level is decreased by one percent on each iteration on each iteration and the corresponding WTP value is derived. The mean of the resulting distribution of WTP to avoid a case of pollution-related chronic bronchitis is \$260,000.



## I.4 Sensitivity Analyses

### I.4.1 Introduction

This section presents results associated with several benefits sensitivity analyses. These sensitivity analyses include: (1) examining the sequence of a PM following ozone analysis and (2) examining the results of using a proportional air quality rollback procedure to adjust ozone concentrations.

### I.4.2 Sequenced Analyses

The PM and ozone benefits estimates presented in chapter 12 represent benefits estimated for air quality changes incremental to partial or full attainment of the current standards. However, the benefits estimates of the alternative PM and ozone standards do not reflect any possible overlap with each other. For example, partial attainment benefits of the 15/50 PM<sub>2.5</sub> and .08, 3rd max. ozone alternatives are estimated incremental from partial attainment of the current standards. However, these estimates do not reflect any overlap of benefits that may occur between the 15/50 PM<sub>2.5</sub> and .08, 3rd max. ozone alternatives since the estimates are calculated independently of each other.

It is important to know if significant benefits overlap exists between the PM<sub>2.5</sub> and ozone alternatives because the total benefits associated with the combined PM and ozone NAAQS is relevant information. However, lack of adequate air quality modeling data precluded the estimation of the ozone following PM analysis. Therefore, the sensitivity analysis was conducted for the PM following ozone sequence, using the proposed standards as case studies.

This sensitivity analysis was conducted using a preliminary set of air quality data that does not exactly match the air quality data used to estimate benefits as presented in chapter 12. Therefore, the results presented in this appendix are not directly comparable to the benefits results presented in chapter 12. Although the preliminary air quality data used in this sensitivity

analysis does not represent the final and most accurate set of air quality data, the results of this analysis may provide insight into the magnitude and/or direction of the benefits results when considering the sensitivity factors.

In a PM following ozone analysis, the ozone benefits results are unaffected (e.g., identical to ozone-only analysis) because the benefits of the ozone standard are calculated incremental to the current ozone standard, regardless of whether a PM alternative follows the ozone analysis. Therefore, the comparison of most interest is the comparison between the PM-only analysis and the PM following ozone analysis. These partial attainment results are estimated incremental to partial attainment of the current ozone and PM<sub>10</sub> NAAQS as well as the .08, 3rd max. ozone standard and are presented in Table I.5. The high end benefits range for the PM-only analysis is approximately \$59 billion to \$109 billion while the high end benefits range for the PM following ozone analysis is approximately \$55 billion to \$104 billion. These results indicate that while some individual endpoints may be slightly overestimated when summing the ozone-only and PM-only results, total benefits estimates would not significantly be overestimated using either set (PM-only or PM following ozone) of results. Also, note that the total benefits estimates are often reported at the 2 significant figure level. Given this level of rounding, there is little detectable difference between the two analyses. Therefore, although individual estimates may be slightly overstated when the PM and ozone NAAQS are summed, total benefits are not expected to be overstated.

#### I.4.3 Proportional Air Quality Rollback for Ozone

The ozone benefits estimates presented in chapter 12 are associated with ozone air quality changes calculated by a quadratic air quality rollback procedure. Recall that a rollback procedure is necessary due to lack of adequate air quality modeling data. The Agency recognizes that the choice of a rollback procedure may significantly affect the benefits results. Therefore, a sensitivity analysis was conducted (using the preliminary air quality data set) to ascertain the influence the choice of an air quality rollback procedure could have.

**Table I.5 Sensitivity Analysis of Sequenced PM and Ozone Alternatives  
PM : National Annual Monetized Health and Welfare Benefits<sup>1</sup>**

Estimates are incremental to the .08 ppm, 3rd max. ozone and current PM NAAQS (50 µg/m<sup>3</sup> annual; 150 µg/m<sup>3</sup> daily)  
(millions of 1990 \$; year = 2010)

ENDPOINT <sup>2</sup>	Annual PM <sub>2.5</sub> (µg/m <sup>3</sup> )  Daily PM <sub>2.5</sub> (µg/m <sup>3</sup> )	Partial Attainment Scenario (High End)	
		PM - Only	PM Following Ozone
		15	15
		50	50
*Mortality <sup>3</sup> :short-term exposure		\$27,000	\$27,000
long-term exposure		\$78,000	\$76,000
*Chronic Bronchitis		\$23,000	\$20,000
<b>Hospital Admissions:</b>			
*all respiratory (all ages)		\$80	\$80
all resp. (ages 65+)		\$110	\$110
pneumonia (ages 65+)		\$50	\$50
COPD (ages 65+)		\$40	\$40
*congestive heart failure		\$40	\$40
*ischemic heart disease		\$50	\$50
*Acute Bronchitis		\$2	\$1
*Lower Respiratory Symptoms		\$7	\$4
*Upper Respiratory Symptoms		\$1	\$1
shortness of breath		\$1	\$1
asthma attacks		\$14	\$13
*Work Loss Days		\$270	\$270
*Minor Restricted Activity Days (MRADs)		\$1,000	\$1,000
Household Soiling		\$960	\$400
Visibility		\$6,170	\$6,031
<b>TOTAL MONETIZED BENEFITS</b>			
using short-term PM mortality		\$59,000	\$55,000
using long-term PM mortality		\$109,000	\$104,000

<sup>1</sup> numbers may not completely agree due to rounding

<sup>2</sup> only endpoints denoted with an \* are aggregated into total benefits estimates

<sup>3</sup> mortality estimates must be aggregated using either short-term exposure or long-term exposure but not both due to double-counting issues

The alternative ozone air quality rollback procedure employed in this sensitivity analysis is referred to as proportional (also called linear) rollback procedure. This method for adjusting PM concentrations decreases baseline PM concentrations on all days by the same percentage. (Recall that quadratic rollback as employed in chapter 12 reduces non-peak ozone values (e.g., wintertime ozone values) by a smaller proportion compared to peak ozone values (e.g., ozone concentrations at design-value monitors). This sensitivity analysis is estimated using the current ozone standard, partial attainment scenario since all subsequent benefits results are estimated incremental to partial attainment of the current standard.

The results of the quadratic rollback procedure compared to the proportional rollback procedure are presented in Table I.6. Note that unlike chapter 12, a smaller number of categories is presented in Table I.6. The choice of a rollback procedure affects only ozone concentration-response functions since ancillary PM air quality changes are estimated using the source-receptor model and are unaffected by the choice of an ozone air quality rollback procedure. In addition, other benefits categories such as nitrogen deposition and air toxics are estimated using the VOC or NO<sub>x</sub> emission reductions as reported in the cost analysis. Therefore, only categories that are estimable and are affected by the choice of the ozone air quality rollback procedure are presented in Table I.6.

An examination of the results in Table I.6 indicate that in general, the ozone health and welfare benefits estimated using a proportional rollback procedure are approximately 2 times greater when compared to the benefits estimates calculated with air quality changes using a quadratic air quality rollback procedure. The directional result of this sensitivity analysis (larger benefits estimates using a proportional rollback procedure compared to a quadratic rollback procedure) is consistent with expectations regarding the results. As explained in section 12.6 of chapter 12, a proportional air quality rollback procedure adjusts baseline ozone concentrations on all days by the same percentage. Alternatively, the quadratic air quality rollback procedure adjusts baseline ozone concentrations using a quadratic formula that reduces non-peak ozone

**Table I.6 Sensitivity Analysis of Proportional Ozone Air Quality Rollback Procedure**  
**Ozone : National Annual Monetized Benefits of Selected Health and Welfare Categories<sup>1</sup>**  
 (billions of 1990 \$; year = 2010)

ENDPOINT <sup>2</sup>	Partial Attainment Scenario	
	Quadratic Rollback	Proportional Rollback
	.12 ppm, 1 hour	.12 ppm, 1 hour
<b>*Mortality</b>	\$0.57	\$1.1
<b>Hospital Admissions:</b>		
*all respiratory (all ages)	\$0.007	\$0.013
all respiratory (ages 65+)	\$0	\$0.038
pneumonia (ages 65+)	\$0.010	\$0.019
COPD	\$0.003	\$0.006
emer. dept. visits for asthma	\$0.002	\$0.004
<b>*Acute Respiratory Symptoms</b> (any of 19)	\$0.001	\$0.002
<b>Asthma Attacks</b>	\$0	\$0
<b>Minor Restricted Activity Days</b> (MRAD's)	\$0	\$0
<b>*Commodity Crops</b>	\$0.038	\$0.075
<b>*Fruits and Vegetables</b>	\$0.150	\$0.270
<b>*Worker Productivity</b>	\$0.014	\$0.029
<b>TOTAL MONETIZED BENEFITS</b>	\$0.77	\$1.5

\*This table does not represent total benefits associated with the standard, only represents benefits associated with a selected collection of benefits categories affected by the choice of an ozone air quality rollback procedure for the high-end estimate. For example, ancillary PM benefits are not listed here because they are unaffected by the ozone air quality rollback procedure.

<sup>1</sup> numbers may not completely agree due to rounding

<sup>2</sup> only endpoints denoted with an \* are aggregated into total benefits estimates

concentrations by a smaller percentage than peak ozone concentrations. The difference between the two procedures is that proportional rollback reduces the majority of the baseline ozone concentrations by a greater percentage when compared to the quadratic rollback procedure. All inputs (e.g., concentration-response functions, valuation estimates, etc.) other than air quality changes are constant between the two analyses. Given that the air quality change is greater using proportional rollback, the benefits results showing larger benefits estimates associated with proportional rollback compared to quadratic rollback is consistent with the relative air quality changes.

### **I.5 Ozone Benefits Using Clinical Studies**

Clinical studies of air pollution involve exposing human subjects to various levels of air pollution in a carefully controlled and monitored laboratory situation. The physical condition of the subjects is measured before, during, and after the pollution exposure. The advantage of clinical studies is that they often can isolate cause-effect relationships between pollutants and certain human health effects. However, there are also drawbacks to using clinical studies for a comprehensive benefits analysis. Drawbacks include limitations on studying severe effects or effects caused by long-term exposure and limitations to the potential study scope due to ethical considerations. However, data estimated from clinical concentration - response functions provide useful and relevant information and are presented here to support the benefits analysis effort. Clinical models are available only for ozone-related exposures and are therefore, only applicable to the ozone benefits analysis.

Table I.7 presents information associated with each clinical concentration-response function. Health endpoints evaluated by the clinical models include: change in forced expiratory volume (DFEV) of  $\geq 10\%$ ,  $\geq 15\%$ ,  $\geq 20\%$ ; coughs, pain upon deep inhalation (PDI), and lower respiratory symptoms.

Each clinical model identifies the change in health effect as a rate; for example, as a per capita value. In order to identify the aggregate population impact, it is necessary to specify the

population affected. The clinical analysis evaluates the concentration-response functions for three sub-population groups: outdoor children, outdoor workers, all other adults other than outdoor workers. These results are then summed to provide a total estimate of benefits.

When evaluating the clinical studies, the concentration-response functions provide an estimate of the number of times (incidences) that a health symptom would occur over a 16-hour day (8 am to 12 am). However, valuation estimates that are used to estimate the economic value of avoiding these health effects are estimated in terms of dollars per avoided “symptom day.” For example, evaluation of a clinical coughing model over a 16-hour day would yield the total number of times a cough is expected to occur during this time period given a particular level of ambient ozone. This estimate does not differentiate between multiple coughs experienced by one person versus one cough experienced by many people.

Due to the definition of a symptom day as reported in the contingent valuation surveys, it is necessary to convert the number of incidences of a health symptom into a comparable count of the number of symptom days. This conversion is accomplished by applying each concentration-response function to the daily time period specified by the model (e.g., two-hour period) reported as having the highest ozone concentration during that day. This time period corresponds to the highest probability of response among the affected population for that day and as such, this daily period will capture the maximum number of people who would experience a health symptom as a result of ozone exposure if activity patterns were constant across the day. This period is used to define the “incidence-day” (i.e., symptom-day) estimates for each concentration-response model.

Table I.7 Clinical Model Descriptive Characteristics

#	Health End-Point	Citation	Study Exposure Period	Benefit Analysis Exposure Period	Functional Form	Alpha	Beta	d	e	r <sup>2</sup>	Concentration Value When Incidence=0
1	DFEV <sub>1</sub> ≥ 10%	Avol et al. (1984)	1.33 hours	1 hour	Linear	-0.2395	3.-4388			0.98	0.0696
2	DFEV <sub>1</sub> ≥ 15%	Avol et al. (1984)	1.33 hours	1 hour	Linear	-0.2400	2.-9713			0.99	0.0808
3	DFEV <sub>1</sub> ≥ 20%	Avol et al. (1984)	1.33 hours	1 hour	Linear	-0.2395	2.-6825			0.99	0.0893
4	DFEV <sub>1</sub> ≥ 10%	Kulle et al. (1985)	2 hours	2 hours	Linear	-0.3225	2.-3500			0.95	0.1372
5	DFEV <sub>1</sub> ≥ 15%	Kulle et al. (1985)	2 hours	2 hours	Linear	-0.2600	1.600			0.93	0.1625
6	DFEV <sub>1</sub> ≥ 20%	Kulle et al. (1985)	2 hours	2 hours	Linear	-0.2375	1.-2500			0.89	0.1900
7	DFEV <sub>1</sub> ≥ 10%	McDonnell et al. (1983)	2.5 hours	2 hours	Logistic		0.-6420	5.-5996	-27.-2927	0.99	
8	DFEV <sub>1</sub> ≥ 15%	McDonnell et al. (1983)	2.5 hours	2 hours	Logistic		0.-4968	9.-4948	-45.-3838	1.00	



#	Health End-Point	Citation	Study Exposure Period	Benefit Analysis Exposure Period	Functional Form	Alpha	Beta	d	e	r <sup>2</sup>	Concentration Value When Incidence=0
9	DFEV <sub>1</sub> > 20%	McDonnell et al. (1983)	2.5 hours	2 hours	Logistic		0.-3347	12.-0073	-60.-4547	1.00	
10	DFEV <sub>1</sub> > 10%	Seal et al. (1993)	2.33 hours	2 hours	Probit	-1.0276	0.-7917			0.99	
11	DFEV <sub>1</sub> > 15%	Seal et al. (1993)	2.33 hours	2 hours	Probit	-0.6639	0.-8401			0.99	
12	DFEV <sub>1</sub> > 20%	Seal et al. (1993)	2.33 hours	2 hours	Probit	-0.3259	0.-9192			0.97	
13	DFEV <sub>1</sub> > 10%	FHM <sup>2</sup>	8 hours	8 hours	Linear	-0.0980	5.-0000			1.00	0.0196
14	DFEV <sub>1</sub> > 15%	FHM	8 hours	8 hours	Linear	-0.2087	4.-9000			1.00	0.0426
15	DFEV <sub>1</sub> > 20%	FHM	8 hours	8 hours	Linear	-0.1462	2.-9250			0.98	0.0500
16	Lower Respiratory Symptoms	Avol et al. (1984)	1.33 hours	1 hour	Linear	-0.2084	2.-6824			0.99	0.0777

#	Health End-Point	Citation	Study Exposure Period	Benefit Analysis Exposure Period	Functional Form	Alpha	Beta	d	e	r <sup>2</sup>	Concentration Value When Incidence=0
17	Moderate to Severe Lower Respiratory Symptoms	Avol et al. (1984)	1.33 hours	1 hour	Linear	- 0.090 2	0.- 5206			0.94	0.1733
18	Cough	Kulle et al. (1985)	2 hours	2 hours	Linear	- 0.265 0	3.- 0000			0.97	0.0883
19	Pain Upon Deep Inhalation	Kulle et al. (1985)	2 hours	2 hours	Linear	- 0.455 0	3.- 8000			0.79	0.1197
20	Moderate to Severe Cough	Kulle et al. (1985)	2 hours	2 hours	Linear	- 0.162 6	0.- 8675			-0.33 <sup>1</sup>	0.1874
21	Moderate to Severe Pain Upon Deep Inhalation	Kulle et al. (1985)	2 hours	2 hours	Linear	- 0.525 0	3.- 0000			0.72	0.1750
22	Cough	McDonnell et al. (1983)	2.5 hours	2 hours	Probit	- 2.095 4	1.- 2098			0.99	
23	Pain Upon Deep Inhalation	McDonnell et al. (1983)	2.5 hours	2 hours	Probit	- 1.607 1	1.- 5124			0.96	
24	Moderate to Severe Cough	McDonnell et al. (1983)	2.5 hours	2 hours	Linear	0.006 2	1.- 2604			0.70	-0.0049

#	Health End-Point	Citation	Study Exposure Period	Benefit Analysis Exposure Period	Functional Form	Alpha	Beta	d	e	r <sup>2</sup>	Concentration Value When Incidence=0
25	Moderate to Severe Pain Upon Deep Inhalation	McDonnell et al. (1983)	2.5 hours	2 hours	Linear	-0.0427	1.-1512			0.96	0.0371
26	Cough	Seal et al. (1993)	2.33 hours	2 hours	Lognormal	0.2469	1.-9248			0.97	
27	Pain Upon Deep Inhalation	Seal et al. (1993)	2.33 hours	2 hours	Lognormal	0.2464	2.-3641			0.99	
28	Moderate to Severe Cough	Seal et al. (1993)	2.33 hours	2 hours	Linear	-0.1445	1.-3704			0.97	0.1054
29	Moderate to Severe Pain Upon Deep Inhalation	Seal et al. (1993)	2.33 hours	2 hours	Probit	-0.3209	0.-9317			0.96	
31	Cough	FHM	8 hours	8 hours	Linear	-0.2928	5.-0750			0.54	0.0577
32	Pain Upon Deep Inhalation	FHM	8 hours	8 hours	Linear	0.7372	10.-1750			1.00	-0.0725
34	Moderate to Severe Cough	FHM	8 hours	8 hours	Linear	-0.1747	2.-3000			0.88	0.0760
35	Moderate to Severe Pain Upon Deep Inhalation	FHM	8 hours	8 hours	Linear	-0.3087	3.-7000			0.93	0.0834

1. The data do not support a meaningful exposure-response relationship for this health end-point. The negative

$r^2$  value is an indicator of this situation.

2. FHM: Folinsbee et. al. (1988), Horstman et. al. (1990), and McDonnell et. al. (1991).

\* See Mathtech, Inc. (1997).

Table I.8 presents valuation information. (Neuman et al, 1993) Note that valuation estimates are only available for two endpoints: any cough and any PDI.

**Table I.8 Willingness-to-Pay Estimates (1990 \$)**

Health Endpoint	WTP Value Per Incidence-Day		
	Low Estimate	Best Estimate	High Estimate
Cough	\$1.26	\$7.00	\$13.84
PDI	\$1.26	\$4.41	\$28.04

The clinical benefits estimates presented here represent benefits attributable to air quality changes within the identified ozone nonattainment and transport areas. The definition of these areas is described in chapter 4. The estimation of post-control ozone air quality is described in chapter 12.

Benefits estimates presented in chapter 12 represent ozone air quality changes projected to occur nationwide due to ozone control measures applied in the ozone cost analysis (see chapter 7). This clinical benefits analysis uses a slightly different procedure for estimating benefits compared to chapter 12. The clinical benefits model does not reflect potential benefits associated with projected air quality changes outside the identified ozone nonattainment and identified transport areas. Although control measures applied inside the ozone attainment areas are projected to affect air quality outside of the nonattainment area boundaries, the clinical model is data-intensive (hourly ozone data for a full calendar year for each county in the continental U.S.).

A test run of the model showed that benefits estimated for nationwide ozone air quality changes provided benefits results only slightly higher (five percent) when compared to benefits estimates calculated only for air quality changes within the ozone nonattainment areas. (Mathtech, 1997) Based on this slight difference, a decision was made to apply the model only

to air quality changes within the nonattainment areas. Given this methodology, the benefits presented here are slightly underestimated due to the limited geographic scope.

The results of this clinical model benefits analysis are presented in Tables I.9 and I.10. The quantified reductions in health effects are presented in Table I.9 while the monetized benefits associated with those reductions are presented in Table I.10. These results cannot be combined with the benefits results presented in Chapter 12, which use epidemiological models to estimate benefits. Some overlap exists between coughs and some of the epidemiologic-measured endpoints such as hospital admissions, respiratory symptoms, or bronchitis. The same concern applies to the other clinical study endpoints.

**Table I.9 Outdoor Workers, Children, and Rest of Adult Population**  
 Partial Attainment  
 Incidence-Days Incremental from the Current Ozone NAAQS  
 (Year = 2010)

<b>Endpoint</b>	<b>0.08, 5th max</b>	<b>0.08, 4th max.</b>	<b>0.08, 3rd max.</b>
DFEV <sub>≥</sub> 10%	2,901,420	2,926,986	3,504,604
DFEV <sub>≥</sub> 15%	2,088,001	2,096,770	2,440,114
DFEV <sub>≥</sub> 20%	1,011,808	1,006,651	1,129,725
Any Cough	2,223,280	2,216,085	2,464,995
Moderate to Severe Cough	329,761	320,525	331,801
Pain Upon Deep Inhalation	6,081,851	6,155,051	7,462,323
Moderate to Severe PDI	455,712	447,848	477,422
Lower Resp. Symptoms	144,160	140,773	134,825
Moderate to Severe Lower Resp. Symp.	0	0	0

**Table I.10 Outdoor Workers, Outdoor Children, Rest of Adult Population**  
 Partial Attainment  
 Incremental from the Current Ozone NAAQS  
 (millions of 1990\$ ; year = 2010)

<b>Endpoint</b>	<b>0.08, 5th max.</b>	<b>0.08, 4th max.</b>	<b>0.08, 3rd max.</b>
DFEV <sub>≥</sub> 10%	n/e	n/e	n/e
DFEV <sub>≥</sub> 15%	n/e	n/e	n/e
Any Cough	\$15.563	\$15.513	\$17.255
Moderate to Severe Cough	n/e	n/e	n/e
Pain Upon Deep Inhalation	\$26.821	\$27.144	\$32.909
Moderate to Severe PDI	n/e	n/e	n/e
Lower Resp. Symp.	n/e	n/e	n/e
Mod. To Severe Lower Resp. Symptoms	n/e	n/e	n/e
Total Monetized Benefits	\$42.384	\$42.656	\$50.164

n/e = not estimated



## I.6 REFERENCES

- Chestnut, L.G. 1995. *Dollars and Cents: The Economic and Health Benefits of Potential Particulate Matter Reductions in the United States*. Prepared for the American Lung Association.
- Ehrlich, I., and H. Chuma. 1990. A Model of the Demand for Longevity and the Value of Life Extension. *Journal of Political Economy* 98(4): 761-782.
- Industrial Economics, Incorporated (IEc). 1994. Memorandum to Jim DeMocker, Office of Air and Radiation, Office of Policy Analysis and Review, U.S. Environmental Protection Agency, March 31.
- Jones-Lee, M.W., et al. 1985. The Value of Safety: Result of a National Sample Survey. *Economic Journal* 95 (March): 49-72.
- Krupnick, A.J. and M.L. Cooper (1992), The Effect of Information on Health Risk Valuations. *Journal of Risk and Uncertainty* 5(2):29-48.
- Mathtech, Inc. (1997) Technical Support Document for Ozone NAAQS Analysis: Benefit Methodology. Prepared for Science Application International Corporation. July.
- Neumann, J. and R. Unsworth (1993). Review of Existing value of Morbidity Avoidance Estimates: Draft Valuation Document, Memorandum prepared by Industrial; Economics, Incorporated for U.S. EPA, Office of Policy Analysis and Review, September 30, 1993.
- Rowe, R.D. and L.G. Chestnut. 1986. Oxidants and Asthmatics in Los Angeles: A Benefits Analysis--Executive Summary. Prepared by Energy and Resource Consultants, Inc. Report to the U.S. EPA, Office of Policy Analysis. EPA-230-09-86-018. Washington, D.C. March
- Rowe, R.D. and T.N. Neithercut. 1987. *Economic Assessment of the Impacts of Cataracts*. Prepared for U.S. Environmental Protection Agency, Benefits Branch-OPPE by Energy and Resource Consultants, Inc., Boulder, Colorado.
- Rowe, R.D., L.G. Chestnut, and W.D. Shaw. 1984. Oxidants and Asthmatics in Los Angeles: A Benefits Analysis. *Evaluation of the Ozone/Oxidants Standards*. Si Duk Lee (ed.) Air Pollution Control Association. Houston. pp. 366-392.
- Schwartz, J., and D.W. Dockery. 1992. Increased mortality in Philadelphia associated with daily air pollution concentrations. *Am. Rev. Respir. Dis.* 145: 600-604.
- U.S. Environmental Protection Agency (1997); Benefits Technical Support Document for

Regulatory Impact Analysis for the Particulate Matter. Office of Air Quality Planning and Standards; Research Triangle Park, N.C.

Violette, D.M. and L.G. Chestnut. 1983. *Valuing Reduction in Risks: A Review of the Empirical Estimates*. Report prepared for the U.S. Environmental Protection Agency, Washington, D.C. EPA-230-05-83-002.

Viscusi, W.K. 1992. *Fatal Tradeoffs: Public and Private Responsibilities for Risk*. (New York: Oxford University Press).

Viscusi, W.K., Magat, W.A., and Huber, J. 1991. Pricing Environmental Health Risks: Survey Assessments of Risk-Risk and Risk-dollar Tradeoffs. *Journal of Environmental Economics and Management* 201: 32-57.