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Human Health Benefits From Sulfate Reductions Under Title IV Of The 1990 Clean Air Act Amendments

HUMAN HEALTH BENEFITS FROM SULFATE REDUCTIONS UNDER TITLE IV OF THE 1990 CLEAN AIR ACT AMENDMENTS

Final Report

Prepared for:

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ABBREVIATIONS

$\mu g/m^3$	micrograms/cubic meter
AOD	Airway Obstructive Disease
ASD	asthma symptom day
BAD	bad asthma day
CEUM	Coal and Electric Utilities Model
CHA	cardiac hospital admission
COH	coefficient of haze
COI	cost of illness
GIS	geographic information system
H^+	hydrogen ion
H_2SO_4	sulfuric acid
HIS	Health Interview Survey
LRS	lower respiratory symptom
MRAD	minor restricted activity day
NAAQS	National Ambient Air Quality Standards
NAPAP	National Acid Precipitation Assessment Program
NH_4HSO_4	ammonium bisulfate
$(NH_4)_2HSO_4$	ammonium sulfate
NO_x	nitrogen oxide
PM_{10}	particulate matter with an aerodynamic diameter of 10 microns or less
$PM_{2.5}$	particulate matter with an aerodynamic diameter of 2.5 microns or less
ppm	parts per million
RAD	restricted activity day
RADM	Regional Acid Deposition Model
RHA	respiratory hospital admission
RRAD	respiratory restricted activity day
SO_2	sulfur dioxide
SO_4	sulfate
TSP	total suspended particulates
U.S. EPA	United States Environmental Protection Agency
VOC	volatile organic carbon
VSL	value of a statistical life
WTP	willingness to pay

EXECUTIVE SUMMARY

S.1 BACKGROUND

Title IV of the Clean Air Act Amendments of 1990 calls for a 10 million ton reduction in annual emissions of sulfur dioxide (SO_2) in the United States by the year 2010, which represents an approximately 40 percent reduction in anthropogenic emissions from 1980 levels. Implementation of Title IV is referred to as the Acid Rain Program; the primary motivation for this section of the Clean Air Act Amendments is to reduce acid precipitation and dry deposition.[1] This assessment has been prepared at the request of the U.S. Environmental Protection Agency (U.S. EPA), Acid Rain Division, to quantify the expected human health benefits associated with the SO_2 emissions reductions required under the Acid Rain Program. This assessment is intended to contribute to assessments of costs and benefits of the Clean air Act, such as the studies called for under Sections 812 and 901 of the 1990 Amendments. The Act requests that benefits and costs be quantified to the extent possible given available scientific and economic information. This report, therefore, focuses on quantification of potential health benefits of Title IV in both numbers of specific health effects expected to be reduced and their monetary valuation.

This report provides estimates of the human health benefits expected to result from changes in ambient sulfate aerosol concentrations in the eastern United States. Title IV requirements are expected to result in significant reductions in SO_2 emissions in the eastern United States.[2] This will mean lower gaseous SO_2 concentrations close to major emissions sources, lower sulfate aerosol concentrations (including acid and nonacid aerosols) throughout the region, and lower acid precipitation throughout the region. This report focuses on ambient sulfate aerosols because the potential human health benefits of this pollutant reduction have not been fully quantified in previous analyses, because the potential human health benefits are substantial, and because a quantitative assessment is feasible for sulfate aerosols, given available scientific and economic information. This report does not attempt to quantify various other possible human health benefits of Title IV, such as those that might result from nitrogen oxide reductions and "piggy back" toxics or particulate reductions.

[1] Throughout this report the terms "acid rain" and "acid precipitation" include dry deposition.

[2] SO_2 emissions are also controlled under Title I of the Clean Air Act.

S.2 METHODS

Sulfate aerosols are a substantial share of total ambient fine particulate matter in the eastern United States. A large body of epidemiology literature examines the relationship between ambient particulate matter and health effects. Some of these studies have specifically examined sulfate aerosols, and many have examined more broad measures of particulate matter such as $PM_{2.5}$ (particulate matter with aerodynamic diameter of 2.5 microns or less) or PM_{10} (particulate matter with aerodynamic diameter of 10 microns or less). Scientific debate and uncertainty continue concerning the extent to which sulfates may or may not be the key causative constituent of this observed association between health effects and particulate matter. Sulfate aerosols, and especially that portion of sulfate aerosols that is acidic, continue to be considered one of the likely causative agents in the observed association between particulate matter and health effects in the eastern United States. In this assessment, the available epidemiology evidence is applied on the presumption that sulfate aerosols are at least a contributing causative constituent of $PM_{2.5}$. This assessment does not assume that sulfate aerosols are the only causative constituent of $PM_{2.5}$.

This assessment also relies on available economic information for estimates of willingness to pay (WTP) for changes in risks of specific health effects. Economic values for changes in risks of human health effects should reflect the full costs to the affected individual and to society. The full costs of an adverse health effect include financial losses such as medical expenses and lost income (referred to as the cost of illness), plus less tangible costs such as pain and discomfort, restrictions on nonwork activities, and inconvenience to others. WTP, as a monetary measure for a change in health risk, is defined as the dollar amount that would cause the affected individual to be indifferent to experiencing an increase in the risk of the health effect or losing income equal to that dollar amount. WTP measures of monetary value for changes in health risks thus exceed health care and other out-of-pocket costs that are associated with illness or premature death, because WTP reflects these as well as other less tangible effects of illness or premature death on a person's quality of life.

Table S-1 lists the five major quantification steps in this assessment and gives a brief explanation of the quantification method selected for each step. Other related assessments are ongoing at the U.S. EPA, such as the Section 812 studies concerning the costs and benefits of the Clean Air Act Amendments as a whole and the review of the National Ambient Air Quality Standards (NAAQS) for particulate matter. Although there are many similarities in the general approaches being taken in the health benefits components of these other assessments and in this assessment for Title IV, many of the details of the assessment methods may differ. Many of these differences stem from the fact that this assessment focuses on SO_2 emissions and sulfate aerosols only, while the NAAQS assessment considers all sources of ambient particulate matter and the Section 812 studies consider not only all sources of ambient particulate matter but all air pollutants regulated under the Clean Air Act.

Table S-1
Quantification Steps for this Assessment of Health Benefits Due to Sulfate Aerosol Reductions

Quantification Steps		Selected Quantification Method
1.	Changes in SO_2 emissions in the United States	Use ICF Resources (1994) estimates for the United States of 1985 emissions, 1997 emissions with Title IV, and 2010 emissions with and without Title IV (prepared for EPA)
2.	Changes in atmospheric sulfate aerosol concentrations in the eastern United States and eastern Canada	Use EPA's Regional Acid Deposition Model (RADM) runs for each of the SO_2 emissions scenarios
3.	Numbers of people residing at each location where atmospheric sulfate concentrations change in the eastern United States and Canada	Match the RADM 80 km × 80 km grid to population data using a Geographic Information System; population based on 1990 Census data for block groups (Chapter 3)
4.	Changes in sulfate-related health effects: changes in numbers of cases of each type of health effect	Use concentration-response functions derived from selected epidemiology studies on health effects of sulfates or $PM_{2.5}$ (Chapter 4)
5.	Monetary valuation of changes in health	Use selected willingness-to-pay estimates from the available economics literature for changes in health risks or health effects (Chapter 5)

S.3 RESULTS

Table S-2 summarizes the estimates of annual human health benefits for the sulfate aerosol reductions attributed to Title IV in 1997 and 2010 for the 31-state eastern United States area. Table S-3 gives the results for Ontario and Quebec. These estimates are based on the default quantification assumptions, some of which are changed in the sensitivity analyses discussed below. The mean total annual estimated health benefit (in 1994 U.S. dollars) for 1997 in the United States is $10.6 billion, and rises to $40.0 billion by the year 2010, when Title IV requirements are expected to be fully implemented.

The health benefit estimates are dominated by premature mortality and chronic bronchitis. The numbers of cases in these health effects categories are relatively small, but the high monetary values per case result in large monetary benefits for these categories. Premature mortality reductions account for about 88 percent of the total health benefits. Chronic bronchitis reductions are an additional 9 percent of the total. Together they represent about 97 percent of the total estimated health benefits.

The largest numbers of cases reduced are for asthma symptom days, restricted activity days, and days with acute lower respiratory symptoms. The restricted activity days are net of days in the hospital and asthma symptom days because these health effects categories may substantially overlap. The lower respiratory symptom days are net of the fraction of restricted activity days that might also be attributed to lower respiratory symptoms. In 2010, the estimated reduction in the number of asthma symptom days because of Title IV is about 6 million in the eastern United States; net restricted activity days prevented is about 9 million; and the estimated number of days with acute lower respiratory symptoms prevented, net of restricted activity days, is about 19 million. Together, these represent about 3 percent of the total monetary health benefits.

Estimates of reductions in health effects in Canada are based on estimates of changes in sulfate aerosol concentrations in Canada predicted to result from changes in SO_2 emissions generated in the United States. The estimated benefits for Canada occur primarily in the Windsor-Quebec corridor, where the greatest share of the Canadian population likely to be affected by the transport of SO_2 emissions from the eastern United States is located. The estimates for Canada represent an additional 9 percent of the Title IV benefits in 1997 estimated for the United States population. The estimates for Canada do not increase substantially from 1997 to 2010 presumably because the upwind locations in the United States that affect this area of Canada see their greatest reduction in SO_2 emissions in the first phase of the Title IV program. In 2010, the estimates for Canada add an additional 2 percent to the 2010 estimates for the United States population.

There are many sources of uncertainty and potential error in the mean estimates of health benefits for Title IV reported. Table S-4 shows results of some specific sensitivity analyses conducted to determine the potential effect on results of different assumptions than those selected for the mean estimates. The uncertainty and sensitivity analyses reported here cover only the uncertainties in the concentration-response functions and in the monetary valuation of health effects. Additional uncertainties also exist in the estimates of changes in SO_2 emissions and ambient sulfate concentrations that are used as inputs to the health benefits estimates.

The uncertainty and sensitivity analyses reported here are those that are reasonably amenable to quantitative treatment. It is important to recognize that there are many sources of uncertainty that are not possible to quantify, and that these sensitivity tests are therefore not a comprehensive treatment of all possible sources of uncertainty. What these tests provide, however, is an indication of how the results might change if we found that some of the key default assumptions in the health effects quantification and valuation procedures were inappropriate.

Most of the selected concentration-response and monetary value estimates are based on statistically derived results. These estimates therefore have some quantified statistical uncertainty based on the estimated statistical variance in the results. For all of the health effects and monetary value estimates, low and high as well as central estimates were selected

Table S-2
Estimates of Annual Human Health Benefits of Title IV for the Eastern United States with Default Assumptions (millions of 1994 dollars)

Health Effect	1997						2010					
	Annual Number of Cases Prevented			Annual Monetary Value			Annual Number of Cases Prevented			Annual Monetary Value		
	20th Percentile	Mean	80th Percentile	20th Percentile	Mean	80th Percentile	20th Percentile	Mean	80th Percentile	20th Percentile	Mean	80th Percentile
Premature Mortality	408	2,568	5,714	$1,428.0	$9,307.2	$19,999.0	1,539	9,678	21,544	$5,386.5	$35,234.8	$75,404.0
Chronic Bronchitis (new cases)	1,648	3,864	6,590	$507.5	$974.0	$1,377.5	6,179	14,564	24,715	$1,903.0	$3,705.8	$5,165.3
Respiratory Hospital Admissions	663	805	918	$5.7	$11.3	$17.1	2,501	3,036	3,462	$21.5	$42.4	$64.6
Cardiac Hospital Admissions	510	673	867	$4.6	$9.4	$13.9	1,924	2,552	3,270	$17.5	$35.7	$52.5
Asthma Symptom Days	791,232	1,604,341	2,373,697	$20.9	$56.9	$93.2	2,983,490	5,951,693	8,950,470	$78.7	$212.9	$351.3
Restricted Activity Days (net)	1,202,785	2,467,066	3,809,253	$70.6	$147.0	$228.6	4,514,939	9,283,999	14,298,930	$265.0	$554.7	$857.9
Days with Lower Respiratory Symptoms (net)	2,028,424	5,002,393	7,259,946	$31.8	$56.7	$90.0	7,614,168	18,619,000	27,251,920	$119.3	$212.8	$338.0
Total Annual Health Benefits				$3,219.1	$10,562.3	$20,684.1				$12,131.5	$39,999.0	$77,915.5

Table S-3
Estimates of Annual Human Health Benefits of Title IV for Ontario and Quebec, Canada with Default Assumptions (millions of 1994 dollars)

Health Effect	1997						2010					
	Annual Number of Cases Prevented			Annual Monetary Value			Annual Number of Cases Prevented			Annual Monetary Value		
	20th Percentile	Mean	80th Percentile	20th Percentile	Mean	80th Percentile	20th Percentile	Mean	80th Percentile	20th Percentile	Mean	80th Percentile
Premature Mortality	35	217	483	$122.5	$801.2	$1,690.5	37	232	517	$129.5	$839.2	$1,809.5
Chronic Bronchitis (new cases)	140	329	562	$43.3	$83.3	$117.4	150	355	601	$46.3	$91.0	$125.8
Respiratory Hospital Admissions	56	68	78	$0.5	$0.9	$1.4	60	73	83	$0.5	$1.0	$1.6
Cardiac Hospital Admissions	43	57	73	$0.4	$0.8	$1.2	46	61	78	$0.4	$0.9	$1.3
Asthma Symptom Days	66,915	133,825	200,746	$1.8	$4.8	$7.9	71,594	142,267	214,783	$1.9	$5.1	$8.4
Restricted Activity Days (net)	97,734	199,194	309,526	$5.7	$12.0	$18.6	104,568	215,270	331,168	$6.1	$13.0	$19.9
Days with Lower Respiratory Symptoms (net)	164,822	401,231	589,916	$2.6	$4.5	$7.3	176,347	433,821	631,165	$2.8	$4.9	$7.8
Total Annual Health Benefits				$273.3	$907.6	$1,746.9				$290.8	$955.0	$1,868.1

based on the estimated statistical variance and analyst judgment. In general, the selected high and low estimates represent plus and minus approximately one statistical standard error.

It is not appropriate to combine all the "low" estimates or all the "high" estimates to calculate upper and lower bounds on the final estimates, because it is highly unlikely that either all the lows or all the highs would be correct. Such extreme assumptions would significantly overstate the statistical uncertainty in the estimates. Instead, we have assigned probability weights to the low, central, and high estimates which when incorporated in the calculation process allow determination of a probability distribution for the total health benefit results.

The results of this procedure are shown in Tables S-2 and S-3 along with the mean estimates for the estimated annual health benefits of Title IV in 2010 for the eastern United States and Canada. All of these estimates are based on the default assumptions, with each estimate representing a different selected point in the estimated probability distribution calculated for the total health benefits. The 20th percentile of the distribution for 2010 in the eastern United States is about $12 billion in benefits with the default assumptions. This means that 20 percent of the estimated values of benefits are below this amount and 80 percent are above it. The 80th percentile of the distribution is about $78 billion in benefits with the default assumptions. This means that 20 percent of the estimated values of benefits are above this amount and 80 percent are below it.

Each of the sensitivity tests illustrated in Table S-4 represents estimates of mean annual health benefits in 1994 dollars. Each is calculated in the same way that the default mean was calculated, except for the specified assumption change. A comparison with the default mean therefore illustrates the effect of the change in the assumption. There is considerable uncertainty about whether there is a "safe" level of sulfate aerosol exposure that does not cause any harmful health effects. There is no definitive quantitative evidence that such a threshold exists, but neither is there proof that any amount of sulfate aerosol exposure causes some harmful effect in at least some people. We selected alternative threshold assumptions of 5.0 $\mu g/m^3$, 3.6 $\mu g/m^3$, and 1.6 $\mu g/m^3$ annual median sulfate concentrations to illustrate the potential effects of alternative threshold assumptions on the results of this analysis. The results indicate that with a threshold of 5.0 $\mu g/m^3$, annual health benefits are substantially reduced relative to the default mean, falling very close to the 20th percentile default estimate. At thresholds above 5.0 the health benefit estimates would diminish even more. A threshold of 3.6 $\mu g/m^3$ results in a mean health benefit estimate that falls about midway between the default mean and the 20 percentile default estimates. At a threshold of 1.6 (or lower), the health benefit estimate is virtually unchanged from the default mean. This illustrates the significance of the threshold question and shows that this continues to be an important research issue from the standpoint of evaluating the health benefits of pollution emission reductions.

There is a possibility that the sulfate-based concentration-response functions may be somewhat upwardly biased because of the typical collinearity between sulfates and other fine particulate

constituents in the ambient air. For this sensitivity test we multiply the sulfate based concentration-response functions by 0.4, which is the average ratio between measured sulfates and measured $PM_{2.5}$ in the eastern United States. This is the maximum adjustment that would be required if the sulfate coefficients represented the total effects of all $PM_{2.5}$. This adjustment reduces the annual health benefit estimate in 2010 in the eastern United States to about $18.5 billion, which is higher than the 20th percentile estimate with the default assumptions. The true sulfate effect is probably between this and the mean default estimate because the sulfate coefficients probably do reflect some, but are unlikely to reflect all, of the effects of other harmful constituents of $PM_{2.5}$ as well as the effects of sulfates alone.

S.4 CONCLUSIONS

The results of this assessment show that the potential health benefits of reductions in exposures to sulfate aerosols in the eastern United States as a result of the SO_2 emissions reductions required by Title IV are substantial. Based on what we believe is a reasonable interpretation of the available epidemiology and economic evidence on potential health effects of sulfate aerosols and their monetary value, we estimate that the annual health benefits of Title IV required reductions in SO_2 in 2010 in the eastern United States are more likely than not to fall between $12 billion and $78 billion, with an estimated mean value of $40 billion. There is reason to expect some possible upward bias at the higher end of this range, and the results of the sensitivity analyses suggest that there is a good chance that the benefits in 2010 fall between $12 billion and the estimated mean of $40 billion. Annual health benefits for eastern Canada resulting from U.S. reductions in SO_2 emissions would add as much as one billion dollars to the U.S. benefit totals in both 1997 and 2010.

We have been careful throughout the report to highlight key assumptions and uncertainties that exist in the quantification procedures used in this assessment, especially in the health effects quantification and valuation portions of the assessment which are the focus of this report. Most of these uncertainties cannot be resolved without substantial new research on several topics.

Table S-4
Sensitivity Analyses Results

Assumptions	Estimated Annual Health Benefits (billions of 1994 dollars)
United States 1997	
Threshold = 5.0 $\mu g/m^3$ SO_4	$3.1
Threshold = 3.6 $\mu g/m^3$ SO_4	$6.7
Threshold = 1.6 $\mu g/m^3$ SO_4	$10.8
Selected SO_4 Health Risks × 0.4	$4.8
United States 2010	
Threshold = 5.0 $\mu g/m^3$ SO_4	$15.0
Threshold = 3.6 $\mu g/m^3$ SO_4	$28.3
Threshold = 1.6 $\mu g/m^3$ SO_4	$39.3
Selected SO_4 Health Risks × 0.4	$18.5
Canada 1997	
Threshold = 5.0 $\mu g/m^3$ SO_4	$0.0
Threshold = 3.6 $\mu g/m^3$ SO_4	$0.0
Threshold = 1.6 $\mu g/m^3$ SO_4	$0.7
Selected SO_4 Health Risks × 0.4	$0.4
Canada 2010	
Threshold = 5.0 $\mu g/m^3$ SO_4	$0.0
Threshold = 3.6 $\mu g/m^3$ SO_4	$0.0
Threshold = 1.6 $\mu g/m^3$ SO_4	$0.9
Selected SO_4 Health Risks × 0.4	$0.5

CHAPTER 1
INTRODUCTION

1.1 NEED FOR THE ASSESSMENT

Title IV of the Clean Air Act Amendments of 1990 calls for a 10 million ton reduction in annual emissions of sulfur dioxide (SO_2) in the United States by the year 2010, which represents an approximately 40 percent reduction in anthropogenic emissions from 1980 levels. Implementation of Title IV is referred to as the Acid Rain Program; the primary motivation for this section of the Clean Air Act Amendments is to reduce acid precipitation and dry deposition.[1] This assessment has been prepared at the request of the U.S. Environmental Protection Agency (U.S. EPA), Acid Rain Division, to quantify the expected human health benefits associated with the SO_2 emissions reductions required under the Acid Rain Program. This assessment is intended to contribute to the assessments of costs and benefits of the Clean Air Act, such as the studies called for under Section 812 of the 1990 Amendments. The Act requests that benefits and costs be quantified to the extent possible given available scientific and economic information. This report, therefore, focuses on quantification of potential health benefits of Title IV in both numbers of specific health effects expected to be reduced and their monetary valuation.

1.2 PURPOSE OF THE REPORT

This report provides estimates of the human health benefits expected to result from changes in ambient sulfate aerosol concentrations in the eastern United States. Title IV requirements are expected to result in significant reductions in SO_2 emissions in the eastern United States.[2] This will mean lower gaseous SO_2 concentrations close to major emissions sources, lower sulfate aerosol concentrations (including acid and nonacid aerosols) throughout the region, and lower acid precipitation throughout the region. This report focuses on ambient sulfate aerosols because the potential human health benefits of this pollutant reduction have not been fully quantified in previous analyses, because the potential human health benefits are substantial, and because a quantitative assessment is feasible for sulfate aerosols, given available scientific and economic information. This report does not attempt to quantify various other possible human health benefits of Title IV, such as those that might result from nitrogen oxide reductions and "piggy back" toxics or particulate reductions.

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[1] Throughout this report the terms "acid rain" and "acid precipitation" include dry deposition.

[2] SO_2 emissions are also controlled under Title I of the Clean Air Act.

Sulfate aerosols are a substantial share of total ambient fine particulate matter in the eastern United States. A large body of epidemiology literature examines the relationship between ambient particulate matter and health effects. Some of these studies have specifically examined sulfate aerosols, and many more have examined more broad measures of particulate matter such as $PM_{2.5}$ (particulate matter with aerodynamic diameter of 2.5 microns or less) or PM_{10} (particulate matter with aerodynamic diameter of 10 microns or less). Scientific debate and uncertainty continue concerning the extent to which sulfates may or may not be the key causative constituent of this observed association between health effects and particulate matter. Sulfate aerosols, and especially that portion of sulfate aerosols that is acidic, continue to be considered one of the likely causative agents in the observed association between particulate matter and health effects in the eastern United States. In this assessment, the available epidemiology evidence is applied on the presumption that sulfate aerosols are at least a contributing causative constituent of $PM_{2.5}$. This assessment does not assume that sulfate aerosols are the only causative constituent of $PM_{2.5}$.

This assessment also relies on available economic information for estimates of willingness to pay (WTP) for changes in risks of specific health effects. Economic values for changes in risks of human health effects should reflect the full costs to the affected individual and to society. The full costs of an adverse health effect include financial losses such as medical expenses and lost income (referred to as the cost of illness), plus less tangible costs such as pain and discomfort, restrictions on nonwork activities, and inconvenience to others. WTP, as a monetary measure for a change in health risk, is defined as the dollar amount that would cause the affected individual to be indifferent to experiencing an increase in the risk of the health effect or losing income equal to that dollar amount. WTP measures of monetary value for changes in health risks thus exceed health care and other out-of-pocket costs that are associated with illness or premature death, because WTP reflects these as well as other less tangible effects of illness or premature death on a person's quality of life.

1.3 CONTEXT OF HEALTH BENEFITS

Health effects benefits due to reductions in ambient sulfate aerosols, which are the focus of this report, are just one category of potential benefits due to Title IV. The potential benefits of the Title IV provisions include a wide range of environmental impacts, including improvements or reductions in:

- human health effects
- effects on aquatics ecosystems, including effects on recreational fishing
- visibility aesthetics
- effects on materials
- effects on terrestrial ecosystems, including effects on forests and crops.

Each of these effects involves complex chemical, atmospheric, biological, psychological, and economic processes. Some of these processes are fairly well understood at this time and others are not. A practical and policy-relevant assessment must recognize the complexities and uncertainties inherent in current scientific knowledge of these processes, but it must also synthesize, simplify, and interpret available information into conclusions that will be useful for policy-makers.

CHAPTER 2
OVERVIEW OF THE ASSESSMENT

2.1 SCHEMATIC OF THE ASSESSMENT

Figure 2-1 shows the processes involved in the formation of acid deposition. This report focuses on SO_2 emissions. SO_2 is a gas that is released when fuels containing sulfur, such as coal, are combusted. SO_2 interacts with other elements in the atmosphere to form secondary sulfate aerosols.[1] The resulting sulfate aerosols are called secondary pollutants because they are not emitted directly, but are formed later.[2] The transformation into sulfate aerosols begins within fairly short distances from the source. Sulfate aerosols can be transported long distances through the atmosphere before deposition occurs. Some of them are acidic sulfate aerosols, which are a primary constituent of acid deposition in the eastern United States.

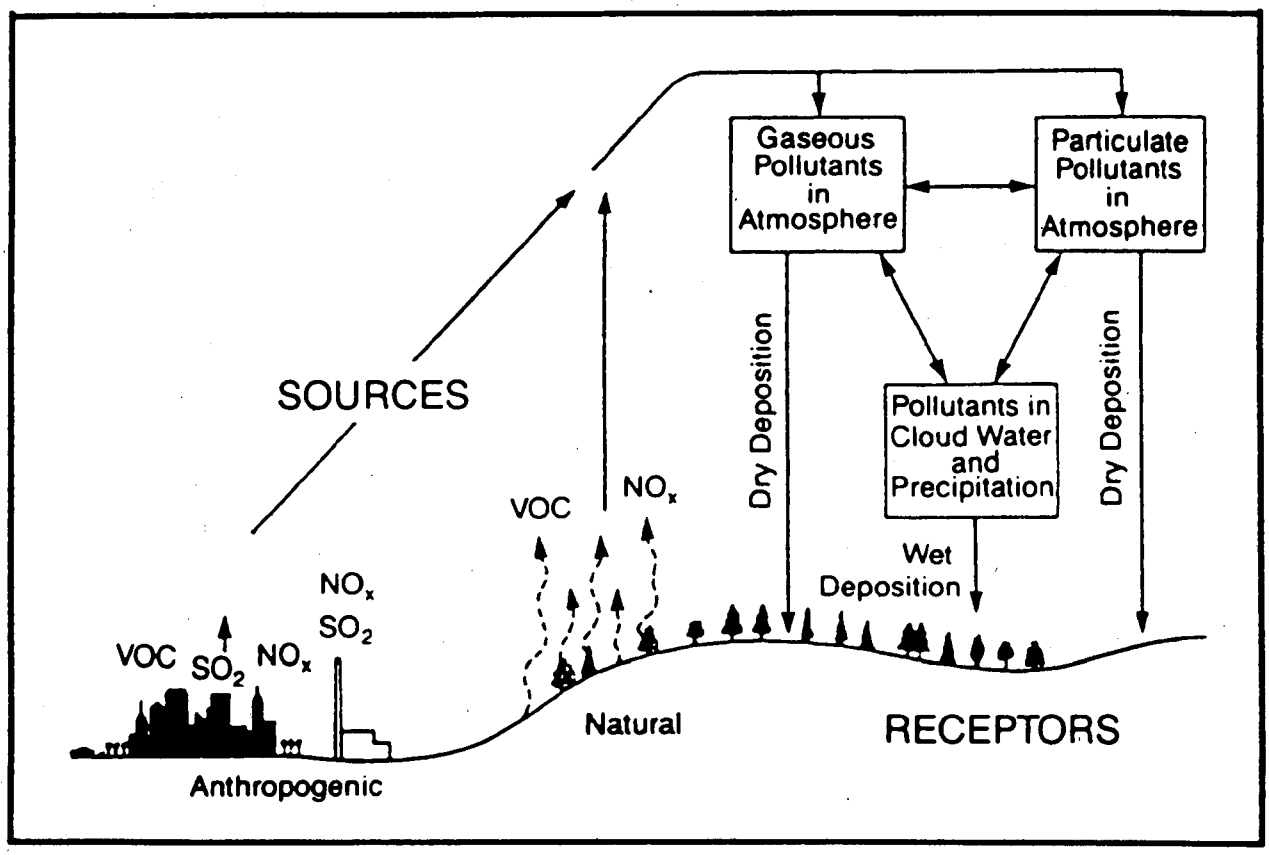
Figure 2-2 shows the relationships among the most common measures of particulate matter in the atmosphere. Different measures are used in different contexts, and many of these terms are used throughout this report. Total suspended particulates (TSP) represent all airborne particulate matter. Particulate matter under 10 microns in aerodynamic diameter (PM_{10}) are particles small enough to be inhaled into the airways of the lungs. PM_{10} is sometimes called thoracic particulate matter. A smaller size category for particulate matter is fine particles, which are particles with aerodynamic diameter of 2.5 microns or less ($PM_{2.5}$).

Most sulfate aerosols are part of $PM_{2.5}$ and most acid aerosols, in the particle phase, are sulfate aerosols. The term acid aerosol is often used to refer to all airborne acids, including those in the vapor phase such as nitric acid (NAPAP, 1991). Such vapors are outside the definition of any of these particle measures. All acidic sulfate aerosols are particles rather than vapors. Sulfate aerosols make up the largest single component of fine particulate matter in most locations in the eastern United States. Measures of average sulfate aerosol concentrations are about 40 percent of measures of average fine particulate matter levels in the eastern

[1] An aerosol consists of liquid or solid particles in air.

[2] Some sulfate aerosols are emitted directly from combustion sources. These are called primary sulfate aerosols, but they make up a very small percentage of total ambient sulfate aerosols.

Figure 2-1
Processes Involved in Acid Deposition



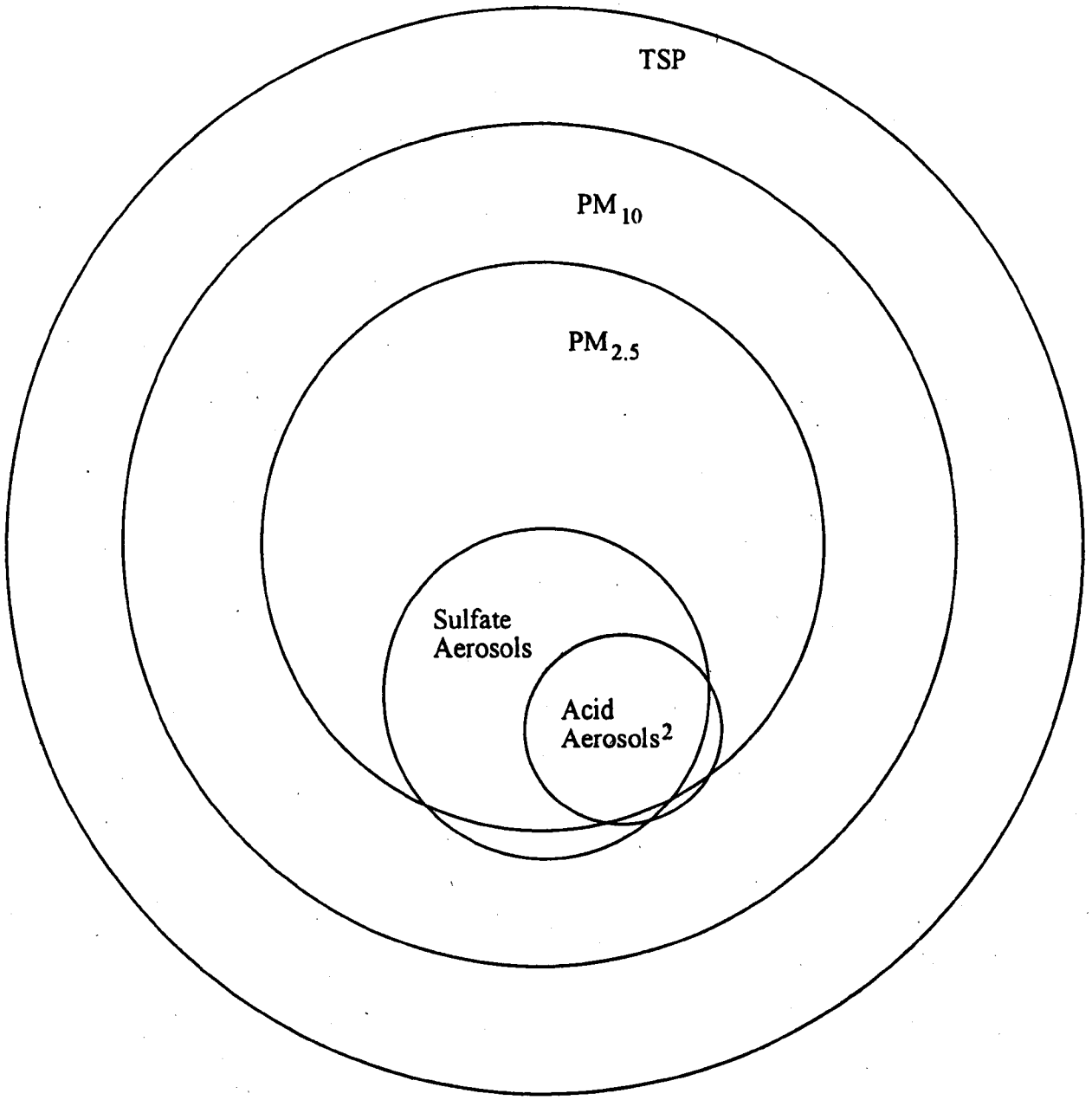
Source: National Acid Precipitation Assessment Program (NAPAP), 1991, p. 174.

United States (Dockery et al., 1993)[3]. Sulfate concentrations are lower in most of the western United States, where fuels with lower sulfur content are more commonly used.

Figure 2-3 shows an overview of the major pathways by which SO_2 emissions may cause human health effects. A comprehensive quantitative assessment of the human health benefits of Title IV must analyze each of these pathways. The Title IV requirements will result in reductions in SO_2 emissions, relative to what would have been emitted in the absence of Title

[3] The 1995 review draft of the PM Criteria Document (US EPA, 1995) reports an average ratio of 0.47 in the eastern U.S. and 0.37 in the central U.S. Our definition of "eastern" includes 31 states, some of which fall in what is commonly called "central" U.S. The 0.4 estimate is therefore reasonably consistent.

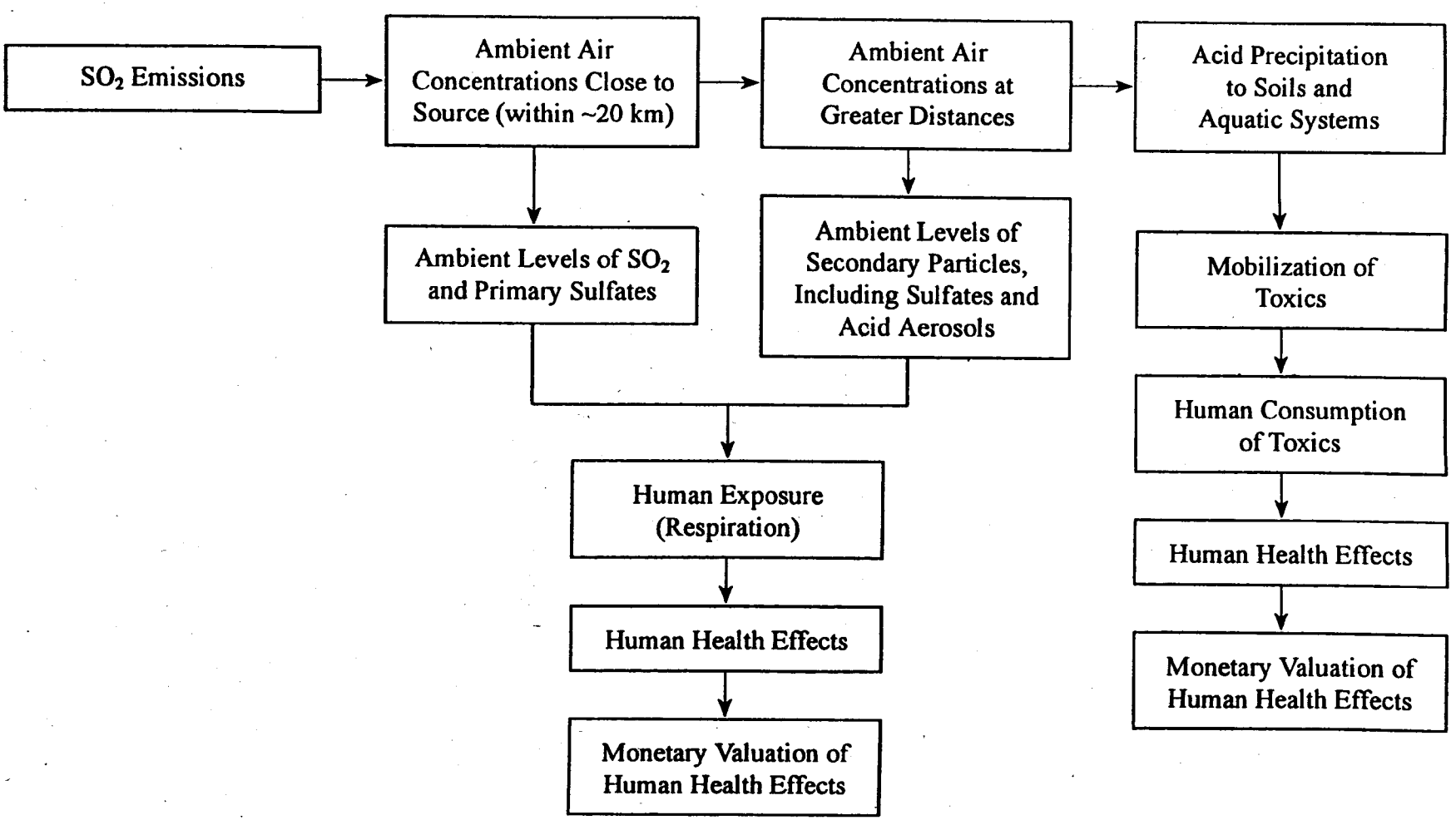
Figure 2-2
Alternative Measures of Particulate Matter in the Atmosphere



1 This figure shows the overlaps in the different measures, but is not drawn to scale in terms of typical relative proportions in the atmosphere. Such proportions vary from place to place and time to time.

2 The term acid aerosols has been used to refer to acids present in the atmosphere in the vapor phase such as nitric acid (NAPAP 1991). Such vapors fall outside the definition of any of these particulate measures. In rare circumstances, such as in the formation of acid fogs, acid aerosols can become larger than PM_{10} (NAPAP 1991).

Figure 2-3
Overview of Human Health Effects Resulting from SO_2 Emissions



IV. Changes in SO_2 emissions result in changes in human exposures to potentially harmful substances in the ambient air, both near and far from the SO_2 source, and through the effects of acid deposition on mobilization of toxic substances in soils and water.

An important point illustrated in Figure 2-3 is that when SO_2 emissions are reduced, potential benefits to human health occur along several avenues. Reductions in ambient air levels of gaseous SO_2 and sulfate aerosols mean reductions in these potentially harmful pollutants in the air that people breathe. Once the sulfate aerosols are deposited on soils and aquatic systems, the acidic portion of these aerosols can contribute to the mobilization of toxic substances already present in the environment. A reduction in acid deposition thus means a reduction in the chance that these substances will be present in the water and food that humans consume.

For reasons discussed in subsequent sections of this chapter, this report focuses on the human health benefits of the expected reductions in exposure to atmospheric sulfate aerosols caused by the Title IV required SO_2 emissions reductions. Table 2-1 lists the five quantification steps in this assessment and gives a brief explanation of the quantification method selected for each step. Some of the rationale for selecting these methods is explained in subsequent sections of this chapter. Subsequent chapters explain the selected quantification methods in detail.

Other related assessments are ongoing at the U.S. EPA, such as the Section 812 studies concerning the costs and benefits of the Clean Air Act Amendments as a whole and the review of the National Ambient Air Quality Standards (NAAQS) for particulate matter. Although there are many similarities in the general approaches being taken in the health benefits components of these other assessments and in this assessment for Title IV, many of the details of the assessment methods may differ. Many of these differences stem from the fact that this assessment focuses on SO_2 emissions and sulfate aerosols only, while the NAAQS assessment considers all sources of ambient particulate matter and the Section 812 studies consider not only all sources of ambient particulate matter but all air pollutants regulated under the Clean Air Act.

The results of this health benefit assessment based on the selected default assumptions are reported in two ways. First, they are reported as annual estimates for the years 1997 and 2010. Title IV, Phase I, is expected to be implemented by 1997, and Title IV is expected to be fully implemented by 2010. The estimated 1997 sulfate concentrations without Title IV are based on Regional Acid Deposition Model (RADM) runs for 1985 emissions estimates, assuming no significant change from 1985 to 1997 in the absence of Title IV. RADM results for 1997 estimated SO_2 emissions with Title IV are compared to 1985 RADM results to calculate Title IV health benefits in 1997. The 2010 estimates are based on ICF Resources estimates of SO_2 emissions with and without Title IV for 2010 and on estimates of ambient sulfate aerosol concentrations from RADM for each of the 2010 emissions scenarios. The annual estimates are based on 1990 population and income levels and are reported in 1994 dollars.

Table 2-1
Quantification Steps for this Assessment of
Health Benefits due to Sulfate Aerosol Reductions

Quantification Steps		Selected Quantification Method
1.	Changes in SO_2 emissions in the United States	Use ICF Resources (1994) estimates of 1985 emissions, 1997 emissions with Title IV, and 2010 emissions with and without Title IV (prepared for EPA)
2.	Changes in atmospheric sulfate aerosol concentrations in the eastern United States	Use EPA's Regional Acid Deposition Model (RADM) runs for each of the SO_2 emissions scenarios
3.	Numbers of people residing at each location where atmospheric sulfate concentrations change in the eastern United States and Canada	Match the RADM 80 km x 80 km grid to population data using a Geographic Information System; population based on 1990 Census data for block groups (Chapter 3)
4.	Changes in sulfate-related health effects: changes in numbers of cases of each type of health effect	Use concentration-response functions derived from selected epidemiology studies on health effects of sulfates or $PM_{2.5}$ (Chapter 4)
5.	Monetary valuation of changes in health	Use selected willingness-to-pay estimates from the available economics literature for changes in health risks or health effects (Chapter 5)

Second, the results are reported as 1995 present value estimates of the total health benefits expected from 1997 though 2010. Health benefits due to Title IV for the years between 1997 and 2010 are interpolated from the RADM-based estimates in proportion to the emissions estimates available for the years between 1997 and 2010 for the scenarios with and without Title IV. Aggregate estimates of total health benefits are reported undiscounted and discounted with two alternative discount rates, both adjusted for average expected population and real income growth.

2.2 UNCERTAINTY AND SENSITIVITY ANALYSES

Any quantitative assessment of this nature is subject to considerable uncertainty due to the complexities of the physical and economic processes involved, and limits in our technical capabilities to fully characterize current interactions and predict future changes. It is important that analysts attempt to characterize the uncertainty in the results of such an assessment so

that policy makers can give appropriate consideration to the results in their decision making processes. This report addresses uncertainty in the following ways:

- Limitations and assumptions in the quantification process are clearly stated and explained.
- A quantitative uncertainty analysis is conducted based on estimated statistical variance in some of the underlying relationships upon with the assessment is based.
- Sensitivity analyses illustrate the effects of changing key default assumptions on the mean results of the assessment.

There are many different valid ways to characterize and present quantitative uncertainty in an assessment of this type. This assessment has used an approach very similar to that developed by Rowe et al. (in press) in a quantitative model to estimate environmental effects of electricity generation in New York. The quantitative uncertainty analysis is based on variations in results within and across selected studies, but specific results are selected as most likely correct and are given probability weights that reflect some analyst judgment as well as empirical evidence.

2.2.1 Quantitative Uncertainty Analysis

The available epidemiology and economics evidence regarding health effects associated with air pollutants is subject to considerable uncertainty. Within a given study there is statistically measurable uncertainty in the estimated concentration-response coefficients or monetary value estimates, and there are differences in results obtained from different studies looking at the same or similar health effects. This assessment uses a quantitative uncertainty analysis similar to the approach developed by Rowe et al. (in press). For each concentration-response relationship and each monetary value estimate presented in this report, low, central, and high estimates are selected. The central estimate is typically selected from the middle of the range reported in the study, or group of studies, that has been selected as providing the most reliable results for that health effect based on the study selection criteria.

These ranges of estimates are not intended to reflect absolute upper and lower bounds, but rather they are ranges of estimates that are reasonably likely to be correct, given available epidemiology and economics study results. For example, ranges based on a single study are selected as plus and minus one standard error, not the absolute highest or lowest results obtained. When several different "reliable" studies are available for a given health effect, the selected range reflects the variation in results across the studies. The reader should be aware that there is analyst judgment in selecting these ranges and that the ranges do not reflect all the uncertainty in the estimates because some of the uncertainty is not quantifiable. This is,

however, an attempt to give a more realistic presentation than is given when only point estimates are reported.

Each low, central, and high estimate is also assigned a probability weight (the weights summing to 100 percent for each quantified health effect and for each monetary value estimate). These probability weights, combined with the low, central, and high estimates, are used to estimate a probability distribution of the total health benefits estimate, which is calculated by multiplying estimated numbers of health effects by the monetary value per case, and summing across all the health effects categories. Calculating a probability distribution for the total health benefit estimate provides an alternative to simply summing all the low estimates or all the high estimates to obtain total low and high estimates. Such simple summing results in a misleadingly large range of values, because it is highly unlikely that all the low estimates (or all the high estimates) are correct. When the low, central, and high estimates are based on results from different studies all judged as equally reliable, an equal probability weight is given to the low, central, and high estimates. When only one study result is selected, the range selected is often plus and minus one statistical standard error of the selected central result. When a standard error is used, the probability weight given to the central estimate is 50 percent, with 25 percent each to the high and low estimates. In a few cases less weight has been given to a high or low estimate based on analyst judgment that there is reason to suspect that particular estimate is less likely to be correct than the other available estimates.

Mean, low, and high values for changes in cases of each health effect and for their monetary values were calculated for the estimated change in sulfate concentrations, using the low, central, and high values and the probability weights assigned to each. These calculation were executed using the @RISK supplemental program for such applications with the Lotus 1-2-3 spreadsheet program (Palisade Corp., 1994). This program selects a sample of all the possible combinations of low, central, and high estimates sufficient to estimate a probability distribution for the total health benefit estimate. From this estimated distribution, we have selected low and high values that represent the 20^{th} percentile and the 80^{th} percentile on the probability distribution of the total estimated health benefit. This means, for example, that there is an 60 percent probability that the "true" value falls between these low and high results, given the magnitudes and the probabilities selected for each of the low, central, and high concentration-response and monetary value estimates.

2.2.2 Sensitivities to Key Default Assumptions

Throughout the report the assumptions and uncertainties in this analysis are acknowledged. In some cases it is possible to define alternative assumptions and to determine how the results are affected if a default assumption were determined to be incorrect. This is an important process for identifying the most important assumptions with regard to their effect on the bottom line, and the results are reflected in the conclusions of the report.

2.3 RESULTS FROM THE 1990 NAPAP ASSESSMENT

Three categories of potential human health effects associated with SO_2 emissions and subsequent secondary pollutants were considered in the 1990 NAPAP State of the Science and Technology reports and the NAPAP 1990 Integrated Assessment:

- direct health effects of gaseous SO_2
- indirect health effects of toxic chemicals released into the environment as a result of acid deposition
- direct health effects of acid aerosols in the ambient air.

2.3.1 NAPAP Conclusions on the Effects of Gaseous SO_2

SO_2 is a criteria air pollutant under the Clean Air Act, and National Ambient Air Quality Standards (NAAQS) have been set to protect public health and welfare. The current primary NAAQS for SO_2 are:

- annual average of 0.03 ppm
- 24-hour average of 0.14 ppm.

Ambient concentrations of SO_2 have been substantially reduced in the United States since 1970, and most of the population now lives in areas that meet the primary NAAQS. Remaining nonattainment areas are limited to geographical areas in the immediate vicinity of a few major point sources.

Much of the recent SO_2 health effects research has focused on acute exposures of asthmatics, who are believed to be more sensitive to SO_2 than other people. Aggravation of asthma symptoms in some individuals who are exercising and who already have asthma has been demonstrated in clinical studies with short-term SO_2 exposures at concentrations close to those that occasionally occur currently in some locations in the United States. Graham et al. (1990) cite conclusions reached by the U.S. EPA that at current SO_2 emission levels in the United States, the only health effect of any concern due to short-term peaks of ambient SO_2 concentrations is the aggravation of asthma symptoms in exercising asthmatics.

NAPAP (1991) reported the U.S. EPA's conclusions that SO_2 concentrations high enough to cause well-documented short-term effects on individuals with asthma currently occur only within about 12 km of a few major point sources in the United States. Graham et al. (1990) report that approximately 100,000 exercising asthmatics may be exposed once each year to SO_2 concentrations high enough and for long enough to cause a reaction in some asthmatics (0.5 ppm for 5 minutes was the assumption used). Graham et al. cite clinical evidence that

approximately 25 percent of asthmatic subjects may have a doubling of airways resistance while exercising when exposed to 0.5 ppm of SO_2. Not all reactive asthmatics will have symptoms severe enough to be noticeable to them. More recent evaluations (U.S. EPA, 1994) indicate that only about 10 to 20 percent of mild or moderate asthmatics are likely to exhibit lung function decrements in response to SO_2 exposures of 0.2 to 0.5 ppm during moderate exercise that would be of distinctly larger magnitude than typical daily variations in lung function or average changes in lung function experienced in response to other often encountered stimuli (e.g., cold/dry air, moderate exercise, etc.). A more substantial percentage (20 to 25 percent) of such asthmatics exposed to 0.6 to 1.0 ppm of SO_2 experience respiratory function decrements and severity of respiratory symptoms that exceed typical daily variations or response to other commonly encountered stimuli that produce short-lived bronchoconstrictor effects like SO_2.

A further reduction in SO_2 emissions, beyond current levels, due to Title IV means that this health effect can be expected to be reduced. Because of the limited geographic scope of this effect however, the economic benefit of reducing this effect is relatively small. If we assume an average monetary value of $34 (see Chapter 5) for preventing a day with aggravated asthma symptoms, the annual aggregate value of preventing this effect would be no more than $1,000,000 even if all 25,000 affected asthmatics have noticeable symptoms and if the Title IV emission reduction eliminates all of this negative health effect.

The analysis and conclusions reported by NAPAP appear to be sufficient for estimating an upper bound on the likely benefits of Title IV due to reductions in short-term effects of peak SO_2 exposures on exercising asthmatics. This category of health benefits for Title IV appears to be relatively small and is fairly well established. It does not appear to warrant further quantitative analysis at this time.

2.3.2 NAPAP Conclusions Regarding Indirect Health Effects of Acid Deposition

NAPAP (1991) provides a summary of the analysis and conclusions reported by Grant et al. (1990) of potential indirect human health effects due to acid deposition. The pathway for such potential effects is illustrated on the right-hand side of Figure 2-2. The mechanism is that acid deposition can cause potentially harmful substances already present in soils or aquatic systems to be mobilized. These substances may then ultimately be consumed by humans through food or water. Such consumption in sufficient quantity may cause adverse health effects.

Grant et al. (1990) assessed the likelihood that current levels of acid deposition could be associated with significant human health effects as a result of the mobilization of methylmercury, lead, cadmium, arsenic, aluminum, copper, selenium, and asbestos. This assessment was made difficult by complexities and uncertainties about the physical processes, the multiple sources of these substances in the environment, and human exposures. The

conclusions are therefore tentative, but they appear to be reasonable given currently available information.

Grant et al. (1990) concluded that at current acid deposition levels, lead and methylmercury are the only substances considered that may be causing measurable health effects as a result of acid deposition. Some subpopulations of individuals are already exposed to high levels of these compounds because of circumstances unrelated to acid deposition, and it is feasible that further exposure due to the mobilization effects of acid deposition might result in adverse health effects. For lead, critical health effects include slowed fetal physical and neurological development, neurobehavioral deficits in young children, including decreased IQ, and hypertension in adults. Critical health effects due to methylmercury include fetal psychomotor retardation and paresthesia in adults.

Only a small segment of the population is likely to be at any appreciable risk because of incremental lead or methylmercury exposures as a result of current levels of acid deposition. The population segments judged to be at some potential risk are as follows:

- Those for whom subsistence fishing is a significant source of food and who fish primarily at acidified lakes may be at risk of harmful effects due to methylmercury in fish. High concentrations of methylmercury have been measured in fish at acidified lakes in the upper Midwest and Northeast.

- Young children and developing fetuses within pregnant women who consume acidified drinking water (without pH or corrosivity treatment) may be exposed to potentially harmful concentrations of lead if the soil or water distribution system contains lead that is leached by the acidified drinking water. These are primarily individuals whose drinking water comes not from municipal systems but from rainwater, surface water, or shallow wells.

Grant et al. (1990) estimate that the first group may contain as many as 10,000 individuals and that the second group may contain approximately 11,000 children and 29,000 women of childbearing age. Estimates of how many of these individuals might be expected to suffer adverse effects were not made, but clearly it would be some fraction of the total. Although some potential health effects of these substances are severe, the number of people estimated to be at any risk of elevated exposure at current acid deposition levels is small.

Uncertainty about the current extent of these health risks due to acid deposition cannot be reasonably reduced at this time without an investment of very significant research resources. Further quantitative analysis of this category of potential health effects does not appear to be warranted at this time.

2.3.3 NAPAP Conclusions on the Effects of Acid Aerosols

Graham et al. (1990) reviewed the available laboratory, clinical, and epidemiological evidence on the human health effects of acid aerosols. NAPAP (1991) summarized the conclusions of this review, which are that (1) there is evidence of harmful respiratory effects for human subjects exposed to some types of acid aerosols and (2) there is not sufficient information available to conduct a quantitative assessment of the current level of health effects due to acid aerosols in the United States.

Acid aerosols are a mixture of several pollutants. In the eastern United States, the predominant fraction of acid aerosols appears to be acidic sulfates, which include sulfuric acid (H_2SO_4), ammonium bisulfate (NH_4HSO_4), and ammonium sulfate [$(NH_4)_2HSO_4$]. NAPAP (1991) notes that the hydrogen ion (H^+) may be the species of concern with respect to human health, but this remains uncertain. Most of the available laboratory and clinical evidence regarding health effects of acid aerosols focuses on acidic sulfates, especially H_2SO_4. NAPAP summarizes the available clinical and laboratory evidence on acidic sulfates as follows:

- Controlled acute exposures to acidic sulfates can cause decreased lung function and reactivity responses in some asthmatics.
- Controlled acute exposures to acidic sulfates can alter mucociliary clearance of the lungs in nonasthmatic and asthmatic humans. This may affect the ability of the lungs to clear inhaled particles, including infectious organisms.
- Long-term exposures of laboratory animals to acidic sulfates reveal changes related to the development of chronic bronchitis, including reduced mucociliary clearance and morphological changes.

Epidemiology research concerning acid aerosols has been quite limited because of little availability of data on ambient acid aerosol concentrations. NAPAP (1991) notes that there is epidemiology evidence that air pollution mixtures known to contain acid aerosols are associated with both mortality and morbidity, but that it is not possible to determine to what extent this association is due to the presence of acid aerosols. Four aerosol pollution measures that have been used in these types of studies are TSP, PM_{10}, $PM_{2.5}$, and sulfates. NAPAP notes that statistically significant associations between mortality and all of these aerosol measures have been found in macroepidemiological studies, and somewhat more consistency has been found in the results for fine particles and sulfates. The macroepidemiological studies compare average mortality rates across locations with different average pollution concentrations.

The NAPAP 1990 Integrated Assessment appropriately concluded that there is not sufficient information available at this time to conduct a credible quantitative assessment of the health effects of acid aerosols in the United States. This is the result of limited data availability for

current concentrations of acid aerosols, as well as limited quantitative evidence on the specific health effects that might be expected for a given concentration of acid aerosol exposure.

The NAPAP assessment, however, did not address the question of whether a quantitative assessment is feasible for sulfate aerosols in general, rather than for just acidic sulfates. This is really the more relevant question with regard to the health benefits of Title IV, because the required reductions in SO_2 emissions will result in reductions in all sulfate aerosols, not just acidic sulfates.

2.4 FOCUS OF THIS ANALYSIS ON SULFATE AEROSOLS

The focus of this analysis is on the potential human health benefits of the reduction in ambient concentrations of sulfate aerosols, including acidic sulfates, that is expected as a result of the Title IV required reductions in SO_2 emissions. This is the middle path in Figure 2-3. This focus was chosen for four primary reasons:

- A quantitative assessment of the health benefits of reducing ambient sulfate aerosol concentrations is feasible given available information, but has not yet been conducted for the type of change in ambient concentrations expected as a result of Title IV.

- A large available body of epidemiology literature concerning the association between ambient aerosol pollutants, including sulfates, and human health effects allows a quantitative assessment to be performed using a modest amount of research resources.

- The required reduction in SO_2 emissions is substantial relative to current emission levels, and the resulting reduction in ambient sulfate aerosol concentrations is also expected to be substantial.

- Given the potential for reductions in risks of mortality, chronic respiratory disease, and acute morbidity as a result of reductions in sulfate aerosol concentrations, and the long distance and wide ranging dispersion of sulfate aerosols, there is a possibility of substantial health benefits.

The other two branches of Figure 2-3, direct effects of gaseous SO_2 and indirect effects of acid deposition, were examined in detail in the 1990 NAPAP analyses. The results suggest that the number of people at potential risk of health effects due to these pathways under current conditions is fairly limited. The potential that these risks will be reduced as a result of Title IV should not be disregarded in a comprehensive assessment of Title IV benefits, but it does not appear that there are sufficiently different data or analysis approaches available today to warrant further analysis of these potential health effects pathways at this time.

2.5 GENERAL LIMITATIONS OF THE ASSESSMENT

Detailed discussions of the assessment approach, assumptions, and limitations are provided in Chapters 3, 4, and 5. In this section, we introduce and highlight what we believe are the key difficulties, limitations, and uncertainties in this assessment and therefore in the results.

This health benefits assessment relies on results of two other analyses conducted for or by EPA. These are the ICF Resources (1994) estimates of emissions of SO_2 with and without Title IV, and the EPA estimates of resulting ambient sulfate aerosol concentrations using RADM with the ICF emissions estimates as input. Each of these analyses relies on specific applications of detailed models developed for these and other purposes, which are briefly described in Chapter 3. Detailed discussions of these analyses, key assumptions, and uncertainties and limitations are provided elsewhere (e.g., ICF Resources, 1994; Chang et al., 1990; Dennis et al., 1990; Dennis et al., 1993), and are not the focus of this report. This report focuses on the approach used to quantify and value health effects associated with these previously estimated changes in ambient sulfate aerosol concentrations, and we provide a thorough discussion of the strengths and limitations of the health effects quantification and valuation procedures. We do not provide a detailed assessment of the analyses conducted previously that we rely upon in this report, but it is important to acknowledge that there is uncertainty in each of these analyses and results, which adds additional uncertainty to the final results of this analysis.

2.5.1 Key Uncertainties in Step 1: Estimating Changes in SO_2 Emissions

To estimate the benefits of Title IV it is necessary to make some assessment of what would have happened in the absence of the Title IV requirements. The benefits of Title IV are then calculated based on the difference between SO_2 emissions levels with and without the Title IV requirements for each future year included in the analysis. Possibly the greatest uncertainty in the first step in the analysis is in estimating what SO_2 emissions would have been over time in the absence of the Title IV requirements.

We refer to the estimate of what emissions would have been without the Title IV requirements as the reference case emissions estimates. For this analysis, we use SO_2 emissions estimates developed by ICF Resources (1994) for the EPA's Acid Rain Division for with Title IV and without Title IV scenarios. These estimates go through the year 2010. The reference case emissions estimates show a slight increase in total annual emissions between 1995 and 2005, and are fairly flat after 2005.

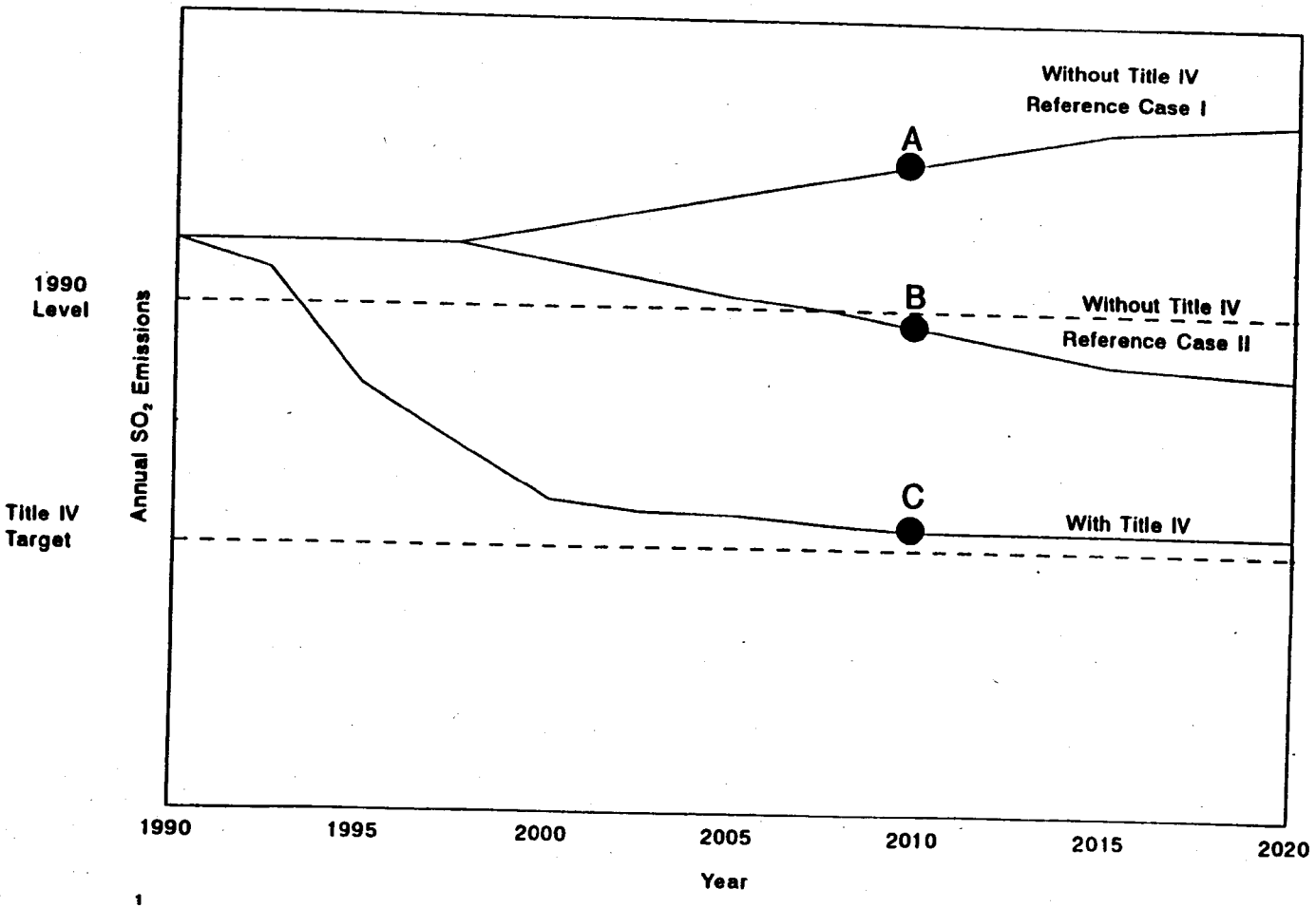
Figure 2-4 illustrates the potential significance of this reference case estimate for the calculation of Title IV benefits. The intent of this figure is to illustrate the potential importance of this question. It is not drawn to scale based on actual quantitative estimates. With Title IV, we expect SO_2 emissions will decline sharply in 1995 and come close to the

Title IV target by the year 2000. Potential use of banked emissions allowances will probably mean that the Title IV target will not be entirely met until 2010. Point C represents expected emissions in 2010 with Title IV. Without Title IV, SO_2 emissions might be higher, lower, or at the same level as in 1990. Reference Case I illustrates that if without Title IV emissions would have risen slightly by 2010, then the emissions reduction attributable to Title IV would be the difference between point A and point C. If, on the other hand, emissions would have decreased slightly in the absence of the Title IV requirements, as shown in Reference Case II, the emissions reduction attributable to Title IV would be the difference between point B and point C. Thus, the predicted reference case of what emissions would have been in the absence of Title IV can make a big difference when it comes to estimating the benefits of Title IV. Even without the Title IV requirements, there are many regulatory and economic factors that are expected to affect SO_2 emissions over the next several decades. NAPAP (1991) reports that future trends in SO_2 emissions without Title IV, would be expected to eventually result in emissions as low as are required under Title IV, but it is highly uncertain how fast this reduction would have occurred. This eventual reduction in emissions, in the absence of Title IV, would be expected to occur because of replacement of old facilities with new facilities that must conform to the stricter New Source Performance Standards under previously established requirements of the Clean Air Act and that have new cleaner technologies available that are more cost-effective to install with new facilities than to retrofit into old facilities.

2.5.2. Key Uncertainties in Step 2: Estimating Changes in Sulfate Aerosol Concentrations

Changes in sulfate aerosol concentrations are based on the intermediate results of RADM, a model developed to estimate acid deposition in the eastern United States as a function of SO_2 emission levels in specified locations. The transformation SO_2 emissions into sulfate aerosols, and the transport of SO_2 and sulfate aerosols through the atmosphere, is a function of complex chemical and meteorological interactions. RADM estimates these relationships for a sample of representative meteorological conditions and predicts annual sulfate concentration distributions at each location based on the estimated frequency of the defined alternative meteorological conditions. RADM has been thoroughly evaluated and tested as reported by Chang et al. (1990), Dennis et al. (1990), and Dennis et al. (1993). One of the most significant uncertainties in using the RADM estimates of airborne sulfate concentrations is that average meteorological conditions do not occur every year. This means that for any given year, the predicted concentrations are less reliable than over a multiple year period over which average meteorological conditions are more likely to prevail.

Figure 2-4
Illustration of Potential Changes in SO_2 Emissions



[1] This figure illustrates the potential effect of uncertainty in the reference case emissions estimate Reference Cases I and II are hypothetical estimates and are not drawn to scale based on any actual estimates.

2.5.3 Key Uncertainties in Step 3: Matching Population to the Sulfate Changes

The RADM grid cells are used as receptor locations to estimate the change in sulfate concentrations for the population. Residents are matched to RADM grid cells assuming that they are all located at the centroid of their census block group. Census block groups cover fairly small geographic areas, so the uncertainty introduced in this step is minimal. Greater uncertainty may exist as a result of people spending a significant share of their time at locations other than where they live. Close by locations such as travel to work create limited uncertainty because the sulfate gradient is fairly gradual from cell to cell. Considerable error could exist for individuals who spend a significant share of the year in locations far from their primary residences. However, this is not likely to be a significant source of uncertainty in this assessment relative to the other sources of uncertainty that are present.

2.5.4 Key Uncertainties in Step 4: Estimating Health Effects

Relying on available epidemiological evidence for estimating health effects associated with human exposure to ambient sulfate aerosols has many advantages, which are discussed in Chapter 4. The primary advantage is that it makes a quantitative assessment feasible with limited research resources and it uses a great deal of health effects evidence that is readily available. There are, however, several important uncertainties and limitations that result from the limitations of the available epidemiological evidence. The three uncertainties that we believe are the most potentially significant as a result of the limitations of the epidemiology evidence are summarized in this section. These and other uncertainties in the health effects calculations are discussed in more detail in Chapter 4.

First, there is uncertainty about the specific biological mechanisms that underlie the observed relationships in epidemiological studies, which raises uncertainty about the confidence with which the results should be interpreted as causative. Epidemiology studies are able to demonstrate whether a statistically significant relationship exists between health effects and pollution concentrations, but the studies do not prove that the relationship is causal. It is possible that a statistically significant relationship is really due to some unidentified factor that is correlated with pollution concentrations. The causation hypothesis is strengthened when epidemiological results are supported by repeated observation in different studies and by biological plausibility and consistency with evidence from other types of health effects studies. Although there is laboratory and clinical evidence of health effects associated with sulfates, as discussed in Chapter 4, the exact biological mechanisms that underlie the observed epidemiological association have not been established. This adds some additional uncertainty that is difficult to fully delineate when using epidemiological relationships to predict how health may change as a result of changes in ambient sulfate aerosol concentrations.

Second, there is uncertainty about the relative harmfulness of sulfates versus other types of pollutant aerosols that are typically present in the ambient air. Sulfates are a significant share

of the mix of fine particulate matter in the ambient air in many locations in the eastern United States. Some epidemiology studies have included sulfate concentrations as a measure of pollution, as well as more comprehensive measures of particulate matter such as $PM_{2.5}$ or PM_{10}. In some cases, epidemiology studies have found a statistically stronger association between health effects and sulfates (e.g., Plagiannakos and Parker, 1988), and other studies have found a stronger association with the more comprehensive measures of particulate matter (e.g., Dockery et al., 1992). Because of the typically high correlation among sulfates and other measures of fine particulate matter in the ambient air, it is difficult to statistically isolate the effects of sulfates alone in epidemiology studies. For this analysis, we examine the clinical, laboratory, and epidemiology evidence as a whole to determine reasonable assumptions about the relative contribution of sulfates to the epidemiological evidence of an association between health effects and fine particulate matter, but this remains an important uncertainty in the analysis.

Third, there is uncertainty about the extent to which health effects occur at lower ambient sulfate concentrations. For sulfate aerosols, and for particulate matter in general, it remains uncertain whether there is a threshold concentration below which health effects no longer occur, or whether the slope of the concentration-response function diminishes significantly at lower concentrations. Epidemiological studies do not always consider the question of thresholds, and epidemiological data are not always sufficient for making such a determination. Many recent epidemiology studies show a statistically significant association between sulfate concentrations and health endpoints over ranges of sulfate concentrations that are typical of current conditions in the eastern United States. For the mean estimates in this assessment we adopt the default assumption that there is no threshold for health effects associated with sulfates. Sensitivity analysis is used to show how the results might change if in fact some threshold exists at selected alternative concentrations.

2.5.5 Key Uncertainties in Step 5: Estimating Monetary Valuation of Health Effects

There are many uncertainties in available estimates and interpretations of monetary valuation for changes in human health effects. Although it is quite clear that changes in human health have both financial and nonfinancial significance to human welfare, determining appropriate monetary measures of the total effect on human welfare is a difficult task. The uncertainty in the monetary estimates is probably greatest for premature mortality risks. Sources of uncertainty in all the monetary estimates are discussed in Chapter 5, but here we highlight two key uncertainties in the monetary value estimates for premature mortality risks.

The first source of uncertainty in the monetary estimates for premature mortality is that there is little empirical economic evidence available about how health status or life expectancy affects an individual's willingness to pay for changes in risks of premature death. Available willingness-to-pay estimates for changes in risks of death are drawn primarily from samples of adults of average age distributions and average health status. It is possible that many of those

at greatest risk of premature mortality because of air pollution exposure are elderly or in relatively poor health. The available empirical evidence on this question is discussed in Chapter 5, but considerable uncertainty remains.

The second source of uncertainty in the monetary estimates for premature mortality is that most of the available estimates are for changes in the risks of accidental death rather than death due to illness, which is more the issue for pollution exposure. This is because the economic literature concerning monetary values for changes in risks of death has been able to exploit available data on wage differentials as a function of different levels of on-the-job risks of fatalities. It is uncertain whether individuals might have different reactions to risks due to illness rather than accidents, and how this might affect willingness to pay to avoid or reduce such risks. There is some evidence that risks of death due to particularly feared illnesses, such as cancer, are considered more abhorrent than risks due to accidents, but that evidence is limited.

CHAPTER 3
CHANGES IN AMBIENT OUTDOOR SULFATE CONCENTRATIONS

This chapter presents the approaches used in this assessment to estimate the changes in ambient outdoor sulfate aerosol concentrations by location attributable to the Title IV required SO_2 emissions reductions. This chapter relies on available results from other analyses conducted for or by the U.S. EPA for estimates of changes in SO_2 emissions and changes in ambient sulfate aerosol concentrations. In this chapter, we briefly describe these other analyses and explain how we use the results in this analysis.

3.1 CHANGES IN SO_2 EMISSIONS

ICF Resources (1994) has prepared for the U.S. EPA estimates of current and future SO_2 emissions by location through 2010 for a Title IV implementation scenario and for a no Title IV scenario. The ICF Resources analysis focuses on the SO_2 emissions in the utility sector, where 85 percent of the Title IV required emissions reduction is expected. This health benefits assessment incorporates, without modification, the ICF Resources annual SO_2 emissions estimates for the eastern United States.

Basic Features of CEUM

- set of interrelated models and databases for analyzing the coal and electric utility industries in an integrated way
- cost-minimizing linear programming model
- SO_2 emissions is one key output: others include NO_x emissions, environmental compliance information (e.g., compliance costs, coal market impacts, numbers of scrubbers used), power plant operational choices (e.g.. new plants built, fuel choice)
- incorporates technical and economic relationships of coal and electric utility markets
- high degree of resolution:
  - most generating units represented individually
  - detailed coal supply, transportation, transmission, and utility demand segments.

The analysis uses ICF Resources' Coal and Electric Utilities Model (CEUM). CEUM is a large linear programming model that develops least-cost compliance options across the utility industry in meeting SO_2 reduction targets. The model considers in detail the interaction between the demand for different types of fuels and the costs of supplying and delivering the fuels, as well as the interaction between utilities' marginal costs of compliance and the projected amount of allowance "banking."

CEUM uses a series of selected economic, energy market, and utility sector assumptions. These assumptions play an important role in estimating emissions with and without Title IV, because factors such as substitute fuel prices, energy demand, and economic growth can all have significant effect on decisions by utilities about building new capacity or retrofitting plants for alternative fuel use.

Emission levels are directly related to levels of electricity production, fuels used, and compliance options employed.

Figure 3-1 shows the ICF Resources estimates of utility SO_2 emissions with and without Title IV from 1990 through 2010. Maximum allowed SO_2 emissions are fairly well defined by the Title IV requirements. There is some uncertainty about how quickly the Title IV emission reduction goals will be met because there are provisions that allow utilities to bank unused emissions allowances and use them at a later time. It is uncertain how much banking the utilities will choose to do, but ICF Resources estimates that all banked allowances will be exhausted by 2010. Uncertainty also exists in predicting the specific location of emissions reductions because emissions allowances can be traded among emitting facilities.

Table 3-1 shows the ICF estimates of annual SO_2 emissions by state for 1997 and 2010, with and without Title IV. Both of the with Title IV estimates include an estimated response of utilities to the opportunities provided in the Title IV program to reduce emissions more than required in the early years of the program and to bank these as emission allowances for future use within a limited time period. The results of the with and without Title IV forecasts show that even with Title IV there are a few locations where SO_2 emissions are expected to increase slightly. However, there is expected to be a significant reduction in total emissions. In 2010, with Title IV, total SO_2 emissions from utilities in the East are expected to be about 7.7 million tons versus an estimated 16.8 million tons in 2010 without Title IV. The without Title IV emissions estimates do reflect emissions reductions expected due to other Clean Air Act Amendment requirements.

As noted in Chapter 2, there is more uncertainty in predicting what emissions would have been in the absence of Title IV than for the with Title IV scenario. Total emission limits are set by Title IV and utilities (as a group) are not expected to emit less than they are allowed under Title IV, because the Title IV limits are well below 1990 emission levels. In the absence of Title IV, there are some factors that would cause future SO_2 emissions to rise and some that would cause SO_2 emissions to decline. In general, economic and population growth results in greater demand for electricity, which may result in higher SO_2 emissions. At the same time, as older plants are retired and cleaner electricity generation processes are developed, SO_2 emissions per unit of electricity generated can be expected to decline. How emissions would change, therefore, depends on the relative significance of these different factors. ICF Resources estimates that in the absence of the Title IV requirements, SO_2 emissions from utilities would have risen slightly from 1990 levels. They predict a slight rise would have occurred between 1995 and 2005, and then a fairly flat trend through 2010.

Current SO_2 emissions vary considerably by location in the eastern United States in part because of significantly different amounts of high sulfur content fuels used in different locations. The reductions in SO_2 emissions expected as a result of Title IV are concentrated in areas that currently have the highest SO_2 emissions. Table 3-2 shows the ICF Resources estimates of the reduction in annual SO_2 emissions attributable to Title IV in 2010 by state for 31 eastern states. The last column shows the emissions reduction per capita in each state.

Figure 3-1
U.S. Utility SO_2 Emission Levels: 1990 through 2010

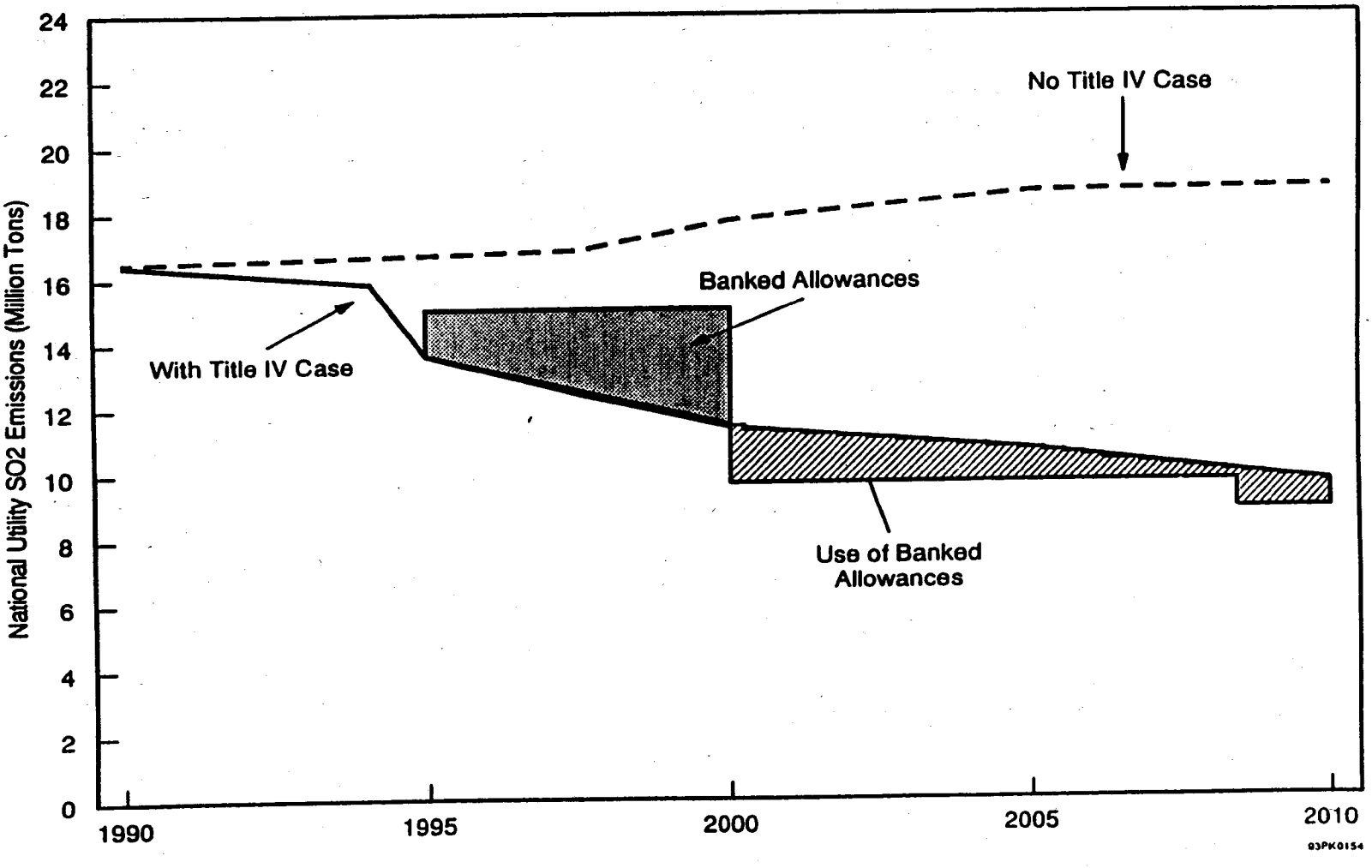


Table 3-1
EPA Forecasts of Annual Utility SO_2 Emissions
(thousand tons) by State[1]

State	1985	1997 (with Title IV)	1997 (no Title IV)	2010 (with Title IV)	2010 (no Title IV)
Maine, Vermont, New Hamp.	87	43	43	46	54
Mass., Conn., R.I.	308	175	175	164	189
New York	413	309	338	259	346
Pennsylvania	1,174	991	1,120	625	1,178
New Jersey	102	102	131	115	164
Maryland, Delaware, D.C.	285	336	340	217	430
Virginia	131	233	225	159	264
West Virginia	951	629	965	569	1,085
North Carolina, South Carolina	499	754	719	547	866
Georgia	998	577	912	414	919
Florida	531	542	748	517	900
Ohio	2,217	1,187	2,455	690	2,399
Michigan	409	428	427	370	397
Illinois	1,045	637	901	460	1,199
Indiana	1,496	738	1,360	536	1,559
Wisconsin	380	269	248	180	397
Kentucky	783	531	817	386	967
Tennessee	802	574	920	297	1,074
Alabama	534	478	661	379	681
Mississippi	102	94	160	94	163
Minnesota	111	140	140	104	136
Iowa	198	185	245	139	266
Missouri	961	455	897	308	944
Arkansas	73	85	85	93	93
Louisiana	79	104	104	71	99
Total 31 Eastern States	14,672	10,596	15,137	7,740	16,769

[1] Emissions estimates from ICF Resources (1994).

Table 3-2
Estimated Reduction in Annual Utility SO_2 Emissions in 2010 Attributable to Title IV by State

State	Emissions Reduction in 2010 (1000 tons)[1]	Population 1990 (1000s)	Reduction per Capita (10^{-2} tons/person)
Maine, Vermont, New Hamp.	7	2,900	0.24
Mass., Conn., R.I.	25	10,306	0.24
New York	87	17,990	0.48
Pennsylvania	553	11,882	4.65
New Jersey	48	7,730	0.62
Maryland, Delaware, D.C.	213	6,054	3.52
Virginia	105	6,187	1.70
West Virginia	516	1,793	28.78
North Carolina, South Carolina	319	10,116	3.15
Georgia	506	6,487	7.80
Florida	384	12,938	2.97
Ohio	1,709	10,847	15.76
Michigan	27	9,295	0.29
Illinois	738	11,431	6.46
Indiana	1,022	5,544	18.43
Wisconsin	217	4,892	4.44
Kentucky	581	3,685	15.77
Tennessee	777	4,877	15.93
Alabama	301	4,041	7.45
Mississippi	69	2,573	2.68
Minnesota	31	4,375	0.71
Iowa	127	2,777	4.57
Missouri	637	5,117	12.45
Arkansas	0	2,351	0.00
Louisiana	28	4,220	0.66

[1] Emissions estimates from ICF Resources (1994). Projected 2010 reductions are the difference between emissions with and without Title IV.

It is clear that a large variability in emissions reductions by location persists even after accounting for differences in population. The largest reductions are in the Appalachian and Midwest regions.

3.2 CHANGES IN SULFATE AEROSOL CONCENTRATIONS

The pollutant of interest in this health benefits assessment is sulfate aerosol, which is a secondary pollutant formed in the atmosphere in the presence of gaseous SO_2 emissions and other atmospheric constituents. The location and amount of SO_2 emissions are two factors that determine sulfate aerosol concentrations. Other factors are weather conditions, wind speed and direction, and the presence and quantities of other elements in the atmosphere that interact with SO_2 to form sulfate aerosols.

For this analysis, we use results from EPA's Regional Acid Deposition Model (RADM), which include estimates of ambient sulfate aerosol concentrations for alternative SO_2 emissions scenarios. Chang et al. (1990) provide a detailed description of RADM, and Dennis et al. (1990, 1993) provide results of evaluations of RADM. Airborne sulfate aerosol concentrations are an intermediate result provided by RADM for the purposes of estimating the eventual deposition of acidic species. RADM reports results, including ambient sulfate aerosol concentrations, for grid cells 80 km by 80 km in size, over the entire area of the eastern United States. SO_2 emission rates by location, as estimated by ICF Resources, are an input into RADM. The RADM estimates used in this health benefit assessment are the ground-level sulfate aerosol (SO_4) concentrations for the following SO_2 emissions scenarios:

- Actual 1985 emissions, used to approximate conditions when the 1990 Amendments went into effect
- Estimated 1997 emissions with Title IV and banking

The Regional Acid Deposition Model

The RADM is a comprehensive model of the atmospheric processes that lead to the formation and deposition of acidic species. The objective of this modeling system is to provide a scientific basis for estimating the change in deposition caused by large changes in precursor emissions. Specifically, the RADM is designed to (1) mathematically represent the nonlinear dynamics both of oxidant formation from precursor emissions of NO_x and VOCs, and of scavenging of sulphur compounds, and (2) mathematically represent the three-dimensional dynamics of transport, transformation, and deposition, including effects of cloud processes. The version of the model used for this analysis (Version 2.6) is designed to report this information on grid cells 80- × 80-km in size, over a domain that extends from east of central Texas to the south of James Bay, Canada, including all of Florida and southeastern Canada. This version of RADM uses six vertical layers from the ground to approximately 16 km in altitude. Version 2.6 has been corrected for some under predicting of sulfate levels that occurred with earlier versions.

The model operates on a mathematical frame of reference in which concentrations are specified as functions of time at fixed positions within the grid cells. The RADM uses the wind flow and precipitation simulated by a mesoscale meteorological model, called the MM-4, over an episodic period chosen to be 3 days. Modules of various chemical and physical processes involving the transport, transformation, and removal of pollutants are included in RADM and they utilize the meteorological simulations obtained from the MM-4. Because each run of the RADM represents a 3-day episode, a method to produce seasonal and annual estimates using a sample of episodic runs is required. Each episode is weighted according to its relative importance toward seasonal and annual wet deposition. RADM is run in each episode, and the results are multiplied by the weighing factors to produce seasonal and annual deposition calculations.

Figure 3-2
RADM 50th Percentile Annual Sulfate Concentration ($\mu g/m^3$)
1985 Base Case

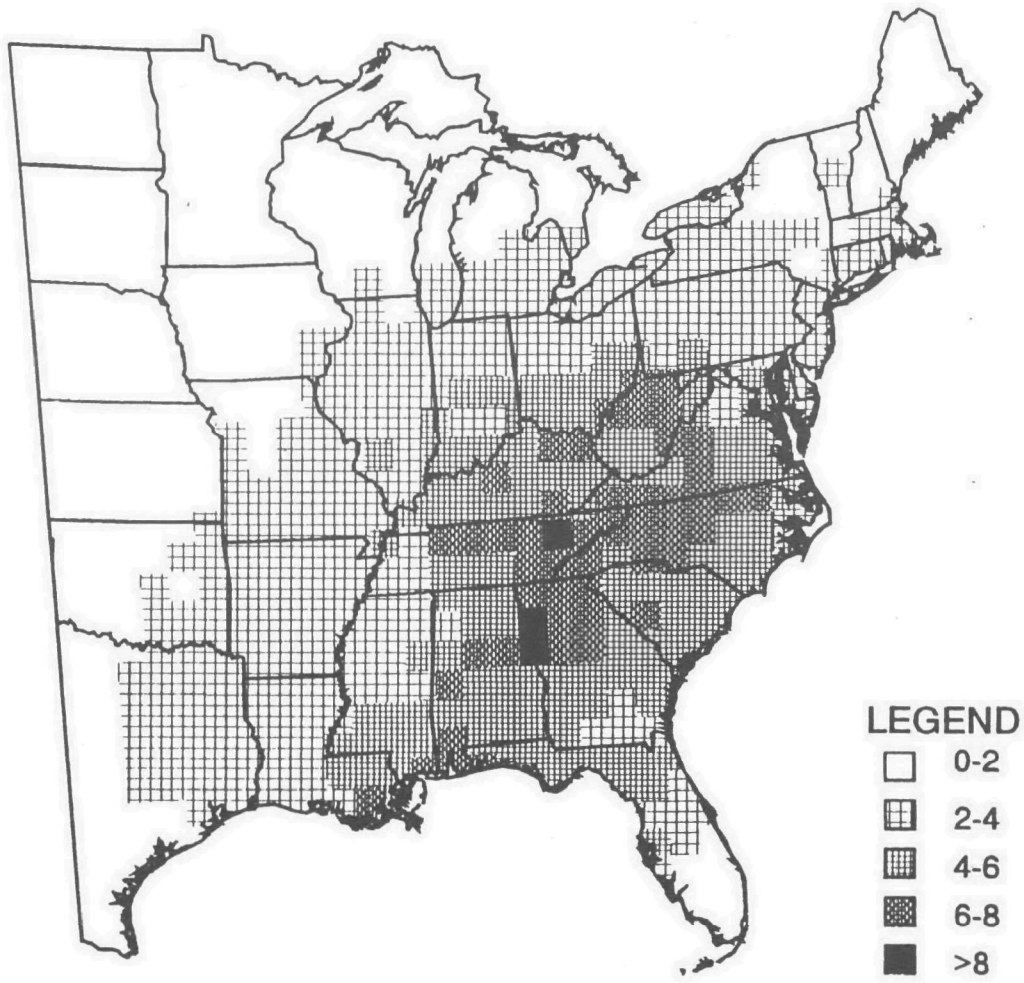


Figure 3-3
RADM 50th Percentile Annual Sulfate Concentration ($\mu g/m^3$)
1997 with Title IV[1]

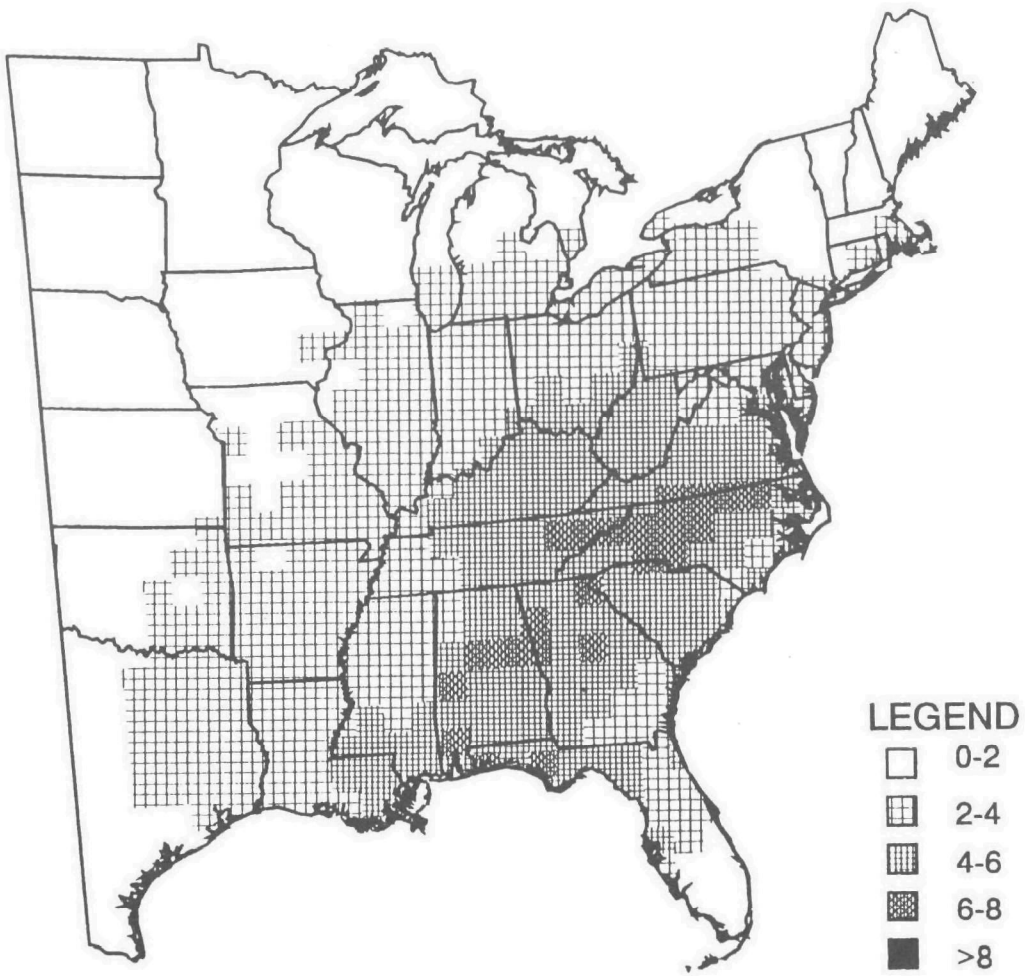


Figure 3-4
RADM 50th Percentile Annual Sulfate Concentration ($\mu g/m^3$)
2010 without Title IV

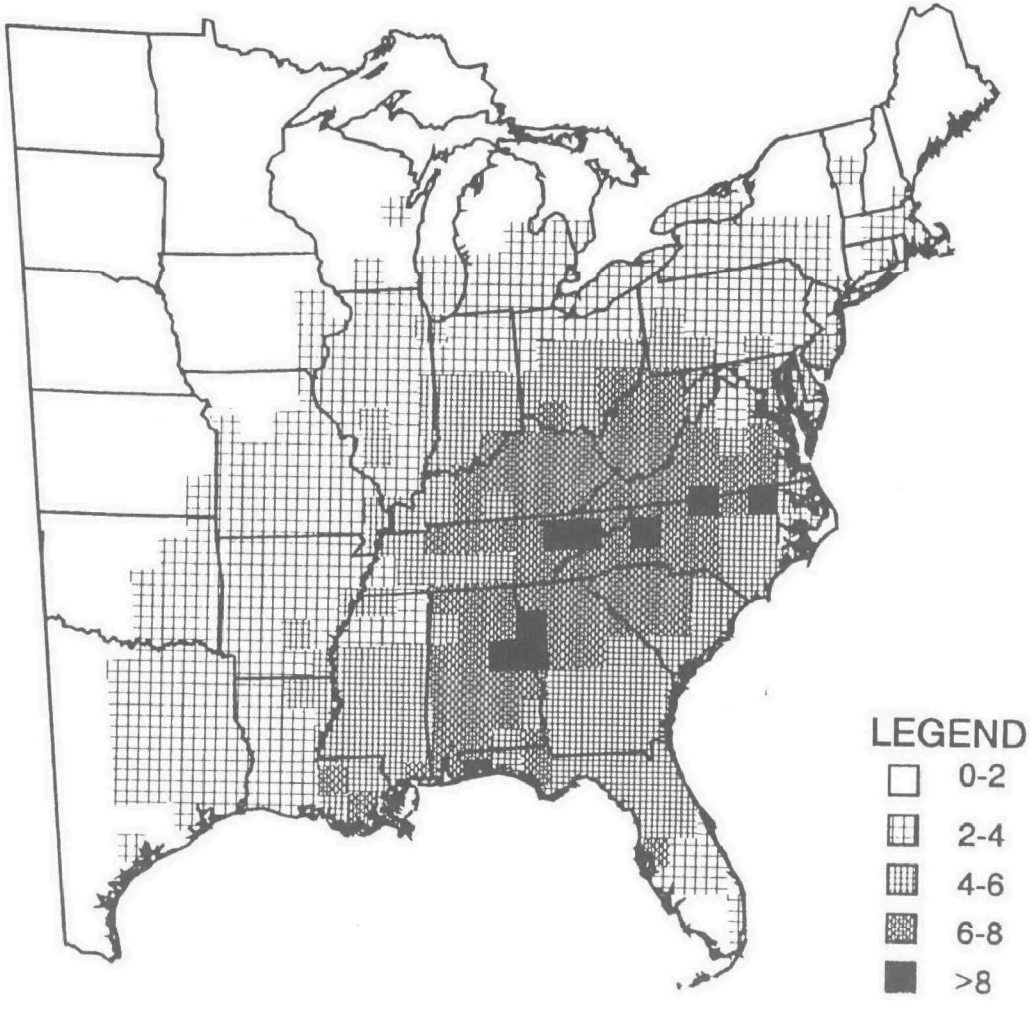
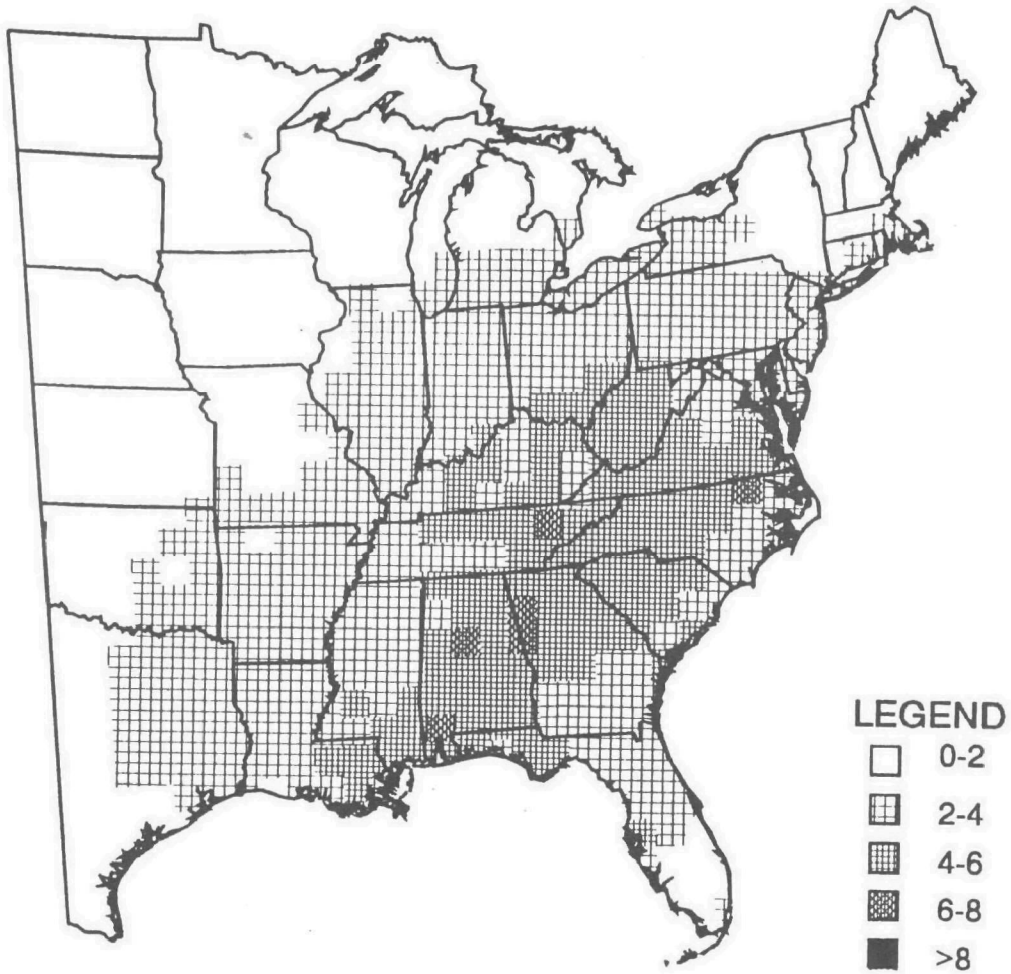


Figure 3-5
RADM 50th Percentile Annual Sulfate Concentration ($\mu g/m^3$)
2010 with Title IV



- Estimated 2010 emissions with Title IV
- Estimated 2010 emissions without Title IV.

Figures 3-2 through 3-5 illustrate the distribution of the RADM sulfate aerosol concentration estimates across the eastern United States for each of the SO_2 emissions scenarios.

RADM results used in this assessment are summarized in Table 3-3. Table 3-3 gives the estimated reduction in median annual SO_4 concentrations for 1997 with Title IV and emission allowance banking versus the SO_4 concentrations under current (1985) conditions and for 2010 with Title IV versus predicted SO_4 concentrations without Title IV. These are ground-level SO_4 reductions for the 50th percentile of the annual distribution of estimated SO_4 concentrations. The results in these tables are the averages of the changes in the 50th percentile concentrations by state based on the results for the 80 km by 80 km RADM grid. Exposures and health effects are calculated at the grid cell level in this assessment, but averages for the states are shown here because the grid level data are too numerous.

The partial states at the western edge of the RADM grid, as shown in Figures 3-2 through 3-5 have been dropped from the quantitative assessment because the sulfate concentration changes expected in this area are small. The RADM grid also covers the southern parts of several Canadian provinces. Significant changes in sulfate concentrations are predicted as a result of the expected reductions in SO_2 emissions in the United States for Ontario and Quebec, so these have been included in the assessment. The portions of these provinces covered in the air quality model include the areas where the vast majority of the populations of these provinces live. The northern edge of the RADM grid is just south of the southern edge of James Bay.

3.3 MATCHING POPULATION TO ATMOSPHERIC SULFATE CHANGES

To calculate the human health benefits associated with the expected reduction in atmospheric sulfate aerosols concentrations, it is necessary to determine the change in ambient outdoor sulfate concentrations where people are. This requires an overlay of the population distribution on the RADM grid to match numbers of people to the estimated changes in sulfate aerosol concentrations.

For this analysis, we use the Geographic Information System (GIS) to match the 1990 population data from the U.S. Census (1990) and the 1991 Canadian Census to the RADM grid, and to estimate the populations in each relevant age group residing in each of the 1330 RADM grid cells. EPA provided us with the latitude-longitude coordinates for the center of each RADM grid cell. These were projected into lambert projected meters using standard parameters for lambert conformal projections of the United States. This gave us an orthogonal grid of points. We then used the THIESSEN procedure to draw grid cell boundaries equidistant between each pair of grid cell points.

For the U.S., the latitude-longitude coordinates for each centroid of each census block group, as provided on U.S. Census Summary Tape File 3A, were then located on the RADM grid. For Canada, the latitude-longitude coordinates for each centroid of each enumeration area, as provided by MapInfo Corp. under license from Statistics Canada, were then located on the RADM grid. Total population, divided into relevant age groupings for the health effects calculations, for each block group or enumeration area was assigned to the grid cell within which the block group or enumeration area centroid was located.[1] The error in assuming that all the population is located at the centroid of the block group or enumeration area is small given that the block groups and enumeration areas are small relative to the size of the RADM grid cells. There are about 300,000 block groups in the study area, each with a total population of about 670. An enumeration area usually contains about 125 dwellings in a rural area and 375-400 dwellings in an urban area.

State or province identifiers for each block group or enumeration area were used to sum to state or province[2] level results after health effects estimates were calculated for each RADM grid cell, based on the differences in predicted sulfate concentrations for the cell under different scenarios.

This assessment estimates health benefits for changes in sulfate concentrations in 1997 and in 2010. The 1990 populations are therefore adjusted for expected average population growth using the mid-forecasts of the U.S. Census and the World Bank population projections for Canada. These adjustments are made at the aggregate level using national average population growth factors.

[1] Block group specific age data were used for the U.S. population. For the Canadian population, country average age distributions (Statistics Canada, 1994) were applied uniformly to each enumeration area.

[2] The RADM grid covers virtually all of Ontario's population, but not all of Quebec is covered. The population of Quebec used in the assessment only includes those persons living in enumeration areas covered by the RADM grid. Approximately 99 percent of Quebec's population is included.

Table 3-3
Average Reductions in Median Annual SO_4 Concentrations ($\mu g/m^3$) by State/Province Due to Title IV

State/Province	1997[1]	2010[2]	State/Province	1997	2010
Alabama	0.44	1.93	Mississippi	0.24	1.01
Arkansas	0.22	0.54	Missouri	0.16	0.45
Connecticut	0.35	0.26	New Hampshire	0.21	0.16
Delaware	0.22	0.86	New Jersey	0.22	0.68
District of Columbia	0.30	1.48	New York	0.29	0.34
Florida	-0.02	1.01	North Carolina	0.30	1.73
Georgia	0.31	1.88	Ohio	0.51	1.43
Illinois	0.31	0.80	Pennsylvania	0.44	0.92
Indiana	0.53	1.28	Rhode Island	0.41	0.31
Iowa	0.00	0.21	South Carolina	0.24	1.82
Kentucky	0.86	2.02	Tennessee	0.84	2.09
Louisiana	0.08	0.70	Vermont	0.21	0.20
Maine	0.11	0.15	Virginia	0.42	1.75
Maryland	0.41	1.29	West Virginia	0.72	2.08
Massachusetts	0.24	0.24	Wisconsin	0.03	0.20
Michigan	0.11	0.29	Ontario	0.13	0.13
Minnesota	-0.03	0.05	Quebec	0.09	0.05

[1] The 1997 reduction is estimated versus 1985 emissions.
[2] The 2010 reduction is estimated versus 2010 without Title IV emissions.

CHAPTER 4
QUANTIFICATION OF HEALTH EFFECTS CHANGES

4.1 BACKGROUND ON HEALTH EFFECTS STUDIES

Several different types of health effects studies are used to measure health responses to environmental pollutants. Different types of studies provide different types of information. Each type of study has different strengths and weaknesses, including variations in the types of health effects that can be considered. Some types of evidence are better suited for use in a quantitative assessment. The brief background review of health effects studies provided in this section is intended to help place the assessment in context for those policy makers who may not be familiar with the health effects literature. Strengths and weaknesses of the assessment are integrally linked to those of the scientific literature upon which the quantitative assessment is based.

4.1.1 Types of Health Effects Studies

The types of studies that provide evidence of health effects following exposure to sulfate aerosols include epidemiology and field studies, human clinical studies, and laboratory and toxicology studies.

Epidemiology and Field Studies

Epidemiological and field studies for sulfate aerosols typically involve estimation of a statistical relationship between the frequency of specific health effects observed in a study population in its normal environment and sulfate aerosol concentrations measured at stationary outdoor monitors in the study area. These studies are therefore able to provide "concentration-response" functions that can be used to estimate the change in the frequency of health effects for a population in its normal environment that would be expected to occur with specific changes in ambient outdoor sulfate aerosol concentrations. A concentration-response function is a quantitative relationship between ambient levels (concentration) of a pollutant and the frequency of specific health effects in a given time period (response). For example, it may give the percentage of study subjects who report cough symptoms on a given day as a function of the concentration of ambient sulfate aerosol on that day.

Epidemiology and field studies often involve time-series analyses of changes in rates of health outcomes within a specific area, sometimes for a pre-selected sample, as air pollution concentrations fluctuate. An example of this study design would be daily observation and

recording of asthma symptoms with a pre-selected group of subjects with diagnosed asthma, and statistical analysis to determine if there is an association between the frequency of the symptoms and fluctuations in sulfate aerosol concentrations from day to day. Epidemiology studies may also use cross-sectional data, looking at differences in health outcomes across several locations at a selected point or period of time. This may involve, for example, a comparison of the prevalence of chronic respiratory disease in different cities with different average sulfate aerosol concentrations. Although cross-sectional studies have the advantage of being able to consider potential effects associated with long-term exposures, it can be very difficult to fully control for potential confounding factors. Time-series studies reduce many of the problems associated with confounding or omitted variables because the same population group is studied over time, but weather and seasonal variation that may be correlated with sulfate aerosol concentrations can pose some similar problems.

Time-series and cross-sectional epidemiology studies can be either cohort studies or population studies. Cohort studies analyze the incidence of health effects in a sample of identified individuals usually selected specifically for the study. For example, a cohort might be a group of study subjects who record daily symptoms for a period of time. A cohort study might also collect data on the health status of a selected sample of individuals and then do a follow-up on the same individuals after a specified length of time to determine what changes in health status have occurred for each individual. Population studies, on the other hand, rely on data available for the population as a whole rather than tracking the effects on specific individuals. For example, a population study may analyze daily mortality rates in a given location as they related to daily particulate matter concentrations. Another example of a population study is a comparison of the prevalence of chronic respiratory disease in different locations with different average pollution concentrations. In general, cohort studies are preferred because characteristics of the individuals in the study sample can be determined, allowing better control for other risk factors, such as smoking or diet. Population data are, however, readily available for many types of health effects and therefore provide an opportunity to conduct epidemiology analyses very cost effectively.

One of the strengths of epidemiology studies is that they analyze actual health effects in human populations at ambient pollution concentrations. Subjects are studied in their normal environment and the health effects are directly observed. A major challenge for epidemiology studies is the difficulty in isolating with confidence the effects of a specific air pollutant such as sulfate aerosol when this may be just one of many complex factors that influence human health. Finding a statistically significant correlation between a health effect and exposure to sulfate aerosols does not prove causality. To support an inference of causality, epidemiology results need to be supported by repeated observation in different studies and by biological plausibility and consistency with evidence from other types of health effects studies.

Human Clinical Studies

Human clinical studies, sometimes also called chamber studies, examine the response of human subjects to pollutant exposures in a controlled laboratory setting. The response of the individual can be monitored and the environment controlled so that the effects of one pollutant can be isolated. Clinical studies on sulfate aerosols have typically exposed subjects to specific sulfate aerosol concentrations for one or several hours and measured responses such as pulmonary function or respiratory symptoms, sometimes in combination with moderate or vigorous exercise. Clinical results can provide evidence of causation because confounding factors are well controlled.

Clinical studies are limited to consideration of short-term reversible health effects that can be purposely induced in human subjects. Also, the health effects of short-term exposure to sulfate aerosols may be different in the everyday environment where other pollutants are also present and the individual's behavior and activities are quite varied. Clinical results are also limited for the purposes of extrapolation and generalization because sample sizes are usually quite small, making generalization somewhat difficult. Clinical results, in combination with supporting epidemiology results, can support an inference of causality between pollution exposure and observed health effects.

Laboratory and Toxicology Studies

Laboratory and toxicology studies use animal subjects, and sometimes human tissue or cells, to study biological responses to pollutants in a controlled laboratory setting. Animal organs and tissue can be directly examined for effects of acute and chronic exposures, revealing a wealth of information about biological responses to sulfate aerosol exposures. These studies provide a great deal of useful and important information about the specific biological pathways and mechanisms by which pollutants cause harm to living organisms. For example, laboratory studies may provide direct biological evidence of how a pollutant decreases the ability of a living organism to defend against disease and infection.

4.1.2 Advantages and Limitations for Assessment Purposes

For a quantitative assessment of the human health benefits of Title IV, we want answers to the following questions:

- How many cases of each specific type of health effect will be avoided because of Title IV?
- How much does the exposed population value the reduction in these health effects?

Epidemiology Advantages and Limitations

For addressing the first question, many epidemiology studies provide sufficient information to infer a concentration-response function, which typically gives a quantitative relationship between the incidence of a given health effect and ambient outdoor air pollutant concentrations. A concentration-response function can be used to predict a change in the number of cases of a given health effect for an estimated change in ambient outdoor pollutant concentration.

Epidemiology-based concentration-response functions available in the literature pertaining to airborne sulfates correlate observed changes in health status or symptoms with ambient outdoor sulfate concentrations. Everyday human activity patterns and specific pollutant doses associated with specific outdoor pollutant concentrations are implicit in the concentration-response functions. Changes in incidence of specific health effects can therefore be estimated as a function of changes in ambient outdoor pollutant concentrations without conducting detailed pollution exposure modeling, as long as we accept the assumption that the human activity patterns will not change significantly when ambient outdoor pollutant concentrations change. There are implicit assumptions in this approach that the relationship between outdoor concentrations and individual exposure that exist in the original study populations are the same in the assessment population.

Epidemiology studies are also useful in addressing the second question because they are able to define health effects in terms of factors that can be directly related to perceived welfare, such as risks of premature death or days with noticeable respiratory symptoms. By drawing on available health data such as vital statistics and national health surveys, or observing changes in health over time for a panel of study subjects, epidemiology studies are able to consider a wide range of health effects. This includes very serious health effects such as premature mortality or chronic disease that are not possible to study with human subjects in controlled exposure environments. Epidemiology studies can be designed to consider potential effects of long-term exposures to air pollutants as well as short-term effects. This is an additional advantage over clinical studies.

The primary limitation in using epidemiology results for predicting changes in health effects as a function of changes in ambient air pollutant concentrations is the uncertainty about whether the causal factors for the observed association with health effects have been fully and accurately specified. Inaccurate predictions could occur, for example, if the actual causal factor is some unspecified pollutant that is highly correlated with the specified pollutant. Thus, a change in the specified pollutant would not necessarily result in a change in health effects, unless the unspecified pollutant were to also change in the same proportion. This source of uncertainty is always present in epidemiology results to some extent, and the potential for error is difficult to quantify because the extent of unspecified, and correlated, causal factors is unknown.

There may also be other important inaccuracies in the specification of the relationship between health effects and ambient air pollutant concentrations in an epidemiological study. Functional forms for the concentration-response relationship and averaging times for pollutant concentrations are examples of things that might be misspecified, resulting in inaccuracies in predictions of health effects changes. For example, if a linear relationship is specified but the actual relationship is significantly nonlinear, the predicted estimates could be either too high or too low, depending on the shape of the actual relationship. These sources of uncertainty are also difficult to quantify because the extent of the error in the original epidemiological specifications is unknown. Uncertainties are greater if epidemiology results are extrapolated beyond the range of concentrations over which the original results were estimated.

Clinical Advantages and Limitations

Clinical study results provide information about the relationship between exposure and health response obtained in a controlled environment. Thus, concerns about whether the observed relationship is actually causal are reduced. Relationships between exposure and response are more accurately measured in the controlled environment of the clinical study. This is in contrast to most epidemiology studies which use ambient outdoor pollutant concentrations as the measure of exposure. Clinical studies therefore provide potentially more accurate, or at least more convincing, dose-response information than is obtained with epidemiology studies. This is an important advantage of clinical study results for quantitative assessment purposes.

There are two significant limitations of clinical study results when it comes to a quantitative assessment of changes in health effects as a function of changes in outdoor pollutant concentrations. First, clinical study results for quantitative assessment purposes are limited because only a small range of exposures and potential health effects can be considered in clinical studies. Clinical studies are generally confined to short-term exposures and to health effects that are reversible and not life-threatening. It is simply not possible to confine human subjects to controlled environments for extended periods of time or to attempt to induce permanent or life-threatening health effects. Clinical results are therefore unable to provide information on the full range of potential health effects of pollutant exposures, if long-term exposures or permanent or life-threatening health effects are suspected.

Second, using clinical study results requires some type of quantitative exposure analysis to link changes in outdoor ambient air quality to pollutant exposures as measured in the clinical (indoor) study setting. This typically requires some analysis or assumptions about how much time people spend in various environments (e.g., indoor, outdoor, automobile) and about the relationship between outdoor pollutant concentrations and pollutant concentrations in each of the other types of human environments. Thus, several extra steps are added to the analysis relative to what is needed when epidemiology study results based on outdoor pollutant concentrations are used.

Two quantitative assessments conducted recently for changes in ambient ozone concentrations provide examples of assessments that have used some available clinical-based dose-response information for acute respiratory symptoms as a function of controlled ozone exposures (Krupnick and Kopp, 1988; Hall et al., 1989). These studies illustrate the difficulty in applying clinical dose-response functions because of the need for detailed exposure analysis; they also provide an interesting comparison between results obtained using epidemiology-based concentration-response functions and clinical-based dose-response functions for the same type of health effect: acute respiratory symptoms.

The clinical studies used in these two assessments provided data on whether respiratory symptoms occurred with subjects exposed to controlled concentrations of ozone while exercising for one, two, or seven hours. To utilize these results to estimate how a change in ozone concentrations in the ambient outdoor air would affect the frequency of respiratory symptoms for a population in its everyday environment, either extensive modeling or assumptions must be used regarding population activity patterns and resultant ozone exposures. Hall et al. (1989) developed a detailed ozone exposure model for the South Coast Air Basin. They found that estimates of the frequency of respiratory symptoms based on the clinical results and the exposure modeling were higher per unit of ambient outdoor ozone relative to the results obtained using available epidemiology study results. Krupnick and Kopp (1988) did not conduct detailed exposure modeling, but rather used a range of alternative assumptions regarding activity patterns and exposures. They obtained estimates that were either higher or lower than the epidemiology-based estimates, depending on the assumptions used in the exposure portion of the analysis.

Laboratory Advantages and Disadvantages

Laboratory study results have the same advantage as that discussed above for clinical study results: pollutant exposures are well controlled in a laboratory setting and variations in confounding factors are reduced. The analyst therefore has more confidence that the observed relationships are causal and that the measured dose-response functions are accurate. Laboratory studies also have the potential to consider the effects of long-term as well as short-term exposures, which extends the range of health effects that might be considered relative to clinical studies.

Laboratory study results have three important limitations when it comes to a quantitative assessment of changes in health effects as a function of changes in pollutant emissions. Similar to clinical study results, using laboratory study results requires some type of quantitative exposure analysis to link changes in outdoor ambient air quality to pollutant exposures as measured in the laboratory study setting. Thus, an extra step is added to the analysis relative to what is needed when epidemiology study results are used. Second, laboratory studies often use animal subjects, which introduces considerable uncertainty when attempting to extrapolate quantitative results to human populations, as is needed in a quantitative assessment. Third, laboratory studies sometimes focus on health effects that are

difficult to interpret in terms of specific illnesses or symptoms. Linkages between cellular and biochemical concentration changes and clinical manifestation of illness are often difficult to quantify.

4.2 SUMMARY OF HEALTH EFFECTS EVIDENCE FOR SULFATE AEROSOLS

This section provides a brief summary of the available health effects evidence concerning sulfate aerosols and other fine particulates ($PM_{2.5}$). This summary is not intended to be a comprehensive review. Its purpose is to highlight the range of available evidence, list the kinds of health effects that have been observed, and to focus specifically on health effects that have been found in association with sulfate aerosols because they are the focus of this assessment. Many of the health effects listed here have also been found to be associated with $PM_{2.5}$ concentrations in locations where sulfate concentrations are low, so none of the findings reported here and elsewhere in this report should be interpreted as suggesting that sulfates are the only harmful constituent of $PM_{2.5}$.

Detailed reviews of available health effects evidence for inhalable particulate matter, including sulfate aerosols, covering results from laboratory, clinical, and epidemiology studies, are provided in the EPA criteria documents and other documents (U.S. EPA, 1982, 1986a, 1986b, 1989, 1995). Additional reviews of part or all of this literature include Ferris (1973), Graham et al. (1990), American Thoracic Society (1991), Gong (1992), Folinsbee (1992), and Lipfert (1994).

4.2.1 Epidemiology Study Findings

A detailed discussion of epidemiology study findings is presented in Section 4.4, including identification of specific concentration-response functions selected for use in this assessment. This section gives an overview of the types of health effects that have been observed in epidemiology studies concerning sulfate aerosols and $PM_{2.5}$.

Epidemiology studies conducted to date provide evidence of statistically significant associations between ambient outdoor concentrations of sulfate aerosols or $PM_{2.5}$, or both, and the following human health effects:

- **Premature mortality.** Evidence has been found in prospective cohort and cross-sectional studies of an association between mortality rates in different locations and average sulfate concentrations in those locations (e.g., Pope et al., 1995). Evidence has also been found in time-series studies of an association between daily mortality rates and sulfate concentrations in several urban areas in the United States and elsewhere (e.g., Dockery et al., 1992).

- **Chronic respiratory disease.** Prospective studies have found higher rates of chronic respiratory disease in locations with higher $PM_{2.5}$ concentrations (e.g., Abbey et al., 1995).

- **Hospital admissions.** Time-series studies show a correlation between daily hospital admission rates and daily sulfate concentrations (e.g., Burnett et al., 1995).

- **Aggravation of asthma symptoms.** Time-series studies with panels of diagnosed asthmatics who record their symptoms and medication usage each day have found an association between the aggravation of asthma symptoms and daily sulfate concentrations (e.g., Ostro et al., 1991).

- **Restricted activity days.** Self-reported number of days on which activities are restricted because of illness during a 14-day recall are recorded in a national sample through the Health Interview Survey. The frequency of such days has been found to be significantly associated with the average $PM_{2.5}$ concentrations in the city of residence during the same 14-day period (e.g., Ostro and Rothschild, 1989).

- **Acute respiratory symptoms.** In a study during which a panel of healthy subjects recorded daily respiratory symptoms, the frequency of such symptoms was found to be correlated with daily sulfate concentrations in the study location (e.g., Ostro et al., 1993).

Taken as a whole, the available epidemiology evidence shows a strong relationship between sulfate aerosols, and other fine particulates, and respiratory-related illness in the United States. The types of illness range from severe acute and chronic illnesses that are associated with increases in risks of death to mild acute symptoms such as coughing and wheezing. There is epidemiology evidence of health effects for both short-term fluctuations in sulfate concentrations within a given location and long-term variations in sulfate concentrations across locations.

4.2.2 Clinical Study Findings

Several studies have examined the health effects of humans exposed briefly through inhalation to moderate concentrations of sulfate aerosols in the form of sulfuric acid (e.g., Amdur et al., 1991; Koenig et al., 1993). The effects observed in some of these acute exposure studies include decreased pulmonary function and decreased bronchial clearance rates.[1] Graham et al. (1990) review this literature and conclude that acute exposures to some acidic sulfates can increase airway resistance, decrease pulmonary function, and increase responsiveness to

[1] This section provides just a brief overview of clinical study findings and draws upon the summary of clinical research on this topic provided by Admur et al. (1991).

bronchoconstrictors, especially in asthmatics, but that considerable variability in the results of different studies suggests uncertainty about which exposures will reproducibly cause these effects.

Decreased pulmonary function in the form of increased airway resistance has been noted in some studies for both adult and adolescent asthmatics following inhalation exposure to sulfuric acid during exercise (Amdur et al., 1991; Koenig et al., 1993). In adult asthmatics, inhalation exposure to 450 $\mu g/m^3$ sulfuric acid for 16 minutes resulted in an increase in airway resistance, whereas exposure to 100 $\mu g/m^3$ caused no response (Amdur et al., 1991). In adolescent asthmatics, inhalation exposure to 68 $\mu g/m^3$ sulfuric acid for 40 minutes resulted in increased airway resistance. The increase in airway resistance of adolescent asthmatics was greater following exposure to a combination of sulfuric acid and 0.1 ppm sulfur dioxide (Amdur et al., 1991). No increase in airway resistance was observed following acute inhalation exposure of nonasthmatics to 1,000 $\mu g/m^3$ sulfuric acid.

Bronchial clearance, a major defense mechanism employed by the body following inhalation of irritant particles, decreased following inhalation exposure to moderate concentrations of sulfuric acid. Studies in humans show that inhalation exposure to less than 200 $\mu g/m^3$ sulfuric acid for one hour stimulates clearance in larger airways; however, clearance is depressed in small airways where more acid deposits. Clearance is restricted in both small and large airways following exposure to 1,000 $\mu g/m^3$ sulfuric acid (Amdur et al., 1991). At exposure concentrations of 100 $\mu g/m,^3$ increasing the exposure time from one to two hours results in an even greater decrease in bronchial clearance, and a persistent reduction in clearance of particles for up to three hours following exposure (Amdur et al., 1991).

The effects noted (i.e., decreased bronchial clearance and decreased respiratory ability) are similar to some of the effects observed in epidemiological studies, including increased incidence of acute respiratory symptoms and depressed lung function (American Lung Association, 1978). However, as described in Section 4.1, these studies are limited for the following reasons:

- Exposure was limited to either sulfuric acid or a combination of sulfuric acid and sulfur dioxide. Synergistic or antagonistic interactions between air pollutants would not be represented in these studies.

- Because of the inherent and understandable limitations of clinical studies, health effects resulting from chronic exposure to sulfate aerosols cannot be observed in clinical studies. It is possible that effects over a longer duration would be more pronounced. Continuous exposure to ambient aerosols results in the simultaneous deposition and redistribution of particles, causing changes such as marked and persistent depression in bronchial clearance, whereas acute exposure results in an initial rapid clearance of inhaled particles (U.S. EPA, 1986a). Indeed, animal exposures show a pattern of decreased clearance that continues well after exposure has ceased.

- The sample size of the exposed populations was quite small, making extrapolation to the actual exposed populations difficult. Additionally, sample demographics were not representative of actual exposed populations.

- The body of work on the effects of sulfuric acid on pulmonary function is not fully consistent, and concentration-response relationships have not yet been demonstrated (Graham et al., 1990).

4.2.3 Animal Toxicological Study Findings

Acute Exposure Animal Studies

The effects of acute inhalation exposure to sulfate aerosols (in the form of sulfuric acid) in animals have been described in several studies. The studies used a variety of species, including mice, rats, guinea pigs, dogs, donkeys, rabbits, and monkeys. Although some studies failed to cause effects in exposed animals, a number of studies show respiratory effects increasing with concentration and decreased particle size. Observed effects, including respiratory system damage, increased airflow resistance, and decreased function of the body's defense mechanisms, are summarized below.

Respiratory system injury, including lesions in the bronchi, bronchioles, larynx, and trachea, was noted in mice and guinea pigs following short-term exposure to high concentrations (60 to 125 mg/m^3) of sulfuric acids (Lee and Mudd, 1979). These are, however, much higher concentrations than are typical of ambient conditions in the United States.

Increased airflow resistance occurred in exposed animals, the magnitude of which is related to both concentration and particle size. At concentrations below 1 mg/m^3, a greater response was observed for 0.3 μm than for 1 μm particles (Amdur et al., 1991; Chen et al., 1991). Significant airflow resistance continued up to at least one hour following exposure, and persisted longer than flow resistance resulting from exposure to sulfur dioxide (Amdur et al., 1991; Chen et al., 1991).

A decreased ability in mechanisms enabling the body to respond to disease and infection was noted (Lee and Mudd, 1979; Amdur et al., 1991). One study determined that a single inhalation exposure to concentrations of sulfuric acid at concentrations that occur in the ambient air decreased the body's resistance to infectious disease (Zelikoff and Schlesinger, 1992). The body's defense mechanisms are impaired as described below:

- There is a decrease in production of interferon, which provides resistance to viral infections (Lee and Mudd, 1979).

- A decrease in bronchial clearance occurs (Amdur et al., 1991; Fujimaki et al., 1992). As described above, bronchial clearance is one of the body's defense mechanisms.

- The release of histamine, a compound believed to cause allergic reactions, is increased. This suggests that sulfuric acid might be one cause of allergic diseases (Fujimaki et al., 1992).

- Both phagocytic activity of macrophages and superoxide production are decreased following inhalation exposure to relatively low concentrations of sulfuric acid (Schlesinger et al., 1992). This decreased function compromises the cellular ability to defend against infection and disease.

Although these findings are consistent to a certain degree the effects observed in epidemiological studies, such as increased respiratory infection and decreased respiratory function in children, it is difficult to determine their applicability for the following reasons:

- Exposures were brief, rather than the daily long-term exposure typical of air pollution exposure.

- Interaction among various air pollutants was not studied.

- Extrapolation of animal findings to humans contains a degree of uncertainty.

Chronic Exposure Animal Studies

There are few laboratory studies describing the effects of chronic inhalation exposure to sulfuric acid, however, observed effects included pulmonary damage, decreased airflow, and decreased bronchial clearance. In one study, monkeys were exposed for two years to 160 $\mu g/m^3$ of sulfur as 0.54 μM of sulfuric acid. This exposure resulted in moderate to severe pulmonary damage, and decreased airflow (Amdur et al., 1991). Bronchial clearance was decreased in both rabbits and donkeys following chronic exposure to concentrations of sulfuric acid ranging from 100 to 250 $\mu g/m^3$; a continued decrease was noted in both species for up to three months after the final exposure (Schlesinger et al., 1979; Gearhart and Schlesinger, 1988, 1989). Although there is uncertainty regarding the extrapolation of exposure concentrations from animals to humans, these studies suggest the possibility of respiratory injury due to chronic exposures to sulfuric acid. It is uncertain whether these exposures are relevant to human populations in the United States at current ambient sulfate concentrations.

4.3 ISSUES IN APPLYING EPIDEMIOLOGY RESULTS IN THIS ASSESSMENT

This quantitative assessment relies on concentration-response functions from the available epidemiology literature concerning human health effects associated with sulfate aerosols, and

in some cases $PM_{2.5}$. Available epidemiology results were selected for quantitative use in this assessment for the following reasons:

- Epidemiology results are based on studies of actual human health data and associated pollution exposures. Extrapolations from animal responses or from artificial clinical exposures are not necessary.
- A large available body of relevant epidemiology literature allows a quantitative assessment to be performed using a modest amount of research resources.
- Available epidemiology results cover a wide range of suspected health effects, including responses to long-term as well as short-term exposures.
- Reasonable assumptions can be employed when applying epidemiology results to calculate changes in health effects as a function of estimated changes in outdoor sulfate concentrations that allow the assessment to be conducted without doing detailed human exposure modeling.
- Epidemiology results are available for health effects that readily lend themselves to monetary valuation, such as premature mortality risks and self-reported symptoms.

The basic approach used in this assessment of applying epidemiology results to estimate health effects changes associated with estimated changes in outdoor air pollutant concentrations has been used in previous assessments for various air pollutants. These include EPA's Regulatory Impact Analyses for particulate matter and sulfur oxides (U.S. EPA, 1984, 1986c, 1988). Other assessments of the benefits of alternative pollution control strategies that used epidemiology results to estimate health benefits include Rowe et al. (in press), Krupnick and Kopp (1988), Hall et al. (1989), Harrison and Nichols (1990), and Thayer (1991).

Applying available epidemiology results to construct specific concentration-response functions for changes in ambient sulfate aerosol concentrations requires specific interpretations and assumptions. This section presents some of the key issues that must be considered, and explains the approaches chosen for this assessment. Whatever choices that are made on each of these specific issues, considerable uncertainty remains in the final results. Using these types of epidemiology results for quantitative assessments of health risks is not universally supported by all health researchers. Concerns exist about the accuracy of the estimated quantitative relationships in epidemiology studies, the fact that epidemiology studies can show an association but do not prove causation, and about transferring results from a specific study context to an assessment context that invariably has some different characteristics.

4.3.1 The Effects of Sulfates versus Other Particulates

Title IV requirements will result in a large reduction in SO_2 emissions, primarily from sources located in the eastern United States. Sources in the western United States are subject to the same emissions limits, but few sources in the western United States currently exceed the Title IV emissions limits. The reduction in SO_2 emissions in the eastern half of the United States will result in a significant reduction in ambient airborne concentrations of sulfate aerosols over a large geographic area. For this quantitative assessment, we want to know specifically how sulfate aerosols affect human health. However, a significant difficulty for this assessment is that epidemiology studies are limited in their ability to isolate the effects of sulfates from the effects of $PM_{2.5}$ as a whole. Because sulfates are a significant component of $PM_{2.5}$, the two pollutant measures are typically highly correlated (Ozkaynak and Thurston, 1987). Furthermore, only a few epidemiology studies have used data on sulfate concentrations as well as $PM_{2.5}$ concentrations so only a few direct comparisons of results are available. Most use one or the other measure.

Sulfate aerosols are a significant share of $PM_{2.5}$ in the United States. In the eastern United States, the ratio of average measured sulfate concentrations to average measured $PM_{2.5}$ concentrations is about 0.4 (Dockery et al., 1993). An important underlying issue in interpreting available epidemiology results for this assessment is whether sulfates are different from other fine particulates in terms of the amount or type of adverse health effects they cause. Although it is reasonable to expect that there may be differences, available information is not sufficient at this time to specify the differences for sulfates or any other common constituent of $PM_{2.5}$. Sulfate measures have been used in many epidemiology studies, but only a few studies have made a direct comparison of results obtained when a sulfate measure is used versus a $PM_{2.5}$ measure for the same location and study population. Several such studies have found statistically significant associations with the health endpoint for both pollutant measures (e.g., Pope et al., 1995; Dockery et al., 1993). Some of these studies have found a statistically stronger association between health effects and sulfates (e.g., Plagiannakos and Parker, 1988; Ostro, 1990) and others have found a statistically stronger association with more comprehensive measures of particulates such as $PM_{2.5}$ or PM_{10} (e.g., Dockery et al., 1992; Abbey et al., 1993a).

Two of these studies (Dockery et al., 1993; Ostro, 1990) have reported sulfate and $PM_{2.5}$ coefficients for the same population groups as well as mean concentrations of each pollutant measure in the study area. We can expect that if the sulfate coefficient fully reflects the effects of all $PM_{2.5}$, or is the sole causal constituent of $PM_{2.5}$, the ratio of the sulfate coefficient to the $PM_{2.5}$ coefficient should equal the inverse ratio of the sulfate and $PM_{2.5}$ concentrations. This is true for the Dockery et al. (1993) results for premature mortality, but not true for the Ostro (1990) results for respiratory restricted activity days. The latter suggest that there is an effect of $PM_{2.5}$ that is not fully reflected in the sulfate coefficient, but that the additional effect per unit $PM_{2.5}$ is about half that for sulfate. These results are suggestive at best, because of the high collinearity between the sulfate and $PM_{2.5}$ measures, and are not

sufficient for determining differences in potency between sulfate particulates and other constituents of $PM_{2.5}$.

The epidemiology evidence is abundant, however, that some or all of the constituents of $PM_{2.5}$, including sulfates, are harmful to human health. Clinical and laboratory studies provide evidence that at least some types of sulfate aerosols are harmful to the respiratory system when subjects are exposed to controlled amounts of sulfates alone. Thus, there is reason to believe that sulfates are contributing, at least in part, to the health effects observed in association with $PM_{2.5}$ and other particulate matter measures. The approach we take in this analysis to address this issue is three tiered:

- First, for health effects that have been statistically associated with sulfate concentrations, we select low, central, and high magnitudes of the estimated relationships between health effect incidence and sulfate concentrations.

- Second, for additional health effects that have been statistically associated with $PM_{2.5}$, we select low, central, and high magnitudes of the estimated relationships and apply them to the predicted changes in sulfate concentrations on the assumption that the estimated association between health effect incidence and $PM_{2.5}$ applies equally on a per $\mu g/m^3$ basis to sulfates, which are a substantial constituent of $PM_{2.5}$.

- Third, we use sensitivity analysis to determine how the results of the analysis would change if we were to assume that the estimated sulfate coefficients that form the basis of the health effects estimates in step one reflect the effects of $PM_{2.5}$ as a whole, not just sulfates, because of the typical collinearity between sulfates and $PM_{2.5}$.

For the sensitivity test on this question, we multiply all the sulfate coefficients by 0.4. This reflects an alternative assumption that the sulfate coefficient reflects the effects of other constituents of $PM_{2.5}$ as well as sulfates. This assumption and the first assumption (that the sulfate coefficients reflect the effects of sulfates only) most likely bound the "true" sulfate effect. The 0.4 adjustment is derived as follows. If we presume that sulfates and $PM_{2.5}$ are 100 percent correlated, then a coefficient estimated for a sulfate measure will reflect all the effects of the nonsulfate portion as well as the sulfate portion of $PM_{2.5}$. We might, for example, have the following estimated concentration-response relationship between a health effect (H) and sulfate levels (S), where B_s is the estimated sulfate coefficient:

$$H = B_s \times S. \qquad (4\text{-}1)$$

If, for example, B_s equals 4, this means that for every unit change in S there are 4 health effects observed. However, because of the collinearity between S and $PM_{2.5}$, B_s may actually reflect the effects of the 1 unit of S and the 1.5 units of collinear nonsulfate $PM_{2.5}$ (the ratio of measured sulfate to $PM_{2.5}$ being 0.4). Thus, if we change S by 1 unit and do not change

the nonsulfate particulates, we would obtain only a 1.6 unit change in H. Therefore, B_s must be multiplied by 0.4, to calculate the health effects associated with a 1 unit change in S alone.

4.3.2 Health Effects Thresholds

Another important uncertainty in this assessment is whether there is a threshold sulfate concentration below which health effects no longer occur, or whether the slope of the concentration-response function diminishes significantly at lower concentrations. Available epidemiological evidence is inconclusive on the question. No clear threshold has been determined, but such a determination is very difficult with typical epidemiological data. Most of the epidemiology studies reported here have estimated linear or log-linear functions that suggest a continuum of effects down to the lowest sulfate concentrations observed in the study sample, and have not attempted to identify a threshold concentration.

For this report, the default assumption adopted is that there is no threshold for health effects associated with ambient sulfate aerosols. In a practical sense, this does not mean that health effects are presumed to occur all the way down to zero sulfate concentrations because the changes in consideration (i.e., those due to Title IV) do not mean the elimination of all anthropogenic sulfate aerosols. If a threshold exists, however, it could have a significant effect on the accuracy of the results of this analysis. Depending on the level of the threshold relative to the estimated exposure concentrations, the existence of a threshold could reduce (but not increase) estimated health effects and benefits.

Because the evidence on whether, and at what concentration, there is a health effects threshold for sulfates remains inconclusive at this time, we report the results of some sensitivity analyses conducted using different assumptions regarding possible threshold concentrations for sulfate aerosols. We select two alternative threshold assumptions based on the low ends of the range of sulfate concentrations over which health effects have been estimated. The highest selected threshold for the sensitivity analysis is 5 $\mu g/m^3$. This is the mean sulfate concentration reported by Abbey et al. (1993a) for the Southern California study area for which a statistically significant association between the sulfate measure and chronic bronchitis incidence was not found. Another selected threshold is an annual average sulfate concentration of 3.6 $\mu g/m^3$, which is the lowest average sulfate concentration in the 151 cities included in the Pope et al. (1995) prospective cohort study on mortality rates in the United States. The third selected threshold for the sensitivity tests is 1.6 $\mu g/m^3$, which is the average sulfate concentration for 50 percent of the observations in the Southern Ontario study on hospital admissions (Burnett et al., 1995). This study reports a statistically significant difference for hospitalization rates between days with average sulfate concentrations of 1.6 $\mu g/m^3$ versus days with average concentrations of 4.13 $\mu g/m^3$ (the next 25 percent of the observations). This is not a direct test for a threshold, but it suggests that effects may occur at sulfate concentrations as low as 1.6 $\mu g/m^3$.

4.3.3 Uncertainty in the Estimates

The available epidemiology evidence regarding health effects associated with air pollutants, including sulfate aerosols, is subject to considerable uncertainty. Within a given study there is statistically measurable uncertainty in the estimated concentration-response coefficients, and there are differences in results obtained from different studies looking at the same or similar health effects. For each concentration-response relationship presented in this report, we have selected low, central, and high estimates. The central estimate is typically selected from the middle of the range reported in the study, or group of studies, that has been selected as providing the most reliable results for that health effect based on the study selection criteria discussed in Section 4.4.

These ranges of concentration-response values are not intended to reflect absolute upper and lower bounds, but rather ranges of estimates that are reasonably likely to be correct, given available health effects data. For example, ranges based on a single study are selected as plus and minus one confidence interval, not the absolute highest and lowest result obtained. When several different "reliable" studies are available for a given health effect, the selected range reflects the variation in results across the studies. The reader should be aware that there is analyst judgment in selecting these ranges and that the ranges do not reflect all the uncertainty in the concentration-response estimates because some of the uncertainty is not quantifiable. This is, however, an attempt to give a more realistic presentation than is given when only point estimates are reported.

Each low, central, and high estimate is also assigned a probability weight (the weights summing to 100 percent for each quantified health effect). These probability weights are used to propagate the uncertainty through the multiplication and aggregation process to total health benefit estimates. This provides an alternative to simply summing all the low estimates or all the high estimates to obtain total low and high estimates. Such simple summing can be misleading because it is highly unlikely that all the low estimates (or all the high estimates) are correct. When the low, central, and high estimates are based on results from different studies all judged as equally reliable, an equal probability weight is given to the low, central, and high estimates. When only one study is selected, the range used is plus and minus one standard error from the mean results of the study. When a statistical standard error is used, the probability weight given to the central estimate is 50 percent, with 25 percent each to the high and low estimates. In a few cases less weight has been given to a high or low estimate based on analyst judgment that there is reason to suspect that particular estimate is less likely to be correct than the other available estimates.

4.3.4 Interpretation and Aggregation of Daily Results

Many of the epidemiology studies that provide information about the health effects associated with particulate matter exposures have examined the daily incidence of a health effect such as

mortality or hospital admissions, and daily sulfate concentrations. The air quality modeling used in this analysis predicts changes in annual average sulfate concentrations, not changes in the daily concentration. Therefore, it is necessary to determine how changes in annual average sulfate concentrations contribute to daily health effects.

Two types of functional forms have been used in the daily epidemiology studies. One is a linear function, in which the estimated coefficient gives the number of additional cases each day as a function of changes in the daily pollution concentration. A linear function gives the following relationship:

$$\Delta C_i = R \times \Delta S_i \times POP, \tag{4-2}$$

where:

ΔC_i	=	additional cases on day i associated with a change in sulfate concentration
R	=	concentration-response coefficient between daily C and S
ΔS_i	=	change in sulfate concentration on day i
POP	=	affected population.

To obtain the number of cases each year, we sum Equation 4-2 over 365 days:

$$\sum_{i=1}^{365} \Delta C_i = R \times POP \sum_{i=1}^{365} (\Delta S_i). \tag{4-3}$$

If we multiply the right-hand side of Equation 4-3 by 365/365, we obtain:

$$\sum_{i=1}^{365} \Delta C_i = R \times POP \times 365 \sum_{i=1}^{365} \frac{(\Delta S_i)}{365}. \tag{4-4}$$

Thus, Equation 4-3 is equivalent to

Annual ΔC = R × POP × 365 × Annual average of daily changes in S.

The annual average of the daily changes in sulfate concentration is the same as the change in the annual average sulfate concentration. A linear coefficient for the daily number of cases due to sulfates, therefore, can be multiplied by 365 to obtain a coefficient for predicting the number of annual cases as function of the change in the annual average sulfate concentration.

The other common functional form is one in which the estimated coefficient gives the percentage change in the number of cases each day as a function of the daily pollution concentration. This gives the following relationship:

$$\Delta C_i/C' = R \times \Delta S_i \times POP, \quad (4\text{-}5)$$

where:

C' = the average daily number of cases of C due to all causes.

Equation (4-5) is simplified by substituting the average daily number of cases per individual. Once C' is moved to the right-hand side of Equation 4-5, ΔC_i can be estimated.

4.4 SELECTION OF CONCENTRATION-RESPONSE FUNCTIONS

This section provides a discussion of the specific epidemiological studies selected (based on the selection criteria discussed below) for quantitative use in this analysis. Concentration-response coefficients are selected from these studies. Ranges of concentration-response coefficients are given for each health effect category. The ranges are based on results from different studies when more than one equally applicable study is identified. All of the selected concentration-response functions are reported as functions of sulfate, based on studies that report health effects associated with sulfates or with $PM_{2.5}$.

4.4.1 Study Selection Criteria

Concentration-response functions were identified and adapted from the available epidemiology literature. These functions allow the estimation of the change in the number of cases of each health effect that would be expected as a result of changes in ambient sulfate concentrations. To be included as a basis for the concentration-response functions used in this assessment, an epidemiology study had to meet several specific criteria.

First, a proper study design and methodology were required. Studies were expected to have data based on continuous monitoring of the relevant pollutants, careful characterization and selection of exposure measures, and minimal bias in study sample selection and reporting. In addition, the studies had to provide concentration-response relationships over a continuum of relevant exposures. Second, studies that recognized and attempted to minimize confounding and omitted variables were included. For example, studies that compared two cities or regions and characterized them as "high" and "low" pollution areas were not used for quantitative purposes because of potential confounding by other factors in the respective areas and vague definition of exposure. Third, controls for the effects of seasonality and weather had to be included. This could be accomplished by stratifying and analyzing the data by season, by examining the independent effects of temperature and humidity, or by other statistical corrections.

A fourth criterion for inclusion was that the study had to include a reasonably complete analysis of the data. Such analysis would include a careful exploration of the primary hypothesis and preferably an examination of the robustness and sensitivity of the results to alternative functional forms, specifications, and influential data points. When studies reported the results of these alternative analyses, the quantitative estimates that we judged as most representative of the overall findings are those that we selected for use in this assessment. Finally, studies that addressed clinical outcomes or changes in behavior that would lend themselves to economic valuation were included. Estimates for endpoints such as changes in lung function, therefore, were not included.

4.4.2 Mortality

Over the last few decades, many epidemiologic studies have found statistically significant associations between sulfate concentrations (and other measures of particulate matter) and premature mortality among the general population. The earliest studies focused on relatively rare episodes of extremely high pollution concentrations in the 1940s and 1950s in the United States and in the United Kingdom (U.S. EPA, 1982). More recent studies have found an association at concentration levels typical of most metropolitan areas in North America.

The earliest studies of this type were cross-sectional studies examining annual mortality rates across U.S. cities with different average sulfate concentrations, often including 100 or more cities (e.g., Evans et al., 1984; Ozkaynak and Thurston, 1987). Very recently, two prospective cohort studies using individual-specific data and tracking mortality for a study sample in multiple cities over multiple years, also found an association between premature mortality and sulfate concentrations (Dockery et al., 1993; Pope et al., 1995). Time-series studies have also found statistically significant associations between daily mortality and daily fluctuations in sulfate concentrations (e.g., Dockery et al., 1992).

Some skepticism remains about whether these studies reflect a true causal relationship primarily because a biological mechanism to fully explain and verify this relationship has not been demonstrated in clinical or laboratory research (Utell and Samet, 1993). However, the epidemiologic studies are consistently finding a statistically significant association between sulfates and mortality, using different study designs and locations, and over a wide range of sulfate concentrations, including levels currently typical of many locations in the United States. It is therefore a reasonable exercise to estimate the reductions in premature mortality that might occur if sulfate concentrations were reduced, on the basis of the available epidemiologic results.

Summary of Selected Quantitative Evidence

This section does not provide a detailed review of all available literature, but focuses on results available in the literature that are best suited for the purposes of this analysis. The

study selection process relied on study selection criteria discussed in Section 4.4.1, and incorporated results from prospective cohort, single-period cross-sectional, and time-series studies. From all three perspectives the results show an association between mortality and sulfate concentrations, and results from all three types of studies are relied upon in selecting a range of risk estimates for use in this analysis.

Two types of long-term exposure studies have found statistically significant associations between mortality rates and particulate matter levels in the United States. The first type is an ecologic cross-sectional study design in which mortality rates for various locations are analyzed to determine if there is a statistical correlation with average air pollutant concentrations in each location. Such studies have consistently found measurably higher mortality rates in cities with higher average sulfate concentrations. However, concern persists about whether these studies have adequately controlled for potential confounding factors. Ozkaynak and Thurston (1987), Evans et al. (1984), and Chappie and Lave (1982) provide examples of ecologic cross-sectional studies. These studies each conducted a thorough examination of data for 100 or more U.S. cities, including average sulfate concentrations for each city, with special emphasis on the effects of including or excluding potential confounding factors such as occupations or migration. Plagiannakos and Parker (1988) combined annual cross-sectional data for 7 years for 9 counties in Ontario, Canada and also found an association between mortality rates and sulfate concentrations.

A second type of long-term exposure study is a prospective cohort study in which a sample is selected and followed over time in each location. In 1993, Dockery et al. published results for a 15-year prospective study based on samples of individuals in 6 cities. In 1995, Pope et al. published results of a 7-year prospective study based on samples of individuals in 151 cities in the United States. These studies are similar in some respects to the ecologic cross-sectional studies because the variation in pollution exposure is measured across locations rather than over time. These studies rely on the same type of pollutant exposure data as that used in the ecologic studies, which is average pollutant concentrations measured at stationary outdoor monitors in a given location. However, the mortality data are for identified individuals, which enables much better characterization of the study population and other health risks than when area-wide mortality data are used. Because they used individual-specific data, the authors of the prospective studies were able to control for premature mortality risks associated with differences in body mass, occupational exposures, smoking (present and past), alcohol use, age, and gender.

Dockery et al. (1993) found a mortality-rate ratio of 1.26 over the 15-year study period from the most polluted to least polluted city. Pope et al. (1995) found a mortality-rate ratio of 1.15 over the 7-year study period from the most polluted to least polluted city. Both of these findings were statistically significant.

The two prospective cohort studies represent a very important contribution to the study of premature mortality and sulfates (and other particulate matter measures) because the

prospective design using individually identified subject allows for better accounting of other risk factors for an individual that might be confounding factors when attempting to isolate the risk associated with air pollution exposure. The findings of a significant association between mortality and sulfate concentrations in this study are very supportive of the findings in previous single-year cross-sectional studies. The prospective studies provide evidence that long-term exposures to higher average sulfate (and other particulate matter) concentrations are associated with statistically significantly higher risks of premature mortality. However, due to limitations in the measure of exposure used in these studies, it is not possible to yet determine the specific length of exposure required to obtain this result, or whether there may be some latency between elevated exposure and elevated risk. This is because the studies have used measures of sulfate and other particulate matter concentrations at the beginning of or during part of the study period as the measure of exposure. Lifetime cumulative exposures are not known. Current period concentrations are probably correlated with lifetime exposures for individuals residing in a given location, but quantitative extrapolation from the results based on this exposure measure are uncertain.

The results of the two prospective studies and four selected cross-sectional studies are summarized in Table 4-1. Results are reported in terms of the estimated percentage change in mortality in the study sample for every $\mu g/m^3$ change in average sulfate concentrations. For example, the Pope et al. results for 151 U.S. cities indicate that for every one $\mu g/m^3$ increase in average sulfate concentrations where subjects live is associated with a 0.75 percent increase in observed mortality in the 7-year study period. The cross-sectional studies typically report results from many different specifications of the mortality regressions, because the intent of some of these studies was to test for the effect of changes in the specification. The results reported here are selected from the middle to low end of ranges of results reported, and are drawn from specifications that include the significant explanatory variables identified in addition to the air pollutant measures.

The results with respect to sulfates fall between 0.3 percent and 1.4 percent, with the exception of the sulfate result for the 6-cities prospective study, which is substantially higher. The results of the prospective studies are generally equal to or higher than the results of the cross-sectional studies, which supports that the cross-sectional results are meaningful, not just spurious statistical associations, and suggests that more accurate accounting of individual mortality risks results in greater risk attributed to air pollution exposure. This conclusion is tentative until more prospective cohort studies have been completed and continue to verify this finding.

In some studies the premature mortality result is also analyzed per unit of $PM_{2.5}$, and this is also shown in Table 4-1. When estimates are reported for both pollutant measures, these are based on estimates that do not account the other pollutant measure. They should therefore be interpreted as different measures of the same health effect based on different but highly collinear measures of fine particulate concentrations. For the Pope et al. results, the ratio of the effects of sulfate to $PM_{2.5}$ exceeds the inverse of the ratio of the mean concentrations of

Table 4-1
Comparison of Selected Mortality Study Results

Study	Study Design	Time Period	Study Location	% Change in Mortality per $\mu g/m^3$	
				SO_4	$PM_{2.5}$
Pope et al. (1995)	Prospective Cohort	1982-1989	50 U.S. cities 151 U.S. cities	 0.75%	0.69%
Dockery et al. (1993)	Prospective Cohort	1974-1989	6 U.S. cities	3.25%	1.40%
Ozkaynak and Thurston (1987)	Cross-Sectional	1980	98 U.S. cities	0.77%	
Evans et al. (1984)	Cross-Sectional	1960	98 U.S. cities	0.29%	
Chappie and Lave (1982)	Cross-Sectional	1960 1969 1974	117 U.S. cities 112 U.S. cities 102 U.S. cities	0.50% 0.54% 1.37%	
Plagiannakos and Parker (1988)	Cross-Sectional	1976-1982	9 Ontario counties	0.50%	

each measure in the study areas. This suggests that the sulfate effect exceeds the $PM_{2.5}$ effect on a per $\mu g/m^3$ basis, but suggests that there are additional effects picked up by the $PM_{2.5}$ coefficient that are not fully reflected in the sulfate coefficient. The Dockery et al. results, however, suggest that there may be no additional $PM_{2.5}$ effects other than those reflected in the sulfate coefficient.

There have also been a substantial number of daily time-series studies examining the relationship between daily mortality and daily particulate matter concentrations in many cities in North America. Dockery and Pope (1994) review and summarize these studies. These studies have for the most part used TSP or PM_{10} as the measure of particulate concentration. One time-series study (Dockery et al., 1992) reports a sulfate coefficient of 0.6 percent change in daily mortality per $\mu g/m^3$ sulfate, which is within the range of results reported in Table 4-1. Dockery and Pope report that overall, the results of the time-series studies range from 0.05 percent to 0.15 percent higher mortality for every $\mu g/m^3$ increase in 24-hour PM_{10}. This range falls just below the range of results reported for sulfates in Table 4-1 from the cross-sectional and prospective studies.

Evidence on Who is at Risk

The results of a time-series study in Philadelphia (Schwartz and Dockery, 1992a) provide estimates of elevated mortality risks separately for those over and under 65 years old. These results suggest that about 90 percent of the premature deaths associated with particulate matter occur in the over-65 group. This finding is consistent with the results of an early cross-sectional mortality study (Lave and Seskin, 1977). Ostro et al. (in press) found that about 80 percent of the premature deaths associated with particulate matter were in the over-65 group in their Santiago, Chile, study. In the United States, about 70 percent of all deaths are individuals 65 years old or older, so it appears that risks associated with air pollution exposure fall in somewhat greater proportion to the elderly.

As discussed in Chapter 5, the age of the individual at risk of premature mortality may have some bearing on the monetary value of changing that risk. For the purposes of this analysis, it is presumed based on evidence in Ostro et al. (in press) and Schwartz and Dockery (1992a) that 85 percent of the individuals at risk of premature mortality associated with sulfate exposures are 65 years old or older.

The results from Pope et al. (1995) show that the greatest association is with deaths associated with cardiopulmonary illness, and that elevated mortality risks are similar for both smokers and nonsmokers in higher pollution locations. Some of the time-series studies (e.g., Schwartz and Dockery, 1992a) have also found significant cause-specific mortality associations indicating that most pollution-associated deaths are cardiopulmonary related. Some of those at risk therefore probably suffer from chronic diseases that might be expected to shorten life expectancy even in the absence of air pollution. This does not, however, rule out the possibility that some of these chronic illnesses could themselves be related to air pollution exposure.

Estimation Approach for this Analysis

For this analysis, the epidemiologic results are being used to predict how mortality rates may change given a change in ambient sulfate concentrations. For this purpose, we select a range of results from the three types of mortality studies. Premature mortality is a very serious health endpoint and there is a large body of epidemiologic literature that has studied mortality as it relates to air pollutant exposure. However, there remain many uncertainties in specific quantitative interpretations of the results of the epidemiologic studies that have studied the association between premature mortality and sulfate concentrations. We therefore select a wider range of findings than those selected for most of the other health endpoints quantified in this assessment.

We select a range of four estimates to reflect the range of results obtained in the mortality studies. For a lowest estimate, we select the 0.1 percent mortality effect found for PM_{10} in the many time-series studies. This is at the low end of the range of mortality effects estimated

and because it is based on PM_{10}, applying it to an estimated change in sulfate concentration presumes that a sulfate aerosol is no more harmful than a typical PM_{10} aerosol. We select a low-central estimate of 0.3 percent based on the low end of the cross-sectional results for sulfates. We select a high-central estimates of 0.7 percent based on the Pope et al. prospective study. This is still within the range of the cross-sectional results. As a high estimate we select 1.4 percent based on the $PM_{2.5}$ results of the 6-cities study and the highest cross-sectional result reported in Table 4-1. Although there are results from some studies that are both lower and higher than this range (e.g., some time-series studies find 0.05 percent or less and the 6-cities result for sulfates is greater than 3 percent), a very large share of the findings for sulfates fall into this range. We give equal probability weights (25%) to all four of the selected risk estimates.

The selected percentage changes in mortality must be multiplied by average annual mortality to calculate the change in annual premature deaths per change in annual average sulfate concentrations. For this we use the average U.S. nonaccidental mortality rate of about 8,000 per million population per year (U.S. Bureau of the Census, 1994). For example, the low-central estimate is 0.3 percent of 8,000 divided by 1,000,000. The selected mortality risk coefficients and calculation procedures are thus:

Low annual SO_4 premature mortality	$= 8 \times 10^{-6} \times POP_j \times (\Delta S_j)$	(4-6a)
Low-central annual SO_4 premature mortality	$= 24 \times 10^{-6} \times POP_j \times (\Delta S_j)$	(4-6b)
High-central annual SO_4 premature mortality	$= 56 \times 10^{-6} \times POP_j \times (\Delta S_j)$	(4-6c)
High annual SO_4 premature mortality	$= 112 \times 10^{-6} \times POP_j \times (\Delta S_j)$	(4-6d)

where:

POP_j = total population in area j
ΔS_j = change in annual average sulfate concentration in area j.

4.4.3 Chronic Respiratory Disease

For more than two decades, there has been some evidence suggesting that higher ambient particulate matter exposures are associated with higher rates of chronic respiratory disease. Much of this evidence, however, has been based on cross-sectional analyses, comparing disease or symptom prevalence rates in different communities with different average pollution levels (e.g., Ferris et al., 1973, 1976; Hodgkin et al., 1984; Portney and Mullahy, 1990). These studies are able to suggest a possible association, but are difficult to use for quantitative estimates of specific concentration-response functions. This difficulty stems primarily from uncertainty about how to characterize the relevant exposure units, in particular the time aspects of exposure. Chronic symptoms presumably occur as a result of long-term exposures, but cross-sectional analyses are not very enlightening about whether, for example, it is the five-year average, the twenty-year average, or the number of times a given level is exceeded

that is the relevant exposure measure. Without this information, it is difficult to predict quantitatively how risks change when exposures change.

Recently published articles (Abbey et al., 1993a, 1993b, 1995) have reported results of a 10-year prospective cohort study conducted at Loma Linda University in California with a large sample of nonsmoking adults. This follow-up allowed for measures of exposure preceding and during the 10-year study period and for obtaining information on changes in chronic respiratory disease incidence over time. Thus, development of new cases of disease were analyzed in relation to individual-specific air pollution exposure history. This study provides for the first time a more definitive concentration-response function for chronic respiratory disease. Uncertainty about the potential effect of exposures that preceded the study period, and lag times between exposure and illness onset still exists with these findings.

The Loma Linda University Study

In the first stage of the Loma Linda University study, a large sample (approximately 7,000) of Seventh Day Adventists (selected because they do not smoke), was interviewed in 1977. Health histories, current respiratory symptoms, past smoking and passive smoking exposure, and residence location histories were obtained. Hodgkin et al. (1984) compared the chronic respiratory disease status of respondents who had lived for at least 11 years in either a high or a low pollution area in Southern California. After adjusting for sex, race, age, education, occupational exposure, and past smoking history, residents of the higher pollution area were found to have a prevalence of airway obstructive disease (AOD) (including chronic bronchitis, asthma and emphysema) that was 15 percent higher than for residents in the low pollution area. Using the same 1977 Loma Linda sample, Euler et al. (1987) report results showing a statistically significant association between past TSP exposure, based on residence zip-code history, and the prevalence of chronic respiratory disease.

Abbey et al. (1993a, 1993b, 1995) report the results of a cohort study with the Seventh Day Adventist sample in 1987, which provides better quantitative concentration-response information. Nearly 4,000 subjects were interviewed in 1987 who had been interviewed previously in 1977. All were 25 years old or more in 1977. Estimates of air pollutant exposures histories were developed based on subjects' reported residence locations from 1967 to 1987 and pollutant measures from stationary outdoor monitors closest to each residence location over the study period. Abbey et al. (1993b) report results of the cohort study based on TSP data from 1973 to 1987. Abbey et al. (1995) added data on $PM_{2.5}$, based on airport visibility data from 1967 to 1987, sulfate data from 1977 to 1987, and data on gaseous air pollutants including ozone, nitrogen dioxide, and sulfur dioxide.

Several different health outcomes were examined including new cases of emphysema, chronic bronchitis, or asthma, in 1987 for those not reporting any definite symptoms of these diseases in 1977. Disease definition was based on self-reported symptoms using the standardized respiratory symptoms questionnaire developed by the National Heart and Lung Institute for

the United States. Respondents were classified as having *definite* symptoms of emphysema, chronic bronchitis or asthma if they met specific criteria for the disease diagnosis. Having definite symptoms of any one of these three was defined as definite airway obstructive disease (AOD). Having definite chronic bronchitis was defined as having symptoms of cough and/or sputum production on most days for at least 3 months/year, for 2 years or more. Emphysema and asthma required a physician's diagnosis as well as associated symptoms.

Logistic models were estimated for mean concentrations of air pollutants and for hours above selected levels for each pollutant. The regressions included independent variables for past and passive smoking exposure, possible symptoms in 1977, childhood respiratory illness, gender, age and education. Abbey et al. (1993b) report a statistically significant association between average long-term TSP exposure levels and AOD, as well as with chronic bronchitis alone.

Abbey et al. (1995) report no statistically significant associations between the gaseous pollutants and the development of new cases of chronic respiratory disease, although aggravation of existing disease was apparent, specially for asthma in relation to ozone exposure. More important, the authors conclude that exposures to gaseous pollutants did not appear to be a significant confounding factor in the measured association between particulate matter exposure and incidence of chronic respiratory disease.

Abbey et al. (1995) report statistically significant associations between TSP exposure and new cases of AOD, as well as with new cases of chronic bronchitis and new cases of asthma (which are two types of AOD); and the magnitude of the TSP results was consistent with the previous reported results (Abbey et al., 1993b). The authors also report a statistically significant association between new cases of chronic bronchitis and the $PM_{2.5}$ measure, and between new cases of asthma and the sulfate measure. The magnitudes of the reported odds ratios for new cases of AOD were similar for selected changes in TSP, $PM_{2.5}$, and sulfates, but the result was statistically significant only for the TSP measure. The authors note that there is probably more measurement error in the $PM_{2.5}$ exposure estimates because of the approximation from airport visibility, and in the sulfate exposure estimates because they were based on data from 1977 to 1987 only.

Abbey et al. (1995) also report evidence that increased severity of AOD is statistically significantly associated with TSP, $PM_{2.5}$, and sulfate exposure for those who reported definite symptoms in 1977. Thus, it appears that particulate matter exposure both aggravates existing cases and causes new cases.

Selected Chronic Bronchitis Risk Estimates from Abbey et al. (1995)

We have selected the chronic bronchitis results from Abbey et al. (1995) for $PM_{2.5}$ for quantification of changes in risks of developing chronic bronchitis in this analysis. The estimates used in this analysis reflect only the development of new cases, not the aggravation of existing cases. The key assumption in this application of the $PM_{2.5}$ results is that sulfates

contribute to this risk at an equal level per $\mu g/m^3$ as other constituents of $PM_{2.5}$. This assumption is partially, but not fully, supported by the Abbey et al. (1995) results. Limitations in both the $PM_{2.5}$ and the sulfate data available for this analysis contribute to the ambiguity in the findings. The quantitative interpretation of this assumption is to apply the risk associated with each $\mu g/m^3$ of $PM_{2.5}$ to the estimated $\mu g/m^3$ change in sulfate concentration without any adjustment to the risk value due to the difference in the particulate measure. Implicit in this is the assumption that sulfates contribute to the $PM_{2.5}$ effect only in proportion to their share of total $PM_{2.5}$ and that other constituents of $PM_{2.5}$ are equally as harmful.

The failure to find a statistically significant relationship between sulfate concentrations and new cases of chronic bronchitis is somewhat troubling with respect to this quantification approach, but it is offset to some extent by the finding of a significant relationship between sulfates and new cases of asthma (another type of AOD) and by the fact that the magnitude of the estimated relationship between AOD as a whole and sulfates is similar to the magnitude estimated for TSP and for $PM_{2.5}$, even though the statistical significance of this relationship was low for the sulfate and $PM_{2.5}$ measures.

Using the $PM_{2.5}$ results for chronic bronchitis in this assessment gives a lower risk per $\mu g/m^3$ than would have been obtained using other feasible quantification approaches based on the Abbey et al. (1995) results. For example, if we applied the estimated relative risk estimate for new AOD cases reported by Abbey et al. (1995) for 7 $\mu g/m^3$ of sulfate and attributed this risk to sulfate alone, the risk coefficient per $\mu g/m^3$ would be about 3 times higher than the selected central estimate based on the $PM_{2.5}$ results. Alternatively, if we used the statistically significant relative risk for new cases of asthma associated with a 7 $\mu g/m^3$ increment of sulfate, the risk coefficient per $\mu g/m^3$ would be about 5 times higher than the estimate based on the $PM_{2.5}$ chronic bronchitis results. Thus, although there is uncertainty in applying the $PM_{2.5}$ results, it is unlikely that they overstate the effect of sulfates on new cases of AOD as a whole.

Abbey et al. report a relative risk of 1.81 for developing a new case of chronic bronchitis during the 10-year follow-up period for an increase in average $PM_{2.5}$ exposure of 45 $\mu g/m^3$. This means that the incidence of new cases of chronic bronchitis is 81 percent higher in locations with average $PM_{2.5}$ concentrations 45 $\mu g/m^3$ higher, or 1.8 percent higher for every 1 $\mu g/m^3$ increase in average $PM_{2.5}$ concentrations. The 10-year incidence of new cases of chronic bronchitis was about 6 percent (117 ÷ 1,868 in the subsample for which $PM_{2.5}$ exposures were estimated). Thus, an individual's probability of developing chronic bronchitis in the 10-year period is $0.018 \times 0.06 = 0.0011$ per 1 $\mu g/m^3$ increase in average $PM_{2.5}$ concentration. We divide this individual risk by 10 to obtain an annual risk of developing chronic bronchitis. The high and low estimates are based on plus and minus one standard error of the estimated risk relationship. The selected low, central, and high estimates for changes in chronic bronchitis are thus:

$$\text{Low annual cases of CB} = 0.5 \times 10^{-4} \times POP_{>25j} \times (\Delta S_j) \quad (4\text{-}7a)$$
$$\text{Central annual cases of CB} = 1.1 \times 10^{-4} \times POP_{>25j} \times (\Delta S_j) \quad (4\text{-}7b)$$
$$\text{High annual cases of CB} = 2.0 \times 10^{-4} \times POP_{>25j} \times (\Delta S_j) \quad (4\text{-}7c)$$

where:

CB	=	adult chronic bronchitis
$POP_{>25j}$	=	population over age 25 years in area j
ΔS_j	=	change in annual average sulfate concentration in area j.

We apply the risk estimates to the adult population age 25 and over because this is the minimum age in the Abbey et al. study group. Chronic bronchitis takes awhile to develop and these risk estimates may not apply to younger individuals.

4.4.4 Acute Morbidity

Epidemiology studies have found health effects associated with ambient sulfates ranging from elevated rates of hospital admissions to small differences in lung function measurements. The studies selected as the basis for quantitative estimates for this report provide evidence with clear clinical significance; i.e., the effects are noticeable to subjects. This means symptoms that are noticeable to the subject and can be expected to have some impact on the individual's well-being. For this reason, we have not included studies that look only at effects on lung function. Although this may be a medically relevant health endpoint, it cannot at this time be translated into changes in symptoms or illness that can be readily valued.

Respiratory Hospital Admissions

Recent evidence indicates an association between ambient sulfates and both respiratory hospital admissions (RHAs) and cardiac hospital admissions (CHAs). Evidence of a relationship between RHAs and CHAs and sulfates, controlling for collinear ozone concentrations, is provided by Burnett et al. (1995) for Ontario, Canada. Additional evidence of a relationship between RHAs and sulfates is provided by Thurston et al. (1994) for Toronto, and by Thurston et al. (1992) for selected cities in New York. For this analysis, specific quantitative estimates are derived from the Burnett et al. (1995) Ontario study because they are for both RHAs and CHAs. The Thurston et al. studies are examined for supporting evidence, but are not used quantitatively because their results are less amenable for providing separate associations for sulfates and ozone. Supporting evidence for an effect of particles on cardiac hospital admissions is provided by Schwartz and Morris (1995).

Burnett et al. (1995) studied the relationship between hospital admissions for respiratory and cardiac disease and both sulfate and ozone from 1983 through 1988 in Ontario, Canada. Air pollution data were obtained from a large network of monitors existing throughout Ontario.

Admissions data from 168 acute care hospitals in Ontario below the 47th parallel were used. After elective admissions were excluded, counts of daily admissions for all ages and for age-specific and disease-specific categories were created. A time-series regression model was used that removed the influences of day-of-week effects, slow moving serial correlations due to seasonal patterns, and differences between hospitals. Ultimately, the effects of air pollution on deviations in the expected number of admissions to each hospital on any given day were estimated. Regression models included temperature effects and were specified with ozone and sulfate considered alone and together as explanatory variables. The results indicated that one-day lags of both ozone and sulfates were associated with respiratory admissions, and that sulfates, but not ozone, were associated with cardiac admissions. The sulfate effects were observed in both the summer and winter quarters, both males and females, and across all age groups (Burnett et al., 1995).

Thurston et al. (1992, 1994) provide supporting evidence of an association between RHA during summer months and either sulfate or ozone concentrations, or both. They do not report results for models that include both ozone and sulfate, so their results for both pollutants are likely confounded by the presence of the other correlated pollutant. However, the results are useful for rough comparison to the Burnett et al. results. Burnett et al. (1994) found that the mean sulfate concentration was associated with a 2.2 percent increase in daily summer RHAs when only sulfate was included in the model, and that the mean ozone concentration was associated with a 6.0 percent increase in daily summer RHAs when only ozone was included in the model. The single pollutant results are similar to results obtained by Thurston et al. (1992) for New York City, which were 3.5 percent for mean sulfate and 5.3 percent for mean ozone. These estimates are also reasonably consistent with the findings obtained in the Toronto study (Thurston et al., 1994).

Bates and Sizto (1989) provide some additional evidence on the issue. They estimated a stepwise regression for respiratory hospital admissions during the summer months in Ontario. First they included temperature, which explained 0.89 percent of the variance in RHA. Then they added sulfate, which increase the explained variance to 3.3 percent. When ozone was then added, the explained variance increased to 5.6 percent. This suggests that adding ozone to the regression explains about as much of the variance as that explained by the sulfate variable.

Low, central, and high estimates of RHAs associated with sulfates are selected based on the results of Burnett et al. (1995). Results were selected from a model that included both sulfates and ozone in the regression, to reduce the chance of overstating the sulfate effect because of the collinearity between sulfates and ozone in the study area. We apply a 50 percent probability to the central estimate, and 25 percent each to the low and high estimates, which are the central minus and plus one standard error. Specifically, Burnett et al. (1995) report a 3.5 percent increase in RHAs for a 13 $\mu g/m^3$ increase in sulfate when ozone was included in the model. The average daily RHA for the study period was 16.0 per million population. Thus, 3.5 percent of the 16.0 daily RHA are attributed to 13 $\mu g/m^3$ sulfate. Therefore, the

daily RHA per 1 $\mu g/m^3$ sulfate is: $0.035 \times (16.0 \times 10^{-6}) \div 13 = 4.31 \times 10^{-8}$. We multiply by 365 to obtain the estimated annual number of RHAs for a change in annual average sulfate concentration. The central estimate of changes in RHA incidence is thus as follows, with the low and high selected as the central minus and plus one standard error:

$$\text{Low annual RHA} = 1.3 \times 10^{-5} \times \Delta S_j \times POP_j \quad (4\text{-}8a)$$
$$\text{Central annual RHA} = 1.6 \times 10^{-5} \times \Delta S_j \times POP_j \quad (4\text{-}8b)$$
$$\text{High annual RHA} = 1.8 \times 10^{-5} \times \Delta S_j \times POP_j \quad (4\text{-}8c)$$

where:

POP_j = total population in area j
ΔS_j = change in annual average sulfate concentration.

Burnett et al. (1995) also reported a statistically significant association between sulfates and cardiac hospital admissions (CHA) throughout the year, while no association was found for ozone. Burnett et al. (1995) report a 3.3 percent increase in CHAs for a 13 $\mu g/m^3$ increase in sulfate when ozone was included in the model. Thus, 3.3 percent of the average daily CHAs per million population (14.4) in the study area gives the number of additional daily CHAs per 13 $\mu g/m^3$ sulfate. Dividing by 13 gives the daily CHAs per $\mu g/m^3$ sulfate [$0.033 \times (14.4 \times 10^{-6}) \div 13 = 3.66 \times 10^{-8}$]. We multiply by 365 to obtain the estimate annual number of RHAs for a change in annual average sulfate concentration. We apply a 50 percent probability to the central estimate, and 25 percent each to the low and high. The central estimate of CHAs is thus as follows, with the low and high selected as minus and plus one standard error of the central estimate:

$$\text{Low annual CHA} = 1.0 \times 10^{-5} \times \Delta S_j \times POP_j \quad (4\text{-}9a)$$
$$\text{Central annual CHA} = 1.3 \times 10^{-5} \times \Delta S_j \times POP_j \quad (4\text{-}9b)$$
$$\text{High annual CHA} = 1.7 \times 10^{-5} \times \Delta S_j \times POP_j. \quad (4\text{-}9c)$$

Aggravation of Asthma Symptoms

Several studies have related particulate matter concentrations to exacerbation of asthma symptoms in individuals with diagnosed asthma. Ostro et al. (1991) report results specifically for day-to-day fluctuations in sulfate concentrations. Ostro et al. had subjects record daily asthma symptoms during the duration of the study. An aggravation of asthma symptoms was defined for each subject based on each individual's manifestation of asthma symptoms. This typically meant a notable increase in symptoms, such as shortness of breath or wheezing, and/or in use of medication relative to what was "normal" for that individual. Daily air pollution concentrations were then examined for correlations with day-to-day fluctuations in asthma symptom frequency, controlling for other factors such as weather and previous-day symptoms.

Ostro et al. (1991) examined the association between several different air pollutants, including sulfates, $PM_{2.5}$, and acidic aerosols, and aggravation of asthma symptoms among adults during winter months in Denver. A significant association was found between the probability of moderate or severe asthma symptoms (measured as shortness of breath) and sulfate particulate concentrations, after controlling for temperature, day of study, previous-day illness, and use of a gas stove. Ozone concentrations were very low, near background concentrations, and do not create a confounding influence. The results suggest the following relationship in the winter months between sulfates and aggravation of asthma symptoms (A).

$$\text{Change in daily probability of A} = [0.0077\ (\pm\ 0.0038)/S] \times \Delta S \qquad (4\text{-}10)$$

Using the reported sulfate mean for the study of 2.11 $\mu g/m^3$ to linearize the function yields the following calculation procedure to estimate daily probability of asthma symptoms per asthmatic based on the Ostro et al. results.

$$\text{Change in daily probability of A} = [0.0036\ (\pm\ 0.0018)] \times \Delta S \qquad (4\text{-}11)$$

There may be an upward bias in the Ostro et al. (1991) results because the data were collected during winter months only. Winter months in Denver are also a period of more frequent respiratory colds that also aggravate asthma symptoms and may in turn cause asthmatics to be more sensitive to air pollutants. We therefore assume for the purposes of this analysis that the measured relationship between aggravation of asthma symptoms and sulfate concentrations applies during only half of the year. To annualize the relationship we therefore multiply by 182.5 rather than by 365.

Using an estimate of 4.7 percent for the portion of the U.S. population with diagnosed asthma (National Center for Health Statistics, 1992) yields the following calculation procedure to estimate annual number of asthma attacks based on the selected Ostro et al. (1991) results.

$$\text{Low annual ASD} = 3.3 \times 10^{-1} \times (\Delta S_j) \times POP_j \times 0.047 \qquad (4\text{-}12a)$$

$$\text{Central annual ASD} = 6.7 \times 10^{-1} \times (\Delta S_j) \times POP_j \times 0.047 \qquad (4\text{-}12b)$$

$$\text{High annual ASD} = 9.9 \times 10^{-1} \times (\Delta S_j) \times POP_j \times 0.047 \qquad (4\text{-}12c)$$

Restricted Activity Days

Restricted activity days (RADs) include days spent in bed, days missed from work, and days when activities are partially restricted due to illness. Ostro (1987) examined the relationship between adult all-cause RADs in a two-week period and $PM_{2.5}$ in the same two-week period for 49 metropolitan areas in the United States. The RAD data were from the Health Interview Survey (HIS) conducted annually by the National Center for Health Statistics. The $PM_{2.5}$ data were estimated from visual range data available for airports in each area. Since fine particles have a more significant impact on visual range than do large suspended particles, a direct relationship can be estimated between visual range and $PM_{2.5}$.

Separate regression estimates were obtained for 6 years, 1976 to 1981. A statistically significant relationship was found in each year and was consistent with earlier findings relating RADs to TSP by Ostro (1983). The mean of the estimated coefficient for $PM_{2.5}$ across the 6 years indicated approximately 91,000 RAD each year per 1 million population for each $\mu g/m^3$ increase in annual average $PM_{2.5}$, and ranged from a low of 53,000 for the 1981 coefficient to a high of 171,000 for the 1976 coefficient.

Additional work conducted by Ostro and Rothschild (1989) added ozone measures to the regressions and found the estimated relationship between RADs and $PM_{2.5}$ to be essentially unchanged. This suggests that the RAD/$PM_{2.5}$ relationship was not confounded by the exclusion of ozone concentrations and is independent of ozone exposures. The newer work also estimated the relationship between respiratory RAD (RRAD) and $PM_{2.5}$ for employed individuals only. It was expected that this relationship might be more stable than that between all-cause RAD and $PM_{2.5}$ for all adults for two reasons: (1) it is expected that pollution induced RADs might be predominantly related to respiratory illness, and (2) workers might define a RAD more consistently than the entire adult population. It was expected, though, that confining the data to RRADs for workers might result in a smaller total number of predicted restricted activity days for a given concentration of pollution, because all effects might not be classified as respiratory and workers may be a healthier and therefore less sensitive group, on average, than all adults. The findings are consistent with this expectation. The average of the $PM_{2.5}$ coefficients for the 6 years suggested an annual increase of approximately 47,000 RRAD per 1 million population for each $\mu g/m^3$ increase in annual average $PM_{2.5}$, and ranged from a low of 31,000 for the 1978 coefficient to a high of 55,000 for the 1980 coefficient.

Ostro (1990) reports results also using data on RRADs for working adults. In this analysis he matched data from EPA's Inhalable Particles Monitoring Network on sulfates and $PM_{2.5}$, based on particulate monitors, with the HIS data for 1979 to 1981. Data on 25 cities resulted and the analysis shows statistically significant relationships between RRAD incidence and both sulfate and $PM_{2.5}$, in separate regressions as necessitated by the collinearity between the two measures of fine particulate. The quantitative results were quite comparable to the Ostro and Rothschild (1989) results for RRADs for working adults, and were also reasonably similar for sulfates and $PM_{2.5}$. Estimated annual RRADs per million population (of working adults) was approximately 56,000 per $\mu g/m^3$ sulfate or 42,000 per $\mu g/m^3$ $PM_{2.5}$.

For this analysis, we calculate changes in RAD incidence as a function of changes in ambient sulfate concentrations based on the estimated relationship between RADs and $PM_{2.5}$. The Ostro (1990) results suggest that this is a reasonable assumption, the effect of which may be to slightly understate the sulfate effect. We choose to use the $PM_{2.5}$ results for quantitative purposes because the sulfate results are available for only a subset of RADs (i.e., RRADs for working adults).

The mean results over the 6 years from Ostro (1987) for all-cause RADs for all adults (mean coefficient = 0.0048) have been selected as the central estimate for this analysis. The mean

results from Ostro and Rothschild (1989) for respiratory RADs for workers (mean coefficient = 0.0158) were selected for the low estimate. This is a low estimate because it excludes some nonrespiratory RADs that might be related to pollution exposures and is based on a healthier than average sample (i.e., workers). The selected high estimate is the mean of the two highest coefficients in the six year analysis (mean coefficient = 0.0076) by Ostro (1987). The reported coefficients give percentage changes in RADs or RRADs for a 1 $\mu g/m^3$ change in $PM_{2.5}$. Daily average estimates from the studies based on HIS data of 0.052 RAD and 0.0083 RRAD per person are used to determine the relationship between number of RADs and $PM_{2.5}$. For example, the central daily individual risk estimate is thus:

$$0.0048 \times 0.052 = 2.5 \times 10^{-4}. \quad (4\text{-}13)$$

Multiplying by 365 to estimate annual changes in RAD incidence we obtain the following low, central and high estimates for changes in annual average sulfate concentrations. The calculations are applied to the adult population 18 years and over.

$$\text{Low annual RAD} = 4.7 \times 10^{-2} \times \Delta S_j \times POP_{\geq 18j} \quad (4\text{-}14a)$$

$$\text{Central annual RAD} = 9.3 \times 10^{-2} \times \Delta S_j \times POP_{\geq 18j} \quad (4\text{-}14b)$$

$$\text{High annual RAD} = 14.6 \times 10^{-2} \times \Delta S_j \times POP_{\geq 18j} \quad (4\text{-}14c)$$

where:

$POP_{\geq 18j}$ = population in location j 18 years of age and older.

Acute Lower Respiratory Symptoms

Krupnick et al. (1990) and Ostro et al. (1993) report analyses of relationships between the daily incidence of acute upper and lower respiratory symptoms among a general population panel of adults in Southern California and daily concentrations of air pollution. These health endpoints include some days with symptoms bothersome enough to result in a restricted activity day, but also include days when noticeable symptoms are present but no change in activities occurs. The statistical analyses incorporated the presence of illness on the prior day, presence of chronic respiratory disease, daily weather conditions, indoor air pollution sources, and controlled for autocorrelation.

The air pollution measures used in the Krupnick et al. analysis were coefficient of haze (COH), a measure of the visibility impairing particulates in the air, and ozone. Krupnick et al. report a statistically significant relationship between daily COH and the daily incidence of respiratory symptoms (upper and lower combined), after controlling for a statistically significant ozone effect. Ostro et al. (1993) conducted separate analyses for upper and lower respiratory tract symptoms, and added sulfates as a measure of daily particulate matter in the study area in place of the COH measure. They continued to find a statistically significant association between daily ozone and both kinds of symptoms. They found a statistically

significant relationship between daily sulfate concentrations and lower respiratory symptoms only, after controlling for ozone. We select these results for quantitative use in this assessment of changes in sulfate concentrations.

Ostro et al. (1993) report an odds ratio for incidence of lower respiratory symptoms in adults of 1.30 for a 10 $\mu g/m^3$ increment of sulfates. The average daily incidence of lower respiratory symptoms is 1.5 percent in the study sample. Thus, the average daily individual probability of having lower respiratory symptoms is $0.03 \times 0.015 = 4.5 \times 10^{-4}$ per $\mu g/m^3$ sulfate. To annualize we multiply by 365. The low and high estimates are based on minus or plus one standard error of the regression coefficient.

$$\text{Low annual LRS} = 6.6 \times 10^{-2} \times (\Delta S_j) \times POP_{\geq 18j} \quad (4\text{-}15a)$$

$$\text{Central annual LRS} = 16.4 \times 10^{-2} \times (\Delta S_j) \times POP_{\geq 18j} \quad (4\text{-}15b)$$

$$\text{Low annual LRS} = 23.0 \times 10^{-2} \times (\Delta S_j) \times POP_{\geq 18j}. \quad (4\text{-}15c)$$

Aggregation Procedures for Acute Morbidity Health Effects

Several of the more broad categories of acute morbidity health effects, such as restricted activity days or days with lower respiratory symptoms, may include days on which effects measured in another function occur, such as days spent in the hospital. To avoid double counting, therefore, it is necessary to subtract some of these potentially overlapping categories. Some additional adjustment will be necessary when one function is for all ages and another is only for adults. In this case, we will assume the incidence of the effect is proportional to the age distribution which is that 83 percent of the U.S. population is 18 and older. The following subtractions are done before monetary valuations are applied and summed. As discussed in Chapter 5 on monetary valuation of human health effects, each RHA is assumed to average 6.8 days and each CHA averages 6.9 days. We assume that all days in the hospital and all asthma symptom days are also restricted activity days and therefore subtract these from total RADs. We also assume that all RADs are also acute respiratory symptom days and therefore subtract a fraction of RADs from LRSs. The Ostro et al. (1993) study reports that 28 percent of the acute respiratory symptoms are lower respiratory tract. We therefore assume that RADs are split between upper and lower respiratory tract in the same proportions. Net RADs and net LRSs are therefore defined as follows:

net RADs = total RADs - (0.83 × 6.8 × RHAs) - (0.83 × 6.9 × CHAs) - (0.83 × ASDs)

net LRSs = LRSs - (0.28 × total RADs).

These adjustments are approximate, but they do eliminate and even possibly over-compensate for overlap in the daily health endpoints. There may remain, however, some subtle overlap between the daily health endpoints and the chronic bronchitis and premature mortality health endpoints. For example, some of the hospital admissions may reflect health effects that are

accompanied by premature death. Because as is shown in Chapter 6, the total health benefits are dominated by the premature mortality and chronic bronchitis effects, the possible impact on the total health benefits of such overlaps is necessarily small.

4.4.5 Summary of Selected Concentration-Response Functions

Table 4-2 lists the selected concentration-response estimates for each of the health effects categories for sulfates. Omissions, biases, and uncertainties are summarized in Table 4-3.

Table 4-2
Selected Coefficients for Human Health Effects Associated with Sulfate Concentration Changes

Health Effect Category	Selected Concentration-Response (probability weights)
Annual mortality risk per 1 $\mu g/m^3$ change in annual average SO_4 concentration. Sources: See Table 4-1	L 8×10^{-6} (25%) L-C 24×10^{-6} (25%) H-C 56×10^{-6} (25%) H 112×10^{-6} (25%)
Chronic bronchitis (CB) annual risk per 1 $\mu g/m^3$ change in annual average SO_4 concentration. Source: Abbey et al. (1995)	For population 25 years and over: L 0.5×10^{-4} (25%) C 1.1×10^{-4} (50%) H 2.0×10^{-4} (25%)
Respiratory hospital admissions (RHA) annual risk factors per 1 $\mu g/m^3$ change in annual average SO_4 concentration. Source: Burnett et al. (1995)	L 1.3×10^{-5} (25%) C 1.6×10^{-5} (50%) H 1.8×10^{-5} (25%)
Cardiac hospital admissions (CHA) annual risk per 1 $\mu g/m^3$ change in annual average SO_4 concentration. Source: Burnett et al. (1995)	L 1.0×10^{-5} (25%) C 1.3×10^{-5} (50%) H 1.7×10^{-5} (25%)
Asthma symptom day (ASD) annual risk factors given a 1 $\mu g/m^3$ change in annual average SO_4 concentration. Source: Ostro et al. (1991)	For population with asthma (4.7% of population): L 3.3×10^{-1} (33%) C 6.7×10^{-1} (34%) H 9.9×10^{-1} (33%)
Restricted activity day (RAD) annual risk factors given a 1 $\mu g/m^3$ change in annual average SO_4 concentration. Sources: Ostro (1987), Ostro and Rothschild (1989)	For population aged 18 years and over: L 4.7×10^{-2} (33%) C 9.3×10^{-2} (34%) H 14.6×10^{-2} (33%)
Day with lower respiratory symptom (LRS) annual risk factors given a 1 $\mu g/m^3$ change in annual average SO_4 concentration. Source: Ostro et al. (1993)	For population aged 18 and over: L 6.6×10^{-2} (25%) C 16.4×10^{-2} (50%) H 23.0×10^{-2} (25%)

Table 4-3
Key Omissions, Biases, and Uncertainties

Omissions/Biases/Uncertainties	Direction of Potential Error	Comments
Concentration-response relationships	?	Statistical association in epidemiology studies does not prove causation. Measurement error and averting behavior could cause downward bias. Omitted confounding variables could cause upward bias.
Transfer of concentration-response relationships	?	Estimates are based on transfers across time and location. Possible unaccounted for differences add uncertainty.
Relationship between sulfates and other measures of particulate matter	+	Collinearity among particulate matter measures add uncertainty to the quantitative interpretation of sulfate based results. This uncertainty is addressed in the sensitivity analysis.
Zero threshold assumption	+	Evidence on possible thresholds is inconclusive. This uncertainty is addressed in the sensitivity analysis.
Age group assumptions	-	The effect of sulfates on mortality for different age groups was based on the results of nonsulfate studies. Effects on children probably understated due to limited studies that include children.
Presumed linearity of concentration-response	?	The effect of assuming a constant risk per unit of sulfate is difficult to assess with available information. Error could occur in either direction.
Assumed independence of baseline health incidence and sulfate concentrations	?	Used average incidence to transform % change/sulfate to the number of cases per change in sulfate concentration. There is no bias if they are independent.
Overall Impact	+	No clear directional bias is entirely dominant, but tendency may be toward upward bias. This is addressed in the sensitivity analyses.

CHAPTER 5
MONETARY VALUATION OF HEALTH EFFECTS CHANGES

5.1 INTRODUCTION

This chapter presents monetary value estimates for the adverse human health effects expected to be reduced because of the reduction in ambient sulfate aerosol concentrations attributable to Title IV. Monetary value estimates are presented for an average case of each type of health effect quantified in this assessment.[1] These monetary value estimates per case are multiplied by the estimated reduction in number of cases to obtain total monetary value estimates for each type of health effect. These are then summed to total monetary value estimates for all health effects benefits attributable to the sulfate aerosol reduction.

5.1.1 Monetary Valuation Concepts for Health Effects

The purpose of this assessment is to quantify the benefit to society of the reduction in health effects expected from the Title IV required SO_2 emissions reductions. Monetary values for changes in risks of human health effects should therefore reflect the full consequences to the affected individuals and to society.

Adverse health effects result in a number of economic and social consequences, including:

1. **Medical costs.** These include personal out-of-pocket expenses of the affected individual (or family), plus costs paid by insurance or medicare, for example.

2. **Work loss.** This includes lost personal income, plus lost productivity whether the individual is compensated for the time or not. For example, some individuals may perceive no income loss because they got sick pay, but sick pay is a cost of business and reflects lost productivity.

3. **Increased costs for chores and caregiving.** These include special caregiving and services that are not reflected in medical costs. These costs may occur because some health effects reduce the affected individual's ability to undertake some or all normal chores, and he or she may require caregiving.

[1] This chapter relies on previous literature reviews prepared for EPA including Violette and Chestnut (1983), Chestnut and Violette (1984), and Fisher et al. (1989).

4. **Other social and economic costs.** These include restrictions on or reduced enjoyment of desired leisure activities, discomfort or inconvenience (pain and suffering), anxiety about the future, and concern and inconvenience to family members and others.

Cost-of-illness (COI) measures include only medical costs plus work loss (Consequences 1 and 2 above), and thus do not reflect the total welfare impact of an adverse health effect. Therefore, using COI measures in a quantitative assessment results in a clear downward bias in the valuation of adverse health effects. COI measures, however, have the practical advantages of being easily understood and often readily available because they are based on available market and expenditure data.

A comprehensive monetary measure of value for changes in health risk is the dollar amount that would cause the affected individual to be indifferent to experiencing an increase in the risk of the health effect or losing income equal to that dollar amount. This monetary measure is the maximum willingness to pay (WTP) to reduce the risk of the health effect and all associated costs. WTP will thus reflect all the reasons an individual might want to avoid an adverse health effect, including financial and nonfinancial concerns.[2] WTP is a more comprehensive measure of value than COI, but it can be more difficult to estimate.

Sometimes in this discussion of monetary valuation for health effects we distinguish between health effects and health risks. A health effect refers to an illness or symptom, including death, that is experienced by someone. A health risk is the quantitative probability that any one individual might experience a given health effect. Changes in air quality cause changes in the number of health effects in the exposed population, but from the point of view of the individual what changes is the risk of experiencing a given health effect. This is because it is unknown exactly which individuals might be affected. WTP estimation techniques for more serious health effects such as premature mortality or chronic illness tend to focus on changes in the risks of such health effects that an individual might experience. For example, WTP studies for premature mortality do not estimate what individuals would be willing to pay to prevent a certain death, but rather estimate what they are willing to pay for small changes in risks of death.

5.1.2 WTP Estimation Techniques for Health Risks

WTP is typically measured by analyzing prices that are paid for goods and services. The maximum price that an individual is willing to pay for a good or service is a measure of how much they value that good or service. Prices cannot be directly observed for preventing health

[2] Financial costs of health effects are not always borne fully by the individual but are shared through health insurance and public health care subsidies. In some instances therefore empirical estimates of WTP to avoid or reduce health effects may not fully reflect these shared costs. For a comprehensive measure of WTP such shared costs should be added to individual WTP.

risks because prevention of health risks is not directly purchased in the market. However, there are instances when the monetary tradeoffs that people are willing to make between income and health risks can be observed or measured. There are two general economic approaches for measuring WTP for nonmarket goods such as health risk prevention. The first is to analyze actual situations in which WTP for health risks may be indirectly revealed; the second is to have subjects respond to a hypothetical situation designed to have them reveal their WTP.[3]

An example of the first approach is a wage-risk study in which wage premiums for risks of death on the job are estimated. This is done by analyzing all the factors that determine differences in actual wages between jobs, including on-the-job risks of death. The amount of additional wages that people are paid per unit of additional risk of fatal injury is a measure of the monetary value of that risk to the individual who voluntarily accepts that risk in exchange for a given wage increment. The primary advantage of this type of study is that it is based on actual behavior. The primary limitations are that it is difficult to find situations in which there is a clear tradeoff between money and risk, and to statistically isolate WTP for a risk increment from other factors involved in the specific behavior.

An example of the second approach is a contingent valuation study in which subjects are presented with a hypothetical situation that involves a tradeoff between income or expenditures and a specific health risk or health effect. The subjects are then asked to estimate what they would be willing to pay to change that risk by a specific amount. It is important that the hypothetical situation presented to study subjects be realistic and easy to understand. The primary concern with this type of study is whether subjects are able to give accurate responses to hypothetical questions.[4]

5.2 ISSUES IN APPLYING WTP ESTIMATES FOR THIS ASSESSMENT

Although WTP for changes in health risks is the conceptually correct monetary value measure for this assessment, there are some limitations in available estimates. These limitations result from uncertainties in the available estimates, inexact matches between the health risks for which WTP estimates are available and the health risks of interest in this assessment, and the lack of available WTP estimates for some of the health risks of interest.

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[3] This section provides a brief introduction to these estimation techniques. For more information see Freeman (1993).

[4] Contingent valuation is a somewhat more controversial technique than some other economic valuation techniques. There are only a few instances when we are relying entirely on estimates from contingent valuation studies for monetary valuation estimates for specific health effects in this report. Snell et al. (1993) review the contingent valuation studies used in this report in light of recent recommended contingent valuation guidelines.

WTP estimates are available for risks of death, but there are some differences between the types of fatal risks for which WTP estimates are available and those of interest in this assessment. WTP estimates are also available for some but not all types of morbidity of concern in this assessment.

5.2.1 Issues in Applying Available WTP Estimates for Premature Mortality

There are several uncertainties in applying the available WTP estimates for valuing changes in premature mortality risks. The justification for using the available WTP estimates is that they provide estimates of what people are willing to pay to reduce their risks of premature mortality by small amounts. The risks involved in this analysis are also small, but there are some differences with regard to who is at risk and what the risk is. First, there is quantitative evidence from the health effects literature that a large share of the individuals at risk are elderly (65 years old or older). Two additional aspects of potential significance are the potential health status of the people at greatest risk and differences regarding the expected cause of death. There is very little available empirical evidence about how these factors might affect the value of reducing risks of premature mortality. There is, therefore, some unresolved uncertainty in applying available WTP estimates in this analysis.

Age

Available evidence of the effect of age on WTP for changes in mortality risks is discussed in Section 5.3. The empirical evidence is quite limited, but it provides a basis for some adjustment in average WTP estimates for elderly individuals for changes in mortality risks. Most available WTP estimates for changes in mortality risks, however, are from studies in which the elderly are not well-represented. The adjustment selected for the elderly in this analysis, therefore, must be acknowledged as relatively uncertain. The adjustment for age explained in Section 5.3 is based on an analysis of the available empirical evidence first presented by Rowe et al. (in press). Other approaches for addressing the age question could be justified, including making no adjustment; however, we selected the approach proposed by Rowe et al. as a reasonable interpretation of limited empirical evidence.

Health Status

The available WTP estimates for changes in mortality risks are based on results from study samples of individuals with average levels of health. Although it cannot be determined from available epidemiologic studies, it is possible that those individuals at greatest risk of premature mortality due to exposure to air pollutants are those who are already in poor health for reasons unrelated to air pollution exposure. Some instances may involve chronic illnesses, because of which the individual may already have a reduced life expectancy even in the absence of pollution exposure. For example, Schwartz and Dockery (1992a) found increased mortality rates due to chronic respiratory disease, pneumonia, and cardiovascular disease

associated with higher levels of particulate matter. Some of these individuals apparently suffer from preexisting chronic disease. There is not sufficient evidence available to say how having a chronic illness might affect WTP for changes in mortality risks, but it is possible that the reduced life expectancy and irreversibly reduced quality of life associated with many chronic illnesses may result in lower WTP to reduce mortality risks.

Cause of Death

It is possible that people are more concerned about avoiding some kinds of death than others. For example, Jones-Lee et al. (1985) results suggest that some people are more afraid of death from cancer than of death from automobile accidents. This may be related to the perceived pain, suffering, and expense associated with the illness that precedes death in the case of cancer. Some studies also suggest that people find involuntary risks, such as pollution exposure, less acceptable than voluntary risks, such as traffic accidents (Violette and Chestnut, 1983). Studies have not been able to separate these different aspects of the different risks of death in terms of the potential effect on WTP. The most reliable WTP studies to date have focused on accidental deaths, primarily on the job and in vehicle accidents. The types of death of interest for this analysis are related to various illnesses, both chronic and acute. Based on the limited evidence available about how people respond to different types of risks, it is likely that if there is any error in applying available WTP estimates in this analysis it will be to understate the WTP to avoid the types of risks of interest in this analysis.

For this analysis, available WTP estimates for changes in risks of death are applied to all estimated mortality risks regardless of the cause of death. Although arguments could be made for small adjustments in some cases, any such adjustment is overshadowed by the level of uncertainty in using these estimates, which cannot be reduced at this time. For example, WTP estimates based on accidental death probably do not reflect the medical costs typically associated with treatment of the chronic or acute illness that may precede premature death due to air pollutant exposure. However, COI estimates suggest that average lifetime medical costs per chronic respiratory disease patient are under $100,000 (Krupnick and Cropper, 1989). This omission is not very significant relative to a selected range of WTP estimates of $2 million to $7 million per fatality.

5.2.2 WTP to COI Ratios

WTP estimates are not available for some of the nonfatal health effects considered in this analysis. In these cases, COI estimates are used and are adjusted upward by a factor of 2 to compensate for the expected ratio of WTP to COI estimates for any given health effect. This adjustment is based on limited available evidence on WTP/COI ratios, but we believe the resulting adjusted health valuation estimates are less biased than would occur if only unadjusted COI estimates were used. This section develops a general WTP/COI ratio to escalate COI values to approximate WTP values. Because this ratio is likely to be specific to

each health effect, any such ratio based on existing studies must be seen as an approximation to improve valuation and reduce known bias that would occur if unadjusted COI estimates were used to value health effects.

This summary of the empirical evidence and the selected ratio for adjusting the COI estimates is taken from Rowe et al. (in press). The empirical literature on this question is limited and other interpretations could be justified, including making no adjustment at this time. Our judgment was that an uncertain adjustment was preferable to no adjustment, because no adjustment results in a clear downward bias in the estimates.

Three studies provide evidence on WTP/COI ratios for the same study population addressing the same change in the same health effect. In each study, the participants were individuals diagnosed with the health effect. These studies addressed changes in incidence of asthma symptoms (Rowe et al., 1984; Rowe and Chestnut, 1986), increased angina symptoms (Chestnut et al., 1988), and risks of cataracts (Rowe and Neithercut, 1987). In each study, participants rated the importance of each of the components of WTP (listed in Section 5.1.1), and provided WTP estimates for reducing or preventing these health effects. The participants rated some non-COI consequences as more important to avoid than the COI consequences. This again suggests that WTP significantly exceeds COI.

The dollar ratio results listed in Table 5-1 are based on estimated individual and social COI in dollars, and on individual WTP in dollars. Individual COI is less than social COI because society incurs some costs the individual does not (because of insurance coverage, sick pay, and other types of compensation). Because social COI exceeds individual COI, the WTP/COI ratio for individuals exceeds the ratio for society. Also available from the asthma and cataract studies are respondent ratings of their COI as a share of their perceived total damages. From these ratings, the individual and society WTP/COI ratios are computed and reported in Table 5-1.

Across the three studies, the total social WTP/COI ratios range from 1.3 to 2.4. The COI in these studies range from a few dollars to $7,000 per episode of cataracts. Based on these results, we select a WTP/COI ratio of 2.0 for this analysis. Thus, we multiply available COI estimates by 2.0 to approximate WTP, when actual WTP estimates are not available for a given health effect.

Basing a WTP/COI adjustment on these study results is admittedly uncertain. The study samples are small and the range of health effects is limited. However, we still judge that it is preferable to make some adjustment than to make no adjustment. Making no adjustment in COI estimates for valuation purposes results in a clear downward bias. We have selected a fairly conservative adjustment factor, based on available evidence, to minimize the chance of overadjusting. Additional evidence that these adjustment factors are conservative exists in the WTP estimates for risks of death. Average COI estimates for fatalities are typically in the

Table 5-1
WTP/COI Ratios

Health Effect		WTP/COI Affected Individual	WTP/COI Society
Asthma Symptoms	Dollar ratio	1.6 to 2.3	1.3 to 1.7
Cataracts	Dollar ratio	4.25	2.4
	Respondent rated share of total damages ratio	5.3	2.1
Angina Symptoms	Respondent rated share of total damages ratio	2.5-4	NA

Sources: Asthma: Rowe et al. (1984), Rowe and Chestnut (1986).
Cataracts: Rowe and Neithercut (1987).
Angina: Chestnut et al. (1988).

middle hundreds of thousands. WTP estimates per fatality are in the millions, a difference of an order of magnitude.

5.3 MONETARY VALUATION ESTIMATES FOR PREMATURE MORTALITY RISKS

Several economic studies have estimated average WTP in the United States for small changes in risks of accidental death. These estimates have been widely used in benefit analysis of public policy options that would result in changes in risks of death for the public (Viscusi, 1992). They are sometimes referred to as "value of life" estimates because they are expressed on a per life basis. But it is important to note that they are based on WTP of the individual for reducing his or her risk of premature death by a small amount, not on the total value of a human life under all circumstances.

The estimates provided by these studies are average dollar amounts that individuals are willing to pay for small reductions in risks of death. For example, one study might find an average WTP of $300 for an annual reduction in risk of death of 1 in 10,000. These estimates are extrapolated to a per life basis by summing individuals' WTP over enough people that a value per life saved is obtained. In this example, this value would be $3 million per life, the result of $300 multiplied by 10,000 people. The term used for this estimate in much of the economics literature is "value of a statistical life" (VSL) to denote that it is a summation of WTP for small changes in risks of premature death.

Available estimates of WTP to prevent small changes in risks of death are based on situations where individuals are observed making tradeoffs between probabilities of death and some benefit, such as income. Most of these studies have estimated wage premiums associated with different levels of on-the-job risks. Additionally, some contingent valuation studies have been conducted in which subjects have been asked what they would be willing to pay to reduce, for example, their risks of fatal accidents at work or in traffic accidents. A few averting behavior studies have also been conducted that estimate costs associated with observed behaviors that reduce risks, such as smoke detector usage in the home or seat belt usage in automobiles.

For the most part, available WTP estimates are for risks of accidental death in circumstances where individuals are voluntarily exposed to risks (e.g., choosing a job or driving in a car). The estimates are also drawn largely from studies of working-age adults. Some potentially important differences exist between the contexts of these available estimates and the environmental health risks being evaluated in the externality model. Environmental health risks are related to illness rather than accidents and may in some cases fall disproportionately on the elderly and those with already compromised health. The potential implications of these differences were discussed in Section 5.2.1. The potential effect of age on WTP is discussed in more detail in Section 5.3.2.

5.3.1 Summary of Available WTP Estimates

Four recent reviews of this literature evaluated and summarized available WTP estimates for small changes in risks of death for potential use in analyses of public policy decisions (Fisher et al., 1989; Miller, 1989; Cropper and Freeman, 1991; Viscusi, 1992). Each review concludes with a list or range of "best" estimates that the authors judged as most appropriate for use in evaluating public policy decisions that result in small changes in risks of death for the public. All of these reviews covered basically the same body of literature, but the most recent review (Viscusi, 1992) included a few additional studies that were not completed when the earlier reviews were done. These reviews are consistent in many of their conclusions regarding which of the available estimates are most appropriate for use in policy analysis, but there are also differences. We take into consideration the conclusions, and their basis, of each of these four reviews in selecting a central, low, and high estimate of WTP for changes in risks of death for use in this analysis. The selected estimates for this analysis are discussed in Section 5.3.3. Ranges of VSL recommended by the authors of each of the four reviews as best for policy analysis are listed in Table 5-2.

Fisher et al. (1989) list 21 studies that each give a VSL estimate. The authors reject three studies listed as "early low-range wage-risk estimates," primarily because of problems in the risk data used. The authors also reject the "consumer market studies," which fall into the category of averting behavior studies, because they argue that each of the estimates is clearly downward biased because of study design problems or data limitations. They also reject one of the "new wage-risk studies" that examined wages for police officers in the United States,

Table 5-2
Recommended Ranges of VSL Estimates

Review	VSL Rounded to Millions (1994 dollars)	
	Low	High
Fisher et al. (1989)	$2	$11
Cropper and Freeman (1991)	$2	$7
Viscusi (1992)	$3	$8
Miller (1989)	$1	$4

because of the limited scope of the study sample and potential problems with the on-the-job death rate data used. This leaves 13 VSL estimates judged by these authors as most appropriate for use in policy analysis. These estimates range from $2 million to $11 million (1994 dollars), and have an arithmetic mean of about $6 million. All but two of the 13 studies are wage-risk studies. The remaining two studies are contingent valuation studies, which obtained results of $4.1 million and $3.8 million. These results fall in the lower half of the overall range. Fisher et al. caution that all the estimates above $8 million are based on wage-risk studies using Bureau of Labor Statistics data for on-the-job risks. These data are limited in that they give risk information by industry, but not by occupation. There is no specific reason why these data would cause any upward bias in VSL results, but results that are not verified by similar conclusions using different data sources are somewhat less robust. The authors therefore conclude that the $2 million to $8 million range is the strongest because it has been verified by different studies using varying data sources, but they do not rule out the possibility that the higher estimates might be correct.

Cropper and Freeman (1991) present an adapted version of Table 1 from Fisher et al. They deleted four of the 21 studies. The authors do not explain these exclusions, but presumably they found them to be less appropriate for policy analysis than the remaining 17. Two of the deleted studies were in categories that were rejected by both sets of reviewers, so their exclusion causes no change in the conclusions. The primary difference in the conclusions of these two reviews is that Cropper and Freeman make a stronger statement that using the Bureau of Labor Statistics on-the-job risk data apparently causes upward bias in the VSL estimates, based on comparisons of results using different types of data. Excluding the estimates based on Bureau of Labor Statistics data leaves six VSL estimates judged as "best" for use in policy analysis. These are from four wage-risk studies and two contingent valuation studies. The wage-risk estimates selected by Cropper and Freeman range from $2.1 million to $7.3 million (1994 dollars), and the contingent valuation estimates selected range from

$3.5 million to $4.1 million. The arithmetic mean of all six selected VSL estimates is $4.1 million.

Viscusi (1992) provides separate discussions and summaries of averting behavior, wage-risk, and contingent valuation studies. His overall conclusion is that the most appropriate range of VSL estimates for use in policy analysis is $3 million to $8 million in 1994 dollars. He also rejects the available averting behavior study results for use in policy analysis because of clear downward biases in the study designs and data. Viscusi lists 27 VSL estimates from 22 wage-risk studies and eight estimates from six contingent valuation studies. Similar to the conclusions of the previous reviewers, Viscusi raises questions about some of the earlier wage-risk studies that used inappropriate risk data and obtained relatively low VSL results. He also raises some questions about some of the wage-risk studies that obtained results above $8 million. Viscusi concludes that the best VSL results from wage-risk studies are between $3 million and $8 million. Viscusi suggests that the two earliest contingent valuation studies were exploratory and that less weight be given to these two estimates (one is very low, the other is very high). The arithmetic mean of the remaining four contingent valuation estimates is either $3.1 million or $5.1 million, depending on whether the median or the mean estimate is selected from one of the studies. The range of the contingent valuation estimates is $1.4 million to $4.3 million or $11.0 million, depending on whether the median or the mean value is selected from one of the studies.

Miller (1989) uses a different approach than that used in the other three reviews and reaches some different conclusions. He selects a larger number of available VSL estimates as potentially appropriate for use in policy analysis, but makes several adjustments in the estimates to reconcile differences in study design or limitations in data. Miller includes 29 VSL estimates as of "reasonably good quality." Included in these 29 estimates are most of the estimates selected in the other reviews as most appropriate for policy analysis. An important difference is that Miller includes results from eight averting behavior studies, which are rejected by the other reviewers as likely to be biased downward. An additional four are from contingent valuation studies, and the remaining 17 are wage-risk estimates. Miller made several adjustments to the estimates, most of which resulted in lowering the estimates, especially for some of the wage-risk studies with the highest results. The adjustments Miller made included (1) converting the wage-risk results to after-tax dollars, (2) adjusting for differences in labor risk data sources, (3) adjusting for failure to include nonfatal injury risks in the analysis, (4) adjusting to a uniform value of time or discount rate if used, and (5) adjusting for differences in perceived versus actual risks. The conceptual arguments for some of these adjustments may be valid, but the reliability of the data used to determine the exact adjustment to make is in many cases questionable. Miller concludes by choosing a mean VSL estimate of $2.7 million (1994 dollars), and a range of $1.4 million to $4.3 million.

5.3.2 The Potential Effect of Age on WTP for Changes in Mortality Risks

Although it has been suspected that age may be a factor in risk of death due to air pollution exposure, until recently there has been little quantitative evidence in the available epidemiologic literature. Schwartz and Dockery (1992a) report evidence that the measured association between daily mortality rates and daily levels of ambient particulate matter is greater for people over the age of 65. They provide sufficient information to estimate the change in the number of deaths expected for people over 65 and under 65 for a given change in ambient particulate matter. It is therefore important to consider whether average WTP for changes in mortality risks might be different for people over 65.

This raises the question of whether WTP for changes in risks of death in the current time period is different for people over 65 than for the average adult. There is limited empirical evidence regarding this question, but some information is available. The expectation is that WTP will be lower for a 65-year-old than for the average adult, because expected remaining years of life are fewer. This expectation is based on the presumption that WTP for one's own safety is derived from the utility one receives from one's own life and that this utility is to some extent a function of the amount of time one expects to remain alive.

Some analysts have suggested that effects of age might be introduced by dividing average WTP per statistical life by average expected years of life remaining (either discounted or not) to obtain WTP per year of life (Miller, 1989; Harrison and Nichols, 1990). Such a calculation implies very strong assumptions about the relationship between life expectancy and the utility a person derives from life, namely, that utility is a linear function of life expectancy. Although this might be correct, it is also plausible that this calculation will result in significant understatement of WTP for the elderly. An understatement could result for a number of reasons. One is that there may be a value to being alive that is independent of the amount of time one expects to live. Another is that as one ages, the remaining time may be more highly valued than it was in midlife.

We have identified one study that provides unconstrained empirical evidence concerning how WTP for small changes in risks of death varies with age. Jones-Lee et al. (1985) conducted a contingent valuation study concerning motor vehicle accidents and report an estimated WTP function for characteristics of the respondents, including age.[5] (There are some other studies that provide some suggestive evidence regarding how WTP for reducing risks may change with age, but each of these studies imposes some constraints on the conclusions in the form of unverified model assumptions.)

[5] This summary and proposed adjustment in the monetary values based on available empirical evidence of the effect of age on WTP for changes in mortality risks is drawn from Rowe et al. (in press).

Jones-Lee et al. conducted a general population survey in the United Kingdom in which about 1,000 respondents were asked how much additional money they would be willing to pay for transportation with a bus company with a better safety record. All relevant risk information was quantitatively specified and the survey appears to have been well designed and executed. Implied WTP per life (VSL) was calculated for each response. For example, the VSL is $6 million when the WTP response is $240 for a reduction in risk of death of 4 in 100,000. Variations in the implied VSL estimates across respondents were then examined as a function of age and other characteristics of the respondents. An appropriate functional form was used that allowed WTP to be a nonlinear function of age (age + age^2).

The results show a statistically significant relationship estimated between age and VSL, which was statistically strongest for the responses to the first bus safety questions. The results indicate gradually increasing VSL until around age 45, then gradually declining VSL. The results for both the bus safety questions imply that VSL for a person aged 65, all other things being equal, is about 90 percent of VSL for a person aged 40.

The Jones-Lee et al. results with respect to age, based on the responses to the first bus safety question, are:

$$\text{VSL} = \text{Constant} + 12{,}489 \times (\text{Age} - \text{Mean Age}) - 660 \times (\text{Age} - \text{Mean Age})^2 + zB_iX_i, \quad (5\text{-}1)$$

where:

VSL = the implicit VSL given by the respondent
B_iX_i = the other independent variables in the WTP regression.

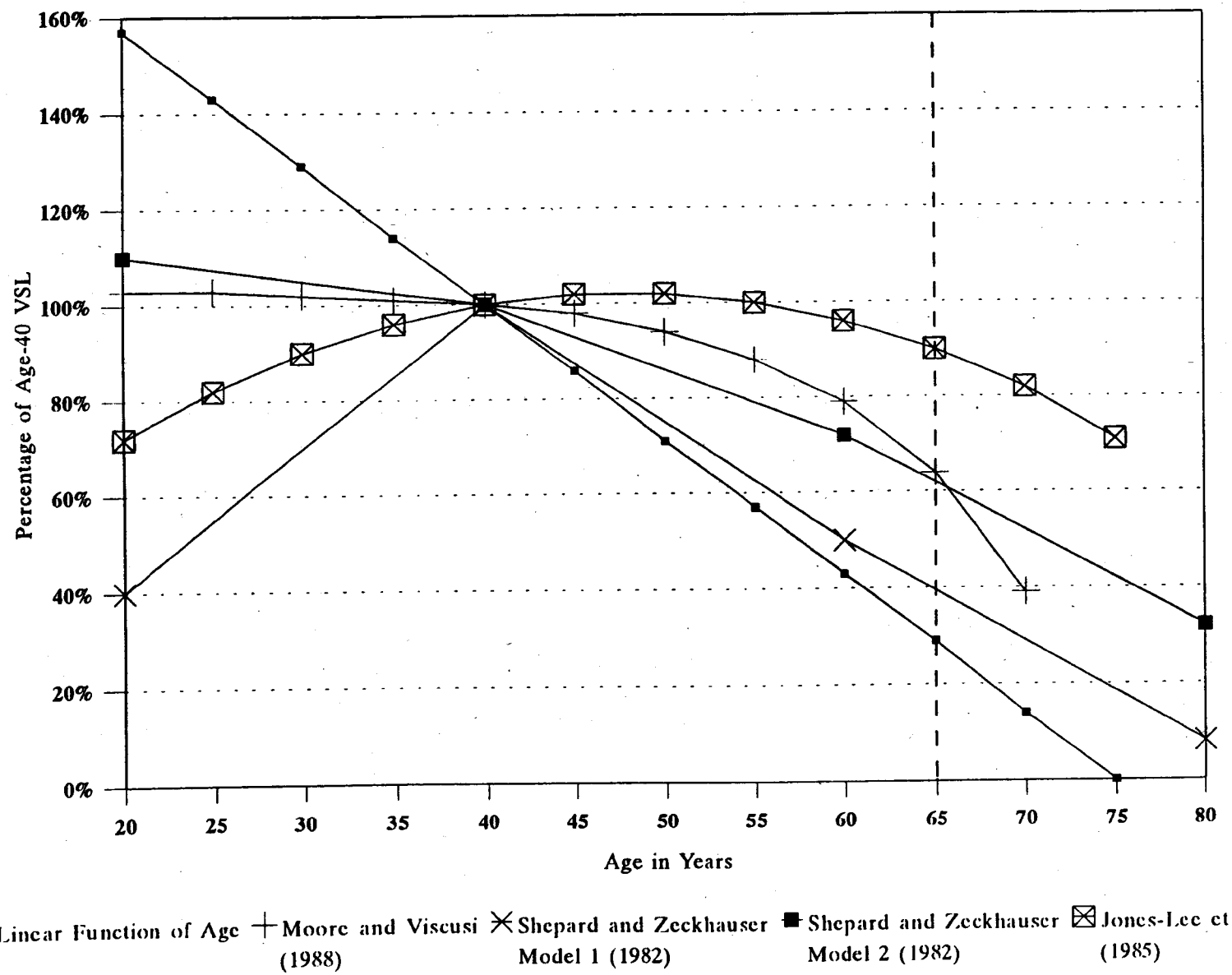
The authors do not report mean age for the sample, but describe the sample as nationally representative. For purposes of interpreting the regression results, we use 40 years as an average age, which is close to the average age of adults in the United States. The average VSL is reported as 1.6 million British pounds. We then calculated illustrative VSL estimates at selected ages using the following formula:

$$\text{VSL} = 1{,}600{,}000 + 12{,}489 \times (\text{Age} - 40) - 660 \times (\text{Age} - 40)^2. \quad (5\text{-}2)$$

This calculation assumes that other factors that influence VSL do not change with age. The risk of error due to this assumption seems small because only the age variables were statistically significant in this regression.

To allow for simple comparison to the results of other studies, we calculated VSL at each age using Equation 5-2. We then calculated VSL at each age as a percentage of VSL at age 40. These percentages are plotted in Figure 5-1.

Figure 5-1
Value of a Statistical Life as a Function of Age



Moore and Viscusi (1988) estimated a wage-risk premium for a sample of workers in the United States. They defined risk on the job as the probability of a fatal accident multiplied by the discounted remaining life years of the individual. They used a nonlinear estimation technique to estimate both the risk coefficient and the implicit discount rate for time. They also included an expected annual annuity variable to account for the possibility that a wage-risk premium might not be as high if available insurance covers some of the risk to dependents. The results showed a significant relationship between wages and risks of fatal accidents and implied a value per statistical life of about $6.5 million (1986 dollars). The finding of a significant (negative) relationship between wages and expected annual annuity suggests that estimates that ignore potential death benefits may understate WTP to reduce risks of death. The estimated discount rate was 10 percent to 12 percent.

The Moore and Viscusi model assumes a constant value per year of life, and future years are discounted at rate r. The model, therefore, does not provide an unconstrained test of how VSL varies with age. VSL at different ages is simply a function of the discount rate, according to this model, and is therefore proportional to discounted remaining life years. The model implies that WTP for small changes in current risks decreases with age throughout a person's lifetime. How fast it declines depends on the discount rate. Moore and Viscusi define discounted remaining life years as:

$$DRLY = 1/r \times [1 - \exp(-r \times R)] \qquad (5\text{-}3)$$

where:

DRLY	=	discounted remaining life years
R	=	expected life years remaining.

The implications of different discount rates on WTP for changes in risks of death can be illustrated as follows. VSL will be proportional to the discounted remaining life years (DRLY). This means that the ratio of VSL at age 40 to VSL at age 65 will be the same as the ratio of DRLY at age 40 and DRLY at age 65. The implications of Moore and Viscusi's results from their linear wage function (r = 9.6 percent) with respect to the age of the worker are shown in Figure 5-1. It should be noted that the estimates are based on a sample of 317 working adults, which included few individuals over age 60 (62 is two standard deviations above the mean age). Also, life expectancies do not actually decline linearly with age, as is assumed in the calculations that underlie Figure 5-1. Average life expectancy at birth in the United States was 75 years in 1983, but was 17 years for 65-year-olds.

Cropper and Freeman (1991) provide a summary of the life-cycle consumption-saving model that can be used to derive a theoretical definition of WTP for changes in the probability of death. This model is based on the premise that utility is a function of consumption. The authors note that if there is additional utility derived from survival per se, then the life-cycle model provides a lower bound estimate of WTP. Of interest is what the model predicts in

terms of how WTP for changes in risks of death in the current time period changes as a function of age. For a quantitative example, this depends on assumptions regarding a lifetime pattern of earnings, endowed wealth, the rate of individual time preference, and other parameters of the model. These will all vary for different individuals, and uncertainty exists empirically about population averages for many of these factors. However, using reasonable values to calibrate the model is illustrative.

Cropper and Freeman (1991) note that if consumption is constrained by income early in life, the model predicts that VSL increases with age until age 40 to 45, and declines thereafter. Shepard and Zeckhauser (1982) illustrate this point with numerical examples for the life-cycle model. When they estimate the model with reasonably realistic parameters and assume no ability to borrow against future earnings or to purchase insurance, they find a distinct hump in the VSL function that has a peak at about 40 years and drops to about 50 percent of the peak by 60 years. When they allow more ability to borrow against future earnings and to purchase insurance, the function flattens and at 60 years drops only to 72 percent of the VSL at age 40.

For comparison purposes, all of the estimates discussed above are plotted in Figure 5-1 along with the relationship between VSL and age implied by a simple linear decline with age. This linear decline implies that VSL at age 65 is about 30 percent of VSL at age 40. This is a much larger decline in VSL as a function of age than implied by the available empirical results reported above. The strongest weight should be given to the Jones-Lee et al. results because they are based on a representative general population survey and were not unduly constrained by an imposed functional form. However, survey results can be highly variable and need to be interpreted cautiously until verifying results from multiple studies are obtained.

The life-cycle model results are quite variable depending on assumptions used to quantify the model. These assumptions have not been verified empirically. Because the model defines utility as a function of consumption and consumption is a function of time, it is expected that if the life-cycle estimates err it is on the side of overstating the effect of age on VSL (in other words, reducing VSL too much at age 65 relative to age 40). The error would result if there is some value to just being alive independent of consumption. At consumption levels above subsistence, this is quite plausible. Therefore, these estimates should be interpreted as representing the maximum plausible reductions in VSL as a function of age.

5.3.3 Monetary Estimates Selected for this Analysis

Obviously, there is some judgment involved in selecting central, high, and low values for the WTP for changes in risks of death. The selected mortality valuation estimates for each age group are summarized in Table 5-3. We selected $4.5 million as the central estimate, $2.5 million as the low, and $9.0 million as the high for those under 65. The central estimate of $4.5 million is consistent with the mean ($4.1 million) of the six estimates indicated by Cropper and Freeman as most appropriate for policy analysis uses. It is within the range of

Table 5-3
Summary of Selected Monetary Values for Mortality Effects

Population Group	VSL Estimate (1994 dollars)		
	Low	Central	High
>65 years	$1.9 million	$3.4 million	$6.8 million
<65 years	$2.5 million	$4.5 million	$9.0 million
Age Weighted Average	$2.0 million	$3.5 million	$7.1 million
Selected Probability Weights	33%	50%	17%

results from both wage-risk and contingent valuation estimates, and is consistent with giving less weight to the wage-risk studies that have relied on Bureau of Labor Statistics risk data. When these are included, the mean estimate from the Fisher et al. review is $6.3 million. In selecting the central estimate we have given less weight to the Miller review because of the uncertainties involved in many of the adjustments he made in the estimates. Both the study selection and the adjustments made by Miller suggest that his conclusions are on the low side in terms of an appropriate VSL estimate for policy analysis. The central estimate of $4.5 million is close to the upper end of the range selected by Miller as appropriate for policy analysis. The low estimate selected for those under 65 is just below Miller's mean VSL estimate of $2.7 million. It is the lower end of the range selected by Fisher et al. and Cropper and Freeman. The selected high estimate falls within the upper estimates of $11 million and $7 million from the first three reviews summarized above. The VSL estimates discussed in Section 5.3.1 are based primarily on samples of working age adults. A few of the contingent valuation studies included individuals of retirement age, but this age is not well represented in the mean VSL values. We therefore apply the selected VSL estimates from these studies to the under 65 years old population.

Available evidence suggests that WTP for small changes in risks of death for people over age 65 can be expected to be lower than WTP for the same change in risk at age 40. However, there is considerable uncertainty about how much lower. The most relevant direct evidence suggests that the decline in VSL with age may be relatively small (e.g., 90 percent of the age 40 WTP at age 65). The evidence strongly suggests that a linear decline in VSL with age significantly understates actual VSL over age 65. Based on our evaluation of the above described evidence regarding VSL and age, we utilize the Jones-Lee et al. results to calculate a weighted average VSL based on the approximate age distribution for the U.S. population age 65 and older. This produces an adjustment to VSL for those 65 years old and older of about 75 percent of the average VSL for adults under age 65. Taking 75 percent of the

estimates per statistical life selected above for adults under 65, we get a central estimate of $3.4 million for those over 65, a low of $1.9 million, and a high of $6.8 million.

A age-weighted average VSL for this analysis is then calculated on the assumption that 85 percent of the sulfate-related deaths are people aged 65 and over (See Chapter 4). The results are shown in Table 5-3. These are the VSL estimates applied to the predicted changes in premature deaths associated with Title IV in this assessment.

The selection of probability weights for the low, central, and high estimates is somewhat arbitrary because there are several uncertainties in using these estimates in this analysis for which no quantitative information is available. The selected weights therefore reflect the uncertainty in the underlying WTP estimates for small changes in risks of accidental death for working-age adults, but do not fully reflect the uncertainty in applying these estimates in this analysis. The weight selected for the central estimate is 50 percent, because the underlying WTP estimates are predominately in the $3 to $6 million range. A weight of 33 percent is given to the low estimate and a weight of 17 percent to the high. This reflects that the high estimate is represented by fewer studies and a somewhat skewed distribution in the available WTP estimates. These weights result in a weighted mean value that approximates the selected central estimate.

5.4 MONETARY VALUATION ESTIMATES FOR MORBIDITY

WTP estimates of value are available for about half of the nonfatal health effects identified in Chapter 4, primarily the least serious health effects. However, most of the WTP studies completed to date have limitations because of small sample sizes and limited variation in the health effect studied, and few of these studies have been replicated. Some interpretations and adjustments in the results of the WTP studies have been necessary in applying them for this analysis. These studies have been reviewed and synthesized in previous air quality benefits studies (Rowe et al., in press; Krupnick and Kopp, 1988; Hall et al., 1989; Thayer, 1991; Unsworth and Neumann, 1993). We rely to a large extent on these previous reviews for specific interpretations.

When WTP estimates are not available at all, the monetary estimates are based on COI information, and the COI values are inflated to WTP estimates, as discussed in Section 5.2. The COI information used in this analysis reflects medical costs and lost productivity due to illness. The average daily wage is used as a measure of lost productivity for days when all normal activities are prevented because of illness. Such days include days spent in the hospital, one day for each emergency room visit, and days spent in bed because of illness. The average wage rate is used as a measure of the average opportunity cost of time for employed and not-employed individuals, on the presumption that those who are not employed value their leisure or household services at a level equal to the wage they forego in choosing not to pursue paid employment. This approach may somewhat overstate foregone wages for

the elderly and women, who make up a large share of the not-employed group and may have less than average earning power in the labor market. On the other hand, this approach does not reflect any productivity losses beyond the average work-day hours, thereby understating productivity losses for employed and not-employed individuals who perform household, childcare, and community service work beyond the usual work-day hours. This omission, however, is offset by the adjustment used to proxy WTP when using the COI estimates. For these calculations, we use the 1994 median daily wage for full-time salaried workers in the United States, which is about $93 (U.S. Dept. of Labor, 1995).

The available WTP studies provide some information on the range as well as the mean WTP values. In general, these ranges are minus 50 percent to plus 50 to 100 percent. A range of plus or minus 50 percent is therefore applied to the central estimates of WTP based on COI data in this analysis to derive the low and high estimates. High and low values are selected from the range of WTP results available when WTP studies have been conducted for those health endpoints. The low, central, and high WTP estimates for all morbidity effects are given equal probability weights. This reflects the limited number of empirical studies providing the WTP estimates and the fairly extensive assumptions and approximations used in deriving all of the estimates.

5.4.1 Adult Chronic Bronchitis

Viscusi et al. (1991) and Krupnick and Cropper (1992) conducted a set of survey exercises to estimate WTP for reducing risks of developing chronic respiratory disease. In both studies, respondents were presented with trade-off options for risks of developing chronic bronchitis (or chronic respiratory disease in general) versus cost of living. Respondents were presented with hypothetical residence location options where in some locations risks of developing chronic respiratory disease are lower but cost of living is higher. An additional trade-off question was for risks of developing chronic bronchitis versus risks of death in an auto accident. An interactive computer program was used to adjust the trade-off until the respondent reached a point of indifference between the two options. At this point, a maximum WTP to prevent developing chronic bronchitis is revealed.

The health endpoint defined in these studies does not exactly match that defined in the Abbey et al. (1995) study, upon which the estimates of new cases of chronic bronchitis are based (see Chapter 4). The primary difference is the level of severity. The WTP studies defined a severe case of chronic bronchitis. The Abbey et al. results reflect a more average case. In this section we present the results of these WTP studies and a procedure for adjusting the results to better reflect the level of severity of interest for this analysis.[6]

[6] This adjustment procedure is based on information reported by Krupnick and Cropper (1992) and was suggested by Alan Krupnick in personal communication.

The samples for the two studies differ. Viscusi et al. selected a representative sample of about 390 respondents. Krupnick and Cropper selected a sample of individuals who had a relative with a chronic respiratory disease. The Krupnick and Cropper sample was smaller (about 190 respondents) and less representative of the general population (lower average age and higher average income), reflecting a large percentage of respondents taken from the University of Maryland staff and students. The intent of the Krupnick and Cropper study was to test for the effect of familiarity with the disease on WTP responses.

Both studies used a definition of chronic bronchitis that reflects a severe case. The description of the disease included persistent symptoms of cough and phlegm, limits in physical activities, and ongoing medical care. Krupnick and Cropper used this definition in one version, and asked respondents to consider the risk of developing "a case of chronic respiratory disease like your relative's" in a second version. The relatives had chronic bronchitis, asthma, or emphysema. Respondents provided information on the severity of the relative's disease based on the number of symptoms present. This ranged from 0 to 13, where 13 reflects the severe chronic bronchitis case defined in the earlier questions. The analysis of WTP responses included the effect of the severity of the relative's case on the WTP response. At the mean of the variables, the estimated elasticity of WTP with respect to severity was 1.16. This means that WTP increased by 1.16 percent for every 1 percent increase in the 0 to 13 symptoms scale.

The WTP results from Viscusi et al. are more appropriate for this assessment because they are from a study sample that is more representative of the general population. The responses reflect the maximum amount the respondents revealed they would be willing to pay to reduce their annual risk of developing chronic bronchitis by a specified amount. The authors then calculated the implicit WTP per statistical case avoided. The median response for the cost of living trade-off was approximately $570,000, and the arithmetic mean was about $1,100,000 in 1994 dollars. The authors caution that the mean is affected by a small number of fairly high estimates and recommend that the median is more representative of the sample. We cautiously accept this recommendation until the accuracy of the high estimates can be further verified in repeated studies and analyses. For a low estimate for a severe case of chronic bronchitis we select the 20th percentile value of $340,000 and for a high estimate we select the 80th percentile value of $900,000.

We use an elasticity estimate for numbers of symptoms to scale the estimates for a severe chronic bronchitis case to better reflect WTP to avoid a more typical case. The elasticity estimate is calculated from results reported by Krupnick and Cropper for a combined analysis of chronic bronchitis, asthma, and emphysema. Using this estimate for chronic bronchitis assumes that the elasticity of WTP with respect to severity is similar for chronic bronchitis to that for all three diseases combined. The mean severity rating reported for the Krupnick and Cropper sample is 6.5, based on the 0 to 13 scale. Using the elasticity at the mean of 1.16, this suggests that WTP for an average case is 58 percent lower than for a case at 13 on the

scale. Using this to adjust the Viscusi et al. estimates, we get a central WTP estimate of $240,000, a low of $140,000, and a high of $380,000 for an average chronic bronchitis case.

It is important to note that these WTP estimates for preventing a new case of chronic bronchitis reflect the perceived welfare effects of living with chronic bronchitis over the entire course of the illness, which can span many years. It is a measure of the present value of the welfare effect that occurs over a multiple-year period. This is somewhat different than the other morbidity effects considered in this analysis which are short-term effects. In using the WTP values for chronic bronchitis we are assigning the full welfare effect for the new chronic bronchitis case in the year in which the clinical onset of the disease occurs. We do the same with the acute morbidity effects, but in those cases the illness typically begins and ends in the same year.

5.4.2 Respiratory Hospital Admissions

WTP estimates for respiratory hospital admissions (RHA) are not available. We therefore use the COI approach. The American Hospital Association reports an average cost per day of a hospital stay of $820 in 1992 dollars (as cited in U.S. Bureau of the Census, 1994). This is inflated to $920 (1994 dollars) using the medical consumer price index. We calculated the average length of stay in the hospital for the 13 ICD-9-CM codes[7] in the Burnett et al. (1995) study (see Chapter 4) using data from the 1992 National Hospital Discharge Survey (Graves, 1994). We found an average length of stay for a respiratory hospital admission of about 6.8 days, which is slightly longer than the overall average length of stay in the hospital for all conditions of approximately 6.2 days (Graves, 1994). The length of stay is multiplied by the average cost per day as an estimate of the medical cost of a RHA. The length of stay is multiplied by the average daily wage (W) as an estimate of the value of lost productivity for employed and not- employed individuals on the presumption that it is a measure of average opportunity costs for all individuals. The medical cost and lost productivity estimates are summed and multiplied by the WTP/COI ratio of 2 to account for additional potential pain and suffering and activity losses not reflected in the COI numbers. The central estimate is thus calculated as follows:

$$\text{Central \$/RHA} = (6.8 \times 910) + (6.8 \times W) \times \text{WTP/COI}. \qquad (5\text{-}4)$$

Therefore, the central estimate is $14,000 (1994 dollars), rounded to the nearest thousand. Applying a plus or minus 50 percent adjustment results in a low estimate of $7,000 and a high estimate of $21,000.

[7] The ICD-9-CM codes included were: 466, 480, 481, 482, 483, 485, 486, 490, 491, 492, 493, 494, and 496. The diseases they correspond to include acute bronchitis, chronic bronchitis, pneumonia, emphysema, and asthma.

5.4.3 Cardiac Hospital Admissions

WTP estimates for cardiac hospital admissions (CHA) are not available. We therefore use the COI approach. The American Hospital Association reports an average cost per day of a hospital stay of $820 in 1992 dollars (as cited in U.S. Bureau of the Census, 1994). This is inflated to $920 (1994 dollars) using the medical consumer price index. We calculated the average length of stay in the hospital for the 4 ICD-9-CM codes[8] in the Burnett et al. (1995) study (see Chapter 4) using data from the 1992 National Hospital Discharge Survey (Graves, 1994). We found an average length of stay for a cardiac hospital admission of about 6.9 days, which is slightly longer than the overall average length of stay in the hospital for all conditions of approximately 6.2 days (Graves, 1994). The length of stay is multiplied by the average cost per day as an estimate of the medical cost of a CHA. The length of stay is multiplied by the average daily wage (W) as an estimate of the value of lost productivity for employed and not- employed individuals on the presumption that it is a measure of average opportunity costs for all individuals. The medical cost and lost productivity estimates are summed and multiplied by the WTP/COI ratio of 2 to account for additional potential pain and suffering and activity losses not reflected in the COI numbers. The central estimate is thus calculated as follows:

$$\text{Central \$/CHA} = (6.9 \times 910) + (6.9 \times W) \times \text{WTP/COI}. \qquad (5\text{-}5)$$

Therefore, the central estimate is $14,000 (1994 dollars), rounded to the nearest thousand. Applying a plus or minus 50 percent adjustment results in a low estimate of $7,000 and a high estimate of $21,000.

5.4.4 Restricted Activity Days

A restricted activity day (RAD) is a measure of illness defined by the Health Interview Survey (HIS) as a day on which illness prevents an individual from engaging in some or all of his or her usual activities. This includes days spent in bed, days missed from work, and days with minor activity restrictions because of illness. WTP estimates for preventing a RAD are not available. We therefore approximate WTP for an average RAD using available COI data and WTP estimates for days with symptoms.

RADs reflect a combination of complete activity restrictions and minor activity restrictions. It is unknown what proportion of RADs attributable to air pollution exposure is minor rather than severe. Recent data from the HIS indicate that about 40 percent of all RADs are bed-

[8] The ICD-9-CM codes included were: 410, 413, 427, and 428. The diseases they correspond to are acute myocardial infection, angina pectoris, cardiac dysrhythmias, and heart failure.

disability days.[9] The results of Ostro (1987) suggest that RADs associated with air pollution exposure may be less severe on average than all RADs. We therefore presume a lower proportion of bed-disability days for this analysis than the national average for all RADs. We select an assumption that 20 percent of RADs due to air pollution exposure are bed-disability days.

WTP studies do provide some information about values for preventing illness symptoms that are probably associated with minor restricted activity days (MRADs). There are no studies specifically addressing the WTP to avoid an MRAD; however, Loehman et al. (1979), Tolley et al. (1986), and Berger et al. (1987) report results from survey respondents who were asked how much they would be willing to pay to avoid a day with various specified symptoms such as serious or minor coughing. The focus of these studies was on respiratory symptoms that might be related to air pollution levels, but the results from each of these studies are difficult to interpret for this analysis because there is fairly wide variability in the responses and because the definitions of symptoms vary. However, Krupnick and Kopp (1988) note that an MRAD is probably more severe than a single minor symptom day (congestion, cough, etc.); hence, they concentrate on the WTP estimates for severe symptoms in Loehman et al. and symptom combinations in Tolley et al. For a central estimate, they select $26 (1994 dollars), which is Loehman's high median value for a severe symptom day.

Productivity losses associated with more serious RADs (bed-disability days) are estimated as equivalent to the daily wage rate for employed individuals. We apply the same measure of lost productivity for not-employed individuals on the presumption that it is a measure of average opportunity costs for all individuals. This lost productivity estimate is multiplied by the WTP/COI ratio of 2 to account for additional potential pain and suffering, additional leisure activity losses, and potential medical costs that are not reflected in the lost productivity estimates. Taking a weighted average of the value for more serious and more minor RADs gives the average value for an air pollution induced RAD as follows:

$$\text{Central \$/RAD} = [0.20 \times W \times \text{WTP/COI}] + [0.80 \times 26]. \qquad (5\text{-}7)$$

Therefore, the central estimate is $60 (rounded to the nearest ten). Applying a plus or minus 50 percent adjustment results in a low estimate of $30 and a high estimate of $90.

5.4.5 Asthma Symptom Days

Krupnick and Kopp (1988) review two studies that provide monetary value estimates for asthma symptom days. The first is a study by Krupnick (1986), which presents the medical expenditures associated with ozone-induced asthma attacks. The expenses vary by the baseline

[9] National Center for Health Statistics (1992) reports average number of restricted activity days for all adults in the United States in 1991 was 16.1, and the average number of bed-disability days was 6.5.

number of attacks and by the assumed prices for medical services. Krupnick and Kopp use these figures as a benchmark for calibrating estimates of WTP.

The second study (Rowe and Chestnut, 1986) is a WTP survey study that obtained asthmatics' estimates of WTP to prevent an increase in "bad asthma days" (BADs). Each respondent defined for himself a BAD on a 1 to 7 severity scale for asthma symptoms. After analyzing the WTP responses, Rowe and Chestnut found WTP estimates that are about 1.8 times greater than the medical costs found by Krupnick. Krupnick and Kopp point out that this finding is consistent with economic logic and lends credibility to both studies. Thus, for WTP values to prevent an asthma attack, Krupnick and Kopp rely on the Rowe and Chestnut estimates.

Rowe and Chestnut found that the WTP responses were positively associated with the baseline number of annual attacks. The values also varied by how an asthmatic defined a BAD. For example, when a BAD was defined as a day with any symptoms, the WTP estimate was $13 in 1994 dollars. At the higher end of the scale, when a BAD was defined as a day with more than moderate symptoms, the WTP was $58. A central estimate is $36. We follow Krupnick and Kopp and adopt these WTP estimates.

5.4.6 Lower Respiratory Symptom Days

Krupnick et al. (1990) estimated the number of study subjects who reported any respiratory symptoms on a given day as a function of air pollutant levels on that day. These included 19 specific symptoms such as coughing, congestion, and throat irritation. The symptoms were noticeable to the subjects, but did not necessarily result in any changes in the person's activities on that day. This health effect therefore includes but is not limited to restricted activity days. In the procedures used to add the health effects cases, restricted activity days are subtracted from acute respiratory symptom days because of the overlap in the definitions of these health effects. The monetary valuation required for acute respiratory days is therefore a value for the days on which symptoms are noticeable but do not restrict normal activities for that day.

Loehman et al. (1979) and Tolley et al. (1986) obtained estimates of WTP to avoid a day with a single minor respiratory symptom such as head congestion or coughing. Their median results per day in 1994 dollars range from $6 to $17. We prefer the median results from these studies because neither study did any adjusting for potentially inaccurate high WTP responses, resulting in reported mean WTP estimates that far exceed the median values. The medians may be too low relative to the average WTP that we would prefer to use in this analysis, but there is less risk of significant upward bias in the median estimates from these studies. We prefer to err in this direction. We select $11 as typical of the range of estimates obtained in these two studies for minor respiratory symptoms. We select a low of $6 and a high of $17.

5.4.7 Summary of Selected Morbidity Values

Table 5-4 provides a summary of the selected monetary values for human morbidity effects.

Table 5-4
Summary of Selected Monetary Values for Morbidity Effects

Morbidity Effect	Estimate per Incident (1994$)			Primary Source	Type of Estimate[1]
	Low	Central	High		
Adult chronic bronchitis	$140,000	$240,000	$380,000	Viscusi et al. (1991) Krupnick and Cropper (1992)	WTP
Respiratory hospital admission	$7,000	$14,000	$21,000	Equation (5-4) Graves (1994)	Adjusted COI
Cardiac hospital admission	$7,000	$14,000	$21,000	Equation (5-5) Graves (1994)	Adjusted COI
Restricted activity day	$30	$60	$90	Equation (5-7) Loehman et al. (1979)	WTP & Adjusted COI
Asthma symptom day	$13	$36	$58	Rowe and Chestnut (1986)	WTP
Lower respiratory symptom day	$6	$11	$17	Loehman et al. (1979) Tolley et al. (1986)	WTP
Selected probability weights for all effects	33%	34%	33%		

[1] WTP = Contingent valuation WTP estimate.
Adjusted COI = COI × 2 to approximate WTP.

CHAPTER 6
RESULTS AND CONCLUSIONS

This chapter presents the quantitative results of the health benefits assessment for Title IV when all the pieces are put together as described in Table 2-1. First, the annual results are presented for 1997 and 2010 using the default assumptions in the calculation of changes in health effects and their monetary valuation for the eastern United States and southern portions of Ontario and Quebec, Canada. These results for 1997 and 2010 are presented in 1994 U.S. dollars and have been adjusted for expected average population growth in the United States and Canada. These results are presented as annual totals for the U.S. and Canadian study areas, with mean estimates from the distribution of the final results for each year presented as well as the 20th and 80th percentiles of the distribution. State-by-state estimates of the mean annual estimates and present value calculations for the 1995 to 2010 period are also presented. Results of some sensitivity analyses are then presented to give a sense of the directions and magnitudes of effects of key assumptions in the assessment calculations.

6.1 ANNUAL RESULTS BASED ON DEFAULT ASSUMPTIONS

Table 6-1 shows the mean, 20th percentile, and 80th percentile estimates based on the default assumptions for the 31-state eastern United States area for 1997 and 2010. Table 6-2 gives comparable results for the southern portions of Ontario and Quebec. The mean estimates are calculated using the probability distributions assigned to each health effect category, as discussed in Chapters 4 and 5. The 1997 estimates compare the annual median sulfate aerosol concentrations predicted as a result of estimated SO_2 emissions in 1997 under the Title IV requirements, with predicted banking of emissions allowances incorporated into the estimates,[1] with sulfate concentrations estimated based on 1985 SO_2 emissions. The 2010 estimates are based on predicted SO_2 emissions in 2010, after Title IV is expected to be fully implemented, versus what SO_2 emissions are predicted to have been in 2010 without Title IV but with all other provisions of the Clean Air Act in place.

The annual estimated mean health benefits in the eastern United States (Table 6-1) for 1997 are $10.6 billion, and they rise to $40.0 billion by the year 2010. The mean estimates for Canada (Table 6-2) add an additional $908 million in 1997 and $955 million in 2010. The mean results represent the estimated annual number of cases of each type of health effect expected to be prevented as a result of Title IV versus what would have occurred without

[1] The banking assumptions suggest that SO_2 emissions will be lower in years between 1995 and 2000 than they would have been without banking, but that the rate of decline in emissions will be somewhat slower after the year 2000 (see Figure 3-1).

Table 6-1
Estimates of Annual Human Health Benefits of Title IV for the Eastern United States with Default Assumptions (millions of 1994 dollars)

	1997						2010					
	Annual Number of Cases Prevented			Annual Monetary Value			Annual Number of Cases Prevented			Annual Monetary Value		
Health Effect	20th Percentile	Mean	80th Percentile	20th Percentile	Mean	80th Percentile	20th Percentile	Mean	80th Percentile	20th Percentile	Mean	80th Percentile
Premature Mortality	408	2,568	5,714	$1,428.0	$9,307.2	$19,999.0	1,539	9,678	21,544	$5,386.5	$35,234.8	$75,404.0
Chronic Bronchitis (new cases)	1,648	3,864	6,590	$507.5	$974.0	$1,377.5	6,179	14,564	24,715	$1,903.0	$3,705.8	$5,165.3
Respiratory Hospital Admissions	663	805	918	$5.7	$11.3	$17.1	2,501	3,036	3,462	$21.5	$42.4	$64.6
Cardiac Hospital Admissions	510	673	867	$4.6	$9.4	$13.9	1,924	2,552	3,270	$17.5	$35.7	$52.5
Asthma Symptom Days	791,232	1,604,341	2,373,697	$20.9	$56.9	$93.2	2,983,490	5,951,693	8,950,470	$78.7	$212.9	$351.3
Restricted Activity Days (net)	1,202,785	2,467,066	3,809,253	$70.6	$147.0	$228.6	4,514,939	9,283,999	14,298,930	$265.0	$554.7	$857.9
Days with Lower Respiratory Symptoms (net)	2,028,424	5,002,393	7,259,946	$31.8	$56.7	$90.0	7,614,168	18,619,000	27,251,920	$119.3	$212.8	$338.0
Total Annual Health Benefits				$3,219.1	$10,562.3	$20,684.1				$12,131.5	$39,999.0	$77,915.5

Table 6-2
Estimates of Annual Human Health Benefits of Title IV for Ontario and Quebec, Canada States with Default Assumptions (millions of 1994 dollars)

Health Effect	1997						2010					
	Annual Number of Cases Prevented			Annual Monetary Value			Annual Number of Cases Prevented			Annual Monetary Value		
	20th Percentile	Mean	80th Percentile	20th Percentile	Mean	80th Percentile	20th Percentile	Mean	80th Percentile	20th Percentile	Mean	80th Percentile
Premature Mortality	35	217	483	$122.5	$801.2	$1,690.5	37	232	517	$129.5	$839.2	$1,809.5
Chronic Bronchitis (new cases)	140	329	562	$43.3	$83.3	$117.4	150	355	601	$46.3	$91.0	$125.8
Respiratory Hospital Admissions	56	68	78	$0.5	$0.9	$1.4	60	73	83	$0.5	$1.0	$1.6
Cardiac Hospital Admissions	43	57	73	$0.4	$0.8	$1.2	46	61	78	$0.4	$0.9	$1.3
Asthma Symptom Days	66,915	133,825	200,746	$1.8	$4.8	$7.9	71,594	142,267	214,783	$1.9	$5.1	$8.4
Restricted Activity Days (net)	97,734	199,194	309,526	$5.7	$12.0	$18.6	104,568	215,270	331,168	$6.1	$13.0	$19.9
Days with Lower Respiratory Symptoms (net)	164,822	401,231	589,916	$2.6	$4.5	$7.3	176,347	433,821	631,165	$2.8	$4.9	$7.8
Total Annual Health Benefits				$273.3	$907.6	$1,746.9				$290.8	$955.0	$1,868.1

Title IV. Estimates for both years and both countries are in 1994 U.S. dollars, and have been adjusted for expected population growth based on the mid-forecasts of the U.S. Census (U.S. Bureau of the Census, 1994). Canadian population growth estimates are from World Bank population projections (Bos et al., 1992).

The health benefit estimates are dominated by the premature mortality and the chronic bronchitis effects. The numbers of cases in these health effects categories are relatively small, but the high monetary values per case result in large monetary benefits for these categories. Premature mortality reductions alone account for about 88 percent of the total health benefits. Chronic bronchitis reductions are about 9 percent of the total. The combination of the premature mortality reductions and the chronic bronchitis reductions represent about 97 percent of the total health benefits.

The largest numbers of cases reduced are for asthma symptom days, restricted activity days, and days with acute lower respiratory symptoms. The restricted activity days are net of days in the hospital and asthma symptom days because these health effects categories may substantially overlap. The lower respiratory symptom days are net of the fraction of restricted activity days that might also be attributed to lower respiratory symptoms. In 2010, the estimated mean reduction in the number of asthma symptom days because of Title IV is about 6 million in the eastern United States; the mean net restricted activity days prevented is about 9 million; and the mean estimated number of days with acute lower respiratory symptoms prevented, net of restricted activity days, is about 19 million. Together, these represent about 3 percent of the total mean monetary health benefits.

The other categories of health effects (respiratory and cardiac hospital admissions) together represent only about 0.2 percent of the total monetary health benefits. This is because relatively small risks and small monetary values combine to give relatively small total benefit amounts.

The estimates of reductions in health effects in Canada are based on estimates of changes in sulfate aerosol concentrations in Canada predicted to result from changes in SO_2 emissions generated in the United States. The estimates for Canada are primarily in the Windsor-Quebec corridor, where the greatest share of the Canadian population likely to be affected by the transport of SO_2 emissions in the eastern United States is located. The estimates for Canada represent an additional 9 percent of the Title IV benefits in 1997 estimated for the United States population. The estimates for Canada do not increase substantially from 1997 to 2010 because the estimated reductions in sulfate concentrations in Canada do not change substantially from 1997 to 2010. This is presumably because the upwind locations in the United States that affect this area of Canada see their greatest reduction in SO_2 emissions in the first phase of the Title IV program. In 2010, the estimates for Canada add an additional 2 percent to the 2010 estimates for the United States population.

Most of the selected concentration-response and monetary value estimates are based on statistically derived results. These estimates therefore have some quantified statistical

uncertainty based on the estimated statistical variance in the results. For all of the health effects and monetary value estimates, low and high as well as central estimates were selected based on the estimated statistical variance and analyst judgment. In general, the selected high and low estimates represent plus and minus approximately one statistical standard error.

It is not appropriate to combine all the "low" estimates or all the "high" estimates to calculate upper and lower bounds on the final estimates, because it is highly unlikely that either all the lows or all the highs would be correct. Such extreme assumptions would significantly overstate the statistical uncertainty in the estimates. Instead, we have assigned probability weights to the low, central, and high estimates which when incorporated in the calculation process allow determination of the probability distribution of the total health benefit results.

The mean, 20th percentile, and 80th percentile estimates shown for each year in Tables 6-1 and 6-2 are the result of this procedure. All three of these estimates for each health effect category are based on the default assumptions, with each estimate representing a different selected point in the estimated probability distribution calculated for the health effect category and for total health benefits. The 20th percentile of the distribution of total health benefits for 2010 in the eastern U.S. is about $12 billion with the default assumptions. This means that 20 percent of the estimated values are below this amount and 80 percent are above it. The 80th percentile of the distribution is about $78 billion with the default assumptions. This means that 20 percent of the estimated values are above this amount and 80 percent are below it. Thus, sixty percent of the distribution of the annual total health benefits in 2010 in the eastern U.S. falls between $12 billion and $78 billion, with a mean value of $40 billion, when the default assumptions are used and the selected probability weights for each selected low, central, and high estimate are incorporated into the calculations.

Table 6-3 lists the estimated 1997 and 2010 mean annual health benefits by state and province. The per capita health benefits are calculated by dividing the total annual benefits in each state or province by the estimated 1997 and 2010 populations in each state and province (based on national average population forecasts). These give a picture of the distribution of the health benefits across the region. Five states have average annual per capita health benefits in 2010 that exceed $400. These are West Virginia, Georgia, Kentucky, Tennessee, and Alabama. Eight more states have per capita benefits between $200 and $400. These are Maryland, Delaware, Virginia, North Carolina, South Carolina, Ohio, Indiana, and Mississippi. The largest per capita benefits are thus in the Ohio River Valley, the central Atlantic states, the central and southern Appalachian states, and the eastern Gulf coast states. The lowest benefits are in the northern states of Minnesota, Maine, Vermont, and New Hampshire.

The average annual per capita estimate for all of the eastern United States for 1997 is about $57, and rises to about $194 in 2010. The average per capita benefit estimate for 1997 and 2010 in Canada is about $50, which is very similar to the 1997 average per capita estimate for the eastern United States.

Table 6-3
Mean Estimated Health Effects Benefits of Title IV by State

State	Annual Monetary Value of Health Benefits (1994 dollars)			
	1997		2010	
	Total (millions)	Average per Capita	Total (millions)	Average per Capita
Maine, Vermont, New Hampshire	$129	$41	$128	$37
Massachusetts, Connecticut, Rhode Island	$956	$86	$580	$47
New York	$1,160	$60	$1,658	$76
Pennsylvania	$943	$74	$2,633	$183
New Jersey	$341	$41	$1,112	$119
Maryland, Delaware, D.C.	$418	$64	$1,614	$221
Virginia	$394	$59	$2,535	$339
West Virginia	$245	$127	$950	$439
North Carolina, South Carolina	$412	$38	$4,818	$394
Georgia	$765	$109	$3,508	$448
Florida	$35	$2	$2,849	$182
Ohio	$1,058	$90	$3,344	$255
Michigan	$325	$32	$1,168	$104
Illinois	$340	$28	$1,713	$124
Indiana	$512	$85	$1,515	$226
Wisconsin	$71	$13	$334	$56
Kentucky	$777	$195	$2,049	$460
Tennessee	$881	$167	$2,741	$465
Alabama	$312	$72	$1,974	$404
Mississippi	$107	$38	$654	$210
Minnesota	($72)	($15)	$88	$17
Iowa	$1	$0	$176	$52
Missouri	$242	$44	$721	$117
Arkansas	$123	$49	$285	$100
Louisiana	$87	$19	$852	$167
31-State U.S. Regional Total	**$10,562**	**$57**	**$39,999**	**$194**
Ontario	$673	$62	$789	$68
Quebec	$235	$32	$166	$21
Canadian Total	**$908**	**$50**	**$955**	**$49**

6.2 AGGREGATE HEALTH BENEFITS 1997 TO 2010

The reduction in SO_2 emissions due to Title IV is expected to increase each year after 1997 until full implementation is reached in 2010. The first year for which ICF Resources (1994) reports a specific estimate for an SO_2 emissions reduction due to Title IV is 1997, when the Phase I requirements are expected to be fully implemented. The health benefits will therefore be expected to occur each year during this period, and increase each year until full implementation of Title IV is reached. The estimates of emissions reductions expected are based on a comparison of emissions expected with and without Title IV. ICF Resources reported Title IV emissions reductions estimates for 1997, 2000, 2005, and 2010.

After 2010, Title IV may continue to result in lower SO_2 emissions than would have occurred without Title IV, but projections of what emissions would have been without Title IV have not been made by EPA beyond 2010. The predicted trend in emissions for this "no Title IV" scenario up to 2010 is fairly flat, with a very slight increase in emissions from 2000 to 2010. Some analysts have predicted that after 2010, SO_2 emissions might have begun to decline even without Title IV requirements, because as old facilities are replaced, the new ones are subject to more stringent new source performance standards and other permitting requirements. However, it remains highly uncertain as to how quickly, if at all, the Title IV emissions limits would have been reached if Title IV had not been enacted.

The RADM was run for this assessment to obtain estimates of ambient outdoor sulfate aerosol concentrations in the eastern United States for 1997 and 2010 with and without Title IV scenarios. To estimate the total health benefits over the 1997 to 2010 period, annual estimates for each year in the period are needed. We estimated the annual health benefits for 1998 to 2009 using the 1997 and 2010 health benefits estimates described in the previous section and the emissions reductions estimates for 1997, 2000, 2005, and 2010 from ICF Resources (1994). We assume that health benefits occur in proportion to the emissions reductions to obtain the health benefits estimates for 2000 and 2005. For example, in the year 2000, the predicted increase in emissions reductions over the 1997 level is about 46.6 percent of the additional reduction expected by 2010 over the 1997 level (see Table 6-4: (6.38 - 4.07)/(9.03 - 4.07) = 0.466). We therefore estimate that 46.6 percent of the difference between health benefits in 1997 and 2010 will be achieved in 2000 (i.e., \$10.562B + 0.466 (\$39.999B - \$10.562B) = \$24.280B). The same procedure was used to estimate the health benefits in the year 2005. We then linearly interpolate between 1997 and 2000, 2000 and 2005, and 2005 and 2010 to obtain estimates of annual health benefits for each intervening year.

The resulting annual estimates of health benefits are reported in Table 6-4. The second column shows the estimated emissions reductions for 1997, 2000, 2005, and 2010 provided by ICF Resources. The annual health benefits estimates in the third and fourth columns for 1997 and 2010 are based on the RADM estimates of changes in sulfate concentrations under each scenario and the health effects and monetary valuation procedures as described in Chapters 4 and 5 of this report. These are mean estimates for the eastern United States and Canada based on the default assumptions. The last row shows aggregated health benefits from 1997 to 2010,

Table 6-4
Mean Annual Health Benefits Estimates
1997 to 2010

Year	Estimated Reduction in Annual SO_2 Emissions Due to Title IV[1] (million tons)	Eastern United States Estimated Annual Monetary Health Benefit (millions of 1994 dollars)	Ontario and Quebec Estimated Annual Monetary Health Benefit (millions of 1994 dollars)
1997	**4.07**	**$10,562**	**$906**
1998		$15,135	$915
1999		$19,707	$922
2000	**6.38**	**$24,280**	**$930**
2001		$26,070	$933
2002		$27,859	$935
2003		$29,649	$938
2004		$31,439	$941
2005	**7.89**	**$33,229**	**$944**
2006		$34,583	$946
2007		$35,937	$948
2008		$37,291	$951
2009		$38,645	$953
2010	**9.03**	**$39,999**	**$955**
Total Undiscounted		**$404,384**	**$13,120**

[1] Based on emissions estimates from ICF Resources (1994).

in 1994 U.S. dollars, undiscounted. Undiscounted, the aggregate health benefit for this period is $404 billion for the United States and $13 billion for Canada.

Table 6-5 presents 1995 present values of total health benefit estimates from 1997 to 2010 for the eastern United States and Canada using two alternative discount rates. Given uncertainty about what the correct discount rate is for aggregating these kinds of benefits over time, we select a 7 percent rate based on OMB recommendations for analyzing government programs (OMB, 1992), and a possible lower rate of 3 percent based on evidence of a social rate of discount.[2] The discount rate has a significant effect on aggregate values over a time period as long as this so it is useful to illustrate the results using alternative rates. We have made an adjustment to each of the selected discount rates, because benefits are expected to grow over time due to increases in real income that have not been accounted for in the annual estimates presented previously. Applying discounting without making these adjustments would inappropriately downward bias the present value estimates. Expected real income growth was accounted for by deducting 0.94 percent from each discount rate based on expected annual average growth in real income from 1997 to 2010 (U.S. Department of Commerce, 1990). Real income growth is expected to increase health benefits by increasing willingness to pay for prevention of health effects. We make a rough assumption here that WTP increases in proportion to real income, although there is not sufficient empirical data to verify the accuracy of this assumption at this time. WTP could in actuality increase in either greater or lesser proportion to real income growth.

Table 6-5
Total Present Value in 1995 of Mean Health Benefits
1997 to 2010 with Default Assumptions

Net Discount Rate[1]	Eastern United States (billions of 1994 dollars)	Ontario and Quebec (billions of 1994 dollars)
6.06%	$234.7	$8.2
2.06%	$333.0	$11.1

[1] The discount rates were derived as: 7.00% - 0.94% (average per capita income growth 1997-2010) giving a net discount rate of 6.06% and 3.00% - 0.94% giving a net discount rate of 2.06% (U.S. Dept. of Commerce, 1990).

[2] Freeman (1993) presents a thorough discussion of discounting and evidence regarding appropriate discount rates for environmental programs. Alternative arguments can be made to support alterative discount rates. We select two rates from the range that is typically discussed.

6.3 SENSITIVITY ANALYSES RESULTS

There are many sources of uncertainty and potential error in the mean estimates of health benefits for Title IV reported in the previous two sections. This section presents results of some specific sensitivity analyses conducted to determine the potential effect on the results of different assumptions than those selected for the default estimates. These alternative assumptions reflect some of the key uncertainties identified in the health effects quantification and valuation chapters. The analyses reported in this section cover only the uncertainties in the concentration-response functions and in the monetary valuation of health effects. Additional uncertainties also exist in the estimates of change in SO_2 emissions and ambient sulfate concentrations that are used as inputs to the health benefits estimates. These uncertainties were discussed qualitatively in Chapter 2, but are not treated quantitatively here because these inputs are based on analyses that have been reported elsewhere.

The uncertainty and sensitivity analyses reported here are those that are reasonably amenable to quantitative treatment. It is important to recognize that there are many sources of uncertainty that are not possible to quantify, and that these sensitivity tests are therefore not a comprehensive treatment of all possible sources of uncertainty. What these tests provide, however, is an indication of how the results might change if we found that some of the key default assumptions in the health effects quantification and valuation procedures were inappropriate.

The selected sensitivity tests are based on different assumptions that we think have some nonzero probability of being accurate. A completely comprehensive range of possible results given all the uncertainties in this assessment would include zero health benefits at the low end and a very large number at the high end. That kind of comprehensive range is probably not very helpful for policy makers without some guidance in understanding the likelihood that different results within the range could be correct. We try to give this interpretation, at least qualitatively, for each of the sensitivity test results.

Each of the sensitivity tests illustrated in Table 6-6 is discussed below. They all represent estimates of mean annual health benefits for 1997 and 2010, in 1994 U.S. dollars. Each is calculated in the same way that the default mean was calculated, except for the specified assumption change. A comparison with the default means in Tables 6-1 and 6-2 therefore illustrates the effect of the change in the assumption.

Health Effects Thresholds

As discussed in Chapter 4, there is considerable uncertainty about whether there is a "safe" level of sulfate aerosol exposure that does not cause any harmful health effects. There is no definitive quantitative evidence that such a threshold exists, but neither is there proof that any amount of sulfate aerosol exposure causes some harmful effect in at least some people. We selected three possible threshold levels to illustrate how this could affect the results. The

Table 6-6
Sensitivity Analyses Results

Assumptions	Estimated Annual Health Benefits (billions of 1994 dollars)
United States 1997	
Threshold = 5.0 $\mu g/m^3$ SO_4	$3.1
Threshold = 3.6 $\mu g/m^3$ SO_4	$6.7
Threshold = 1.6 $\mu g/m^3$ SO_4	$10.8
Selected SO_4 Health Risks × 0.4	$4.8
United States 2010	
Threshold = 5.0 $\mu g/m^3$ SO_4	$15.0
Threshold = 3.6 $\mu g/m^3$ SO_4	$28.3
Threshold = 1.6 $\mu g/m^3$ SO_4	$39.3
Selected SO_4 Health Risks × 0.4	$18.5
Canada 1997	
Threshold = 5.0 $\mu g/m^3$ SO_4	$0.0
Threshold = 3.6 $\mu g/m^3$ SO_4	$0.0
Threshold = 1.6 $\mu g/m^3$ SO_4	$0.7
Selected SO_4 Health Risks × 0.4	$0.4
Canada 2010	
Threshold = 5.0 $\mu g/m^3$ SO_4	$0.0
Threshold = 3.6 $\mu g/m^3$ SO_4	$0.0
Threshold = 1.6 $\mu g/m^3$ SO_4	$0.9
Selected SO_4 Health Risks × 0.4	$0.5

existence of a threshold could only decrease, not increase, the results because it means that further reductions in sulfate levels in areas that are already at or close to the threshold would not yield any health benefits.

We selected alternative threshold assumptions of 5.0 $\mu g/m^3$, 3.6 $\mu g/m^3$, and 1.6 $\mu g/m^3$ annual median SO_4 concentrations to illustrate the potential effects of alternative threshold assumptions on the results of this analysis. As discussed in Chapter 4, none of these concentrations has been identified as a true threshold, but each represents a mean or low end

value for the range of concentrations considered in one of the epidemiology studies that concentration-response functions were taken from. The threshold calculation was implemented as follows. Any RADM grid cell with a base case 2010 (without Title IV) level of annual 50th percentile SO_4 at the threshold concentration or less was assigned zero health benefits for the Title IV emissions reductions.[3] Further, health benefits were calculated only for reductions in annual 50th percentile down to the threshold. For the 5.0 $\mu g/m^3$ threshold, for example, if the level without Title IV was 5.5 and the level with Title IV was 4.5, the health benefits calculations for that grid cell were made only for the 5.5 minus 5.0 reduction of 0.5. Any additional reduction below 5.0 was presumed to provide no health benefit.

The results indicate that with a threshold of 5.0 $\mu g/m^3$ SO_4, annual health benefits are substantially reduced relative to the default mean, falling very close to the 20 percentile estimates. At thresholds above 5.0 the health benefit estimates would diminish even more. A threshold of 3.6 $\mu g/m^3$ SO_4 results in a health benefit estimate that falls about midway between the default mean and the 20 percentile default estimates. At a threshold of 1.6 (or lower), the health benefit estimate is virtually unchanged from the default mean. This illustrates the significance of the threshold question and shows that this continues to be an important research issue from the standpoint of evaluating the health benefits of pollution emission reductions.

Lower Health Risks for Sulfates

As discussed in Chapter 4, there is a possibility that the sulfate based concentration-response functions may be somewhat upwardly biased because of the typical collinearity between sulfates and other fine particulate constituents in the ambient air. For this sensitivity test we multiply the sulfate based concentration-response functions by 0.4, which is the average ratio between measured sulfates and measured $PM_{2.5}$ in the eastern United States. This is the maximum adjustment that would be required if the sulfate coefficients represented the total effects of all $PM_{2.5}$. This adjustment reduces the annual health benefit estimate to about $8.5 billion, in 2010, which is close to the 20th percentile estimate with the default assumptions. The true sulfate effect is probably between this and the mean default estimate because the sulfate coefficients probably do reflect some, but are unlikely to reflect all, of the effects of other harmful constituents of $PM_{2.5}$ as well as the effects of sulfates alone.

[3] RADM estimates that in a few locations sulfate concentrations will be higher with Title IV than without Title IV. In general, these places have very low sulfate concentrations and may fall below the threshold concentration under consideration. Because a cell below the threshold concentration is assigned zero health benefits, no negative health benefits are calculated in the threshold analyses. This is why the 1997 results using the 1.6 threshold assumption slightly exceed the mean results when no threshold is assumed.

6.4 CONCLUSIONS

The results of this assessment show that the potential health benefits of reductions in exposures to sulfate aerosols in the eastern United States as a result of the SO_2 emissions reductions required by Title IV are substantial. Based on what we believe is a reasonable interpretation of the available epidemiology and economic evidence on potential health effects of sulfate aerosols and their monetary value, we estimate that the annual health benefits in the United States of the Title IV required reductions in SO_2 in 2010 are more likely than not to fall between $12 billion and $78 billion. There is reason to expect some possible upward bias at the higher end of this range. The results of the sensitivity analyses suggest that there is a good chance that the annual benefits in the United States fall between $12 billion and $40 billion.

We have been careful throughout the report to highlight key assumptions and uncertainties that exist in the quantification procedures used in this assessment, especially in the health effects quantification and valuation portions of the assessment which are the focus of this report. Most of these uncertainties cannot be resolved without substantial new research on several topics. The most important empirical uncertainties in the health effects quantification are:

- What is the relative harmfulness of sulfate aerosols versus other fine particulate matter?
- Is there a threshold for health effects from sulfate aerosols, and if so, what is it?
- Is there sufficient evidence to presume that the observed association between sulfate concentrations and human health effects is causative?

The most important uncertainties in the monetary valuation of health effects are:

- Are WTP estimates for risks of accidental deaths in populations of average health status applicable to premature mortality risks associated with air pollutant exposures?
- How do WTP values for premature mortality and other health risks vary for the elderly and for those whose health is already poor?
- Will the available WTP estimates for chronic respiratory disease be verified by new WTP research?

CHAPTER 7
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