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Benefits of the Proposed Inter-State Air Quality Rule

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Benefits of the Proposed Inter-State Air Quality Rule

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SECTION 1

EXECUTIVE SUMMARY

The Clean Air Act (CAA) contains a number of requirements to address nonattainment of the fine particulate matter ($PM_{2.5}$) and the 8-hour ozone national ambient air quality standards (NAAQS), including requirements that States address interstate transport contributing to such nonattainment. CAA Section 110(a)(2)(D) requires that the State Implementation Plans (SIPs) necessary to meet these standards contain adequate provisions to prohibit air pollutant emissions within those States from "contribut[ing] significantly to nonattainment in, or interfer[ing] with maintenance by," a downwind State. The EPA is proposing a rule to reduce interstate transport of fine particulate matter and ozone (Inter-State Air Quality Rule hereinafter referred to as IAQR) in 29 States and the District of Columbia to ensure that SIPs provide for necessary regional reductions of emissions of sulfur dioxide (SO_2) and/or nitrogen oxides ($NO_{x)}$, that are important precursors of $PM_{2.5}$ (NO_x and SO_2) and ozone (NO_x). The EPA is proposing that emissions reductions be implemented in two phases, with the first phase in 2010 and the second phase in 2015.

This document presents the health and welfare benefits of the IAQR and compares the benefits of this proposal to the estimated costs of implementing the rule in 2010 and 2015. Significant health and welfare benefits are likely to occur as a result of this rule. Thousands of deaths and other serious health effects would be prevented each year. The EPA is able to monetize annual benefits of approximately $58 billion in 2010 and approximately $84 billion in 2015. Table 1-1 presents the primary estimates of reduced incidence of PM- and ozone-related health effects for the years 2010 and 2015 for the regulatory control strategy. In interpreting the results, it is important to keep in mind the limited set of effects we are able to monetize. Specifically, the table lists the PM- and ozone-related benefits associated with the reduction of ambient PM and ozone levels. These benefits are substantial both in incidence and dollar value. In 2010, we estimate that reduction in exposure to $PM_{2.5}$ will result in approximately 9,600 fewer premature deaths annually associated with $PM_{2.5}$, as well as 5,200 fewer cases of chronic bronchitis, 13,000 fewer nonfatal heart attacks (acute myocardial infarctions), 8,900 fewer hospitalizations (for respiratory and cardiovascular disease combined), and significant reductions in days of restricted activity due to respiratory illness

Table 1-1. Estimated Reductions in Incidence of Health Effects

Endpoint	Constituent	2010 Estimated Reduction	2015 Estimated Reduction
Premature Mortality-adult	$PM_{2.5}$	9,600	13,000
Mortality-infant	$PM_{2.5}$	22	29
Chronic bronchitis	$PM_{2.5}$	5,200	6,900
Acute myocardial infarction-total	$PM_{2.5}$	13,000	18,000
Hospital admissions - respiratory	$PM_{2.5}$, O_3	5,200	8,100
Hospital admissions - cardiovascular	$PM_{2.5}$	3,700	5,000
Emergency room visits, respiratory	$PM_{2.5}$, O_3	7,100	9,400
Acute bronchitis	$PM_{2.5}$	12,000	16,000
Lower respiratory symptoms	$PM_{2.5}$	140,000	190,000
Upper respiratory symptoms	$PM_{2.5}$	490,000	620,000
Asthma exacerbation	$PM_{2.5}$	190,000	240,000
Acute respiratory symptoms (MRADs)	$PM_{2.5}$, O_3	6,400,000	8,500,000
Work loss days	$PM_{2.5}$	1,000,000	1,300,000
School loss days	O_3	180,000	390,000

MRADs = minor restricted activity days

(with an estimate of 6.4 million fewer cases). We also estimate substantial health improvements for children from reductions in upper and lower respiratory illnesses, acute bronchitis, and asthma attacks. Ozone health-related benefits are expected to occur during the summer ozone season (usually ranging from May to September in the eastern U.S.). Based on modeling for 2010, ozone-related health benefits are expected to include 1,000 fewer hospital admissions for respiratory illnesses, 120 fewer emergency room admissions for asthma, 280,000 fewer days with restricted activity levels, and 180,000 fewer days where children are absent from school because of illnesses. In addition, recent reports by Thurston and Ito (2001) and the World Health Organization (WHO) support an independent ozone mortality impact, and the EPA Science Advisory Board has recommended that the EPA reevaluate the ozone mortality literature for possible inclusion in the estimate of total benefits. Based on these new analyses and recommendations, EPA is sponsoring three independent meta-analyses of the ozone-mortality epidemiology literature to inform a determination on inclusion of this important health endpoint. Upon completion and

peer-review of the meta-analyses, EPA will determine whether benefits of reductions in ozone-related mortality will be included in the benefits analysis for the final IAQR.

Table 1-2 presents the estimated monetary value of reductions in the incidence of health and welfare effects. PM-related health benefits and ozone benefits are estimated to be approximately $56.9 billion and $82.4 billion annually in 2010 and 2015, respectively. Estimated annual visibility benefits in Southeastern Class I areas brought about by the IAQR are estimated to be $880 million in 2010 and $1.4 billion in 2015. All monetized estimated values are stated in 1999$. Table 1-3 presents the total annual monetized benefits for the years 2010 and 2015. This table also indicates with a "B" those additional health and environmental effects that we were unable to quantify or monetize. These effects are additive to the estimate of total benefits, and the EPA believes there is considerable value to the public of the benefits that could not be monetized. A listing of the benefit categories that could not be quantified or monetized in our estimate is provided in Table 1-4. Major benefits not quantified for this proposed rule include the value of increases in yields of agricultural crops and commercial forests, value of improvements in visibility in places where people live and work and recreational areas outside of federal Class I areas, and value of reductions in nitrogen and acid deposition and the resulting changes in ecosystem functions.

In summary, EPA's primary estimate of the annual benefits of the rule is approximately $58 + B billion in 2010. In 2015, total monetized annual benefits are approximately $84 + B billion. These estimates account for growth in the willingness to pay for reductions in environmental health risks with growth in real gross domestic product (GDP) per capita between the present and the years 2010 and 2015.

1.1 Benefit-Cost Comparison

The estimated annual social benefits of the rule are compared to the annual estimated cost to implement the proposed rule in Table 1-3. Estimates of the annual costs of implementing the rule are $3 and $4 billion in 2010 and 2015, respectively (1999$). For further information concerning the costs of the proposed rule, please see "Preliminary Analysis of the Costs of the Inter-State Air Quality Rule—January 2004."

Table 1-2. Estimated Monetary Value of Reductions in Incidence of Health and Welfare Effects (millions of 1999$)

Endpoint	Constituent	2010 Estimated Monetary Value of Reductions	2015 Estimated Monetary Value of Reductions
Preamature Mortality-adult	$PM_{2.5}$	$53,000	$77,000
Chronic bronchitis	$PM_{2.5}$	$1,900	$2,700
Acute myocardial infarction	$PM_{2.5}$	$1,100	$1,500
Acute respiratory symptoms (MRADs)	$PM_{2.5}$, O_3	$320	$440
Work loss days	$PM_{2.5}$	$140	$170
Mortality-infant	$PM_{2.5}$	$130	$180
Hospital admissions, respiratory	$PM_{2.5}$, O_3	$85	$130
Hospital admissions, cardiovascular	$PM_{2.5}$	$78	$110
School loss days	O_3	$13	$28
Worker productivity	O_3	$8.0	$17
Asthma exacerbation	$PM_{2.5}$	$8.0	$11
Acute bronchitis	$PM_{2.5}$	$4.3	$5.7
Lower respiratory symptoms	$PM_{2.5}$	$2.3	$3.0
Upper respiratory symptoms	$PM_{2.5}$	$13	$17
Emergency room visits, respiratory	$PM_{2.5}$, O_3	$2.0	$2.6
Visibility, Southeastern Class I areas	Light extinction	$880	$1,400
TOTAL + B*		$58,000	$84,000

MRADs= minor restricted activity days

B= nonmonetized benefits

* Note total dollar benefits are rounded to the nearest billion and column totals may not add due to rounding.

Table 1-3. Summary of Annual Benefits, Costs, and Net Benefits of the Inter-State Air Quality Rule

Description	2010 (billions of 1999 dollars)	2015 (billions of 1999 dollars)
Social costs[a]	$2.9	$3.7
Social benefits[b,c]		
Ozone-related benefits	$0.1	$0.1
PM-related health benefits	$56.8 + B	$82.3 + B
Visibility benefits	$0.9	$1.4
Net benefits (benefits-costs)[c,d]	$55 + B	$80 + B

[a] Note that costs are the annual total costs of reducing pollutants including NO_x and SO_2.

[b] As the table indicates, total benefits are driven primarily by PM-related health benefits. The reduction in premature fatalities each year accounts for over 90 percent of total benefits. Benefits in this table are associated with NO_x and SO_2 reductions.

[c] Not all possible benefits or disbenefits are quantified and monetized in this analysis. B is the sum of all unquantified benefits and disbenefits. Potential benefit categories that have not been quantified and monetized are listed in Table 1-4.

[d] Net benefits are rounded to the nearest billion. Columnar totals may not sum due to rounding.

Thus, the annual net benefit (social benefits minus social costs) of the program is approximately $55 + B billion in 2010 and $80 + B billion in 2015. Therefore, implementation of the proposed rule is expected to provide society with a net gain in social welfare based on economic efficiency criteria. As Table 1-2 shows, although mortality benefits account for over 90 percent of total monetized benefits, the economic value of morbidity benefits alone exceed the cost of the proposed rule. As discussed in section IX of the notice for this rulemaking, we did not complete air quality modeling that precisely matches the IAQR region. We anticipate that any differences in the estimates presented due to the modeling region analyzed will be small.

Every benefit-cost analysis examining the potential effects of a change in environmental protection requirements is limited to some extent by data gaps, limitations in model capabilities

Table 1-4. Additional Nonmonetized Benefits of the Inter-State Air Quality Rule

Pollutant	Unquantified Effects
Ozone Health	Premature mortality[a] Increased airway responsiveness to stimuli Inflammation in the lung Chronic respiratory damage Premature aging of the lungs Acute inflammation and respiratory cell damage Increased susceptibility to respiratory infection Non-asthma respiratory emergency room visits
Ozone Welfare	Decreased yields for commercial forests Decreased yields for fruits and vegetables Decreased yields for commercial and non-commercial crops Damage to urban ornamental plants Impacts on recreational demand from damaged forest aesthetics Damage to ecosystem functions
PM Health	Low birth weight Changes in pulmonary function Chronic respiratory diseases other than chronic bronchitis Morphological changes Altered host defense mechanisms Non-asthma respiratory emergency room visits
PM Welfare	Visibility in many Class I areas Residential and recreational visibility in non-Class I areas Soiling and materials damage Damage to ecosystem functions
Nitrogen and Sulfate Deposition Welfare	Impacts of acidic sulfate and nitrate deposition on commercial forests Impacts of acidic deposition to commercial freshwater fishing Impacts of acidic deposition to recreation in terrestrial ecosystems Reduced existence values for currently healthy ecosystems Impacts of nitrogen deposition on commercial fishing, agriculture, and forests Impacts of nitrogen deposition on recreation in estuarine ecosystems Damage to ecosystem functions
Mercury Health	Neurological disorders Learning disabilities Developmental delays Potential cardiovascular effects* Altered blood pressure regulation* Increased heart rate variability* Myocardial infarction* Potential reproductive effects*
Mercury Deposition Welfare	Impact on birds and mammals (e.g., reproductive effects) Impacts to commercial, subsistence, and recreational fishing Reduced existence values for currently healthy ecosystems

[a] Premature mortality associated with ozone is not separately included in this analysis.

* These are potential effects as the literature is either contradictory or incomplete.

(such as geographic coverage), and uncertainties in the underlying scientific and economic studies used to configure the benefit and cost models. Deficiencies in the scientific literature often result in the inability to estimate quantitative changes in health and environmental effects, such as potential increases in fish populations due to reductions in nitrogen loadings in sensitive estuaries. Deficiencies in the economics literature often result in the inability to assign economic values even to those health and environmental outcomes that can be quantified. Although these general uncertainties in the underlying scientific and economics literatures (that can cause the valuations to be higher or lower) are discussed in detail in the economic analyses and its supporting documents and references, the key uncertainties that have a bearing on the results of the benefit-cost analysis of this proposed rule include the following:

- the exclusion of potentially significant benefit categories (such as health and ecological benefits of reductions in mercury emissions),
- errors in measurement and projection for variables such as population growth and baseline incidence rates,
- uncertainties in the estimation of future-year emissions inventories and air quality,
- variability in the estimated relationships of health and welfare effects to changes in pollutant concentrations,
- uncertainties in exposure estimation,
- uncertainties in the size of the effect estimates linking air pollution and health endpoints,
- uncertainties about relative toxicity of different components within the complex mixture, and
- uncertainties associated with the effect of potential future actions to limit emissions.

Despite these uncertainties, we believe the benefit-cost analysis provides a reasonable indication of the expected economic benefits of the proposed rulemaking in future years under a set of reasonable assumptions.

In addition, in valuing reductions in premature fatalities associated with PM, we used a value of $5.5 million per statistical life. This represents a central value consistent with a range of values from $1 to $10 million suggested by recent meta-analyses of the wage-risk value of statistical life (VSL) literature.

The benefits estimates generated for the Proposed IAQR are subject to a number of assumptions and uncertainties, which are discussed throughout the document. As Table 1-2 indicates, total benefits are driven primarily by the reduction in premature fatalities each year, which account for over 90 percent of total benefits. For example, key assumptions underlying the primary estimate for the mortality category include the following:

(1) Inhalation of fine particles is causally associated with premature death at concentrations near those experienced by most Americans on a daily basis. Although biological mechanisms for this effect have not yet been definitively established, the weight of the available epidemiological evidence supports an assumption of causality.

(2) All fine particles, regardless of their chemical composition, are equally potent in causing premature mortality. This is an important assumption, because PM produced via transported precursors emitted from EGUs may differ significantly from direct PM released from automotive engines and other industrial sources, but no clear scientific grounds exist for supporting differential effects estimates by particle type.

(3) The C-R function for fine particles is approximately linear within the range of ambient concentrations under consideration. Thus, the estimates include health benefits from reducing fine particles in areas with varied concentrations of PM, including both regions that are in attainment with fine particle standard and those that do not meet the standard.

Although recognizing the difficulties, assumptions, and inherent uncertainties in the overall enterprise, these analyses are based on peer-reviewed scientific literature and up-to-date assessment tools, and we believe the results are highly useful in assessing this proposal.

We were unable to quantify or monetize a number of health and environmental effects. A full appreciation of the overall economic consequences of the proposed rule requires consideration of all benefits and costs expected to result from the proposed rule, not just those benefits and costs that could be expressed here in dollar terms. A listing of the benefit categories that could not be quantified or monetized in our estimate is provided in Table 1-4. These effects are denoted by "B" in Table 1-3 above and are additive to the estimates of benefits.

We are unable to quantify changes in levels of methylmercury contamination in fish associated with reductions in mercury emissions for this proposal. However, this proposal is anticipated to decrease annual EGU mercury emissions nationwide by 10.6 tons in 2010 or approximately 23.5 percent, by 11.8 tons in 2015 or 26.3 percent, and by 14.3 tons or 32 percent in 2020. Emission reduction percentage decreases are based upon expected mercury emissions changes from fossil-fired EGUs larger than 25 megawatt capacity. In a separate action, EPA is proposing to regulate mercury and nickel from certain types of electric generating units using the maximum achievable control technology (MACT) provisions of section 112 of the CAA or, in the alternative, using the performance standards provisions under section 111 of the CAA. This proposal will have implications for mercury reductions, and potential interactions may exist between the rulemakings. Information concerning potential interactions in the two rulemakings is discussed in the notice for proposed rulemaking for the CAA Section 112 proposal and in the document *Benefit Analysis of the CAA Section 112 Proposal to Reduce Mercury Emissions* included in the docket for the rulemaking.

SECTION 2

INTRODUCTION AND BACKGROUND

For this rulemaking, the EPA has assessed the role that transported emissions from upwind States play in contributing to unhealthy levels of $PM_{2.5}$ and 8-hour ozone in downwind States. Based on this assessment, the EPA is proposing emissions reduction requirements that would apply to upwind States under the Clean Air Act. This report assesses the health and welfare benefits of the proposed rule. This document presents the health and welfare benefits of the IAQR and compares the benefits of this proposal to the estimated costs of implementing the rule in 2010 and 2015. Significant health and welfare benefits are likely to occur as a result of this rule, and these benefits are enumerated in this document. This chapter contains background information relative to the rule and an outline of the chapters of the report.

2.1 Background

Congress recognized that interstate pollution transport from upwind States can contribute to unhealthy pollution levels in downwind States. Therefore, the CAA contains provisions in section 110(a)(2)(D) that require upwind States to eliminate emissions that contribute significantly to nonattainment downwind. Under section 110(a)(2) States are required to submit plans to the EPA within 3 years of issuance of a revised National Ambient Air Quality Standard. Among other requirements, these plans are required to prohibit emissions in the State that contribute significantly to nonattainment downwind.

The EPA's proposal finds that 29 States and the District of Columbia contribute significantly to nonattainment, or interfere with maintenance, of the NAAQS for $PM_{2.5}$ and/or 8-hour ozone in downwind States. The EPA is proposing to require these upwind States to revise their State Implementation Plans to include control measures to reduce emissions of SO_2 and/or NO_x. SO_2 is a precursor to $PM_{2.5}$ formation, and NO_x is a precursor to both ozone and $PM_{2.5}$ formation. Reducing upwind precursor emissions will assist the downwind $PM_{2.5}$ and 8-hour ozone nonattainment areas in achieving the NAAQS. Moreover, attainment would be achieved in a more equitable, cost-effective manner than if each nonattainment area attempted to achieve attainment by implementing local emissions reductions alone. The

relevant regions for PM2.5 and ozone significant contribution are depicted in Figures 2-1 and 2-2, respectively.

2.2 Regulated Entities

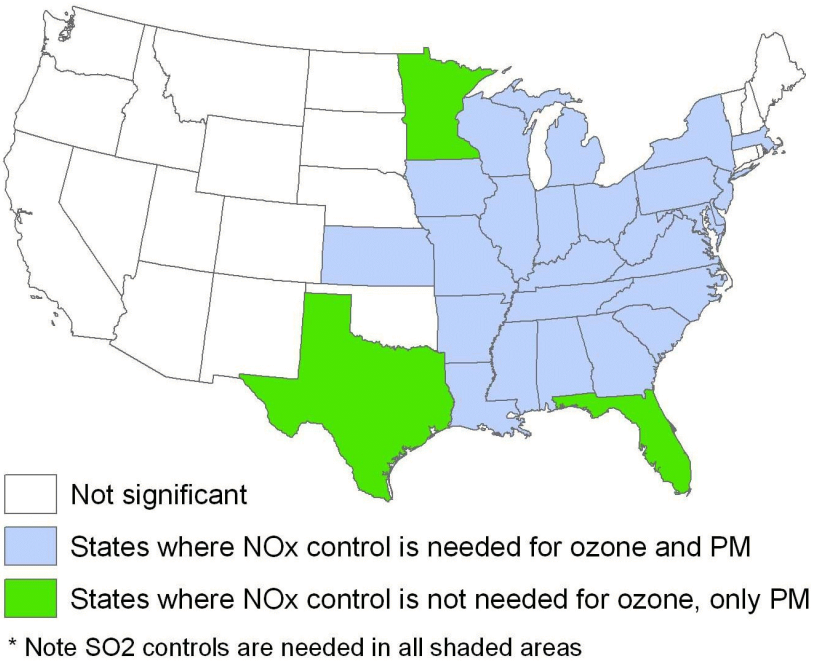
This action does not propose to directly regulate emissions sources. Instead, it proposes to require States to revise their SIPs to include control measures to reduce emissions of NO_x and SO_2. The proposed emission reduction requirements that would be assigned to the States are based on controls that are known to be highly cost effective for electric generation units (EGUs). However, States would have the flexibility to choose what sources to control. While the EPA is soliciting comments on the potential for pollution control from other sources, the analysis conducted assumes controls for EGUs only.

2.3 Control Scenario

The analysis conducted assumes that a cap-and-trade program will be used to achieve the level of emission control requirements desired. The EPA would establish regional emission budget determinations for SO_2 and NO_x to address the transport problem. In this proposal, these requirements would effectively establish emission caps in 2010 for SO_2 and NO_x of 3.9 million tons and 1.6 million tons, respectively. These budgets would be lowered in 2015 to provide SO_2 and NO_x emission caps of 2.7 million tons and 1.3 million tons, respectively in the proposed control region. These quantities were derived by calculating the amount of emissions of SO_2 and NO_x that the EPA believes can be controlled from large EGUs in a highly cost-effective manner. When fully implemented, this would result in nationwide SO_2 emissions of approximately 3.5 million tons. This is significantly lower than the 8.95 million tons of SO_2 emissions allowed under the current Title IV Acid Rain SO_2 Trading Program. The EPA expects that States will elect to join a regional cap-and-trade program for these pollutants.

2.4 Cost of Emission Controls

The EPA analyzed the costs of IAQR using the Integrated Planning Model (IPM). The EPA has used the IPM to analyze the impacts of regulations on the power sector. A description of the methodology used to model the costs and economic impacts to the power sector may be obtained in "Preliminary Analysis of the Costs of the Inter-State Air Quality Rule" January 2004. It is estimated that the annual cost of implementing this proposal in 2010 is $2.9 billion and in 2015 is $3.7 billion in the transport region (1999$).



States where NO_x control is not needed for ozone, only PM.

States where NO_x control is needed for ozone and PM

Figure 2-1. States Identified as Having Significant Contribution to PM2.5

2.5 Organization of this Report

This document describes the health and welfare benefits of the proposed rule. The document is organized as follows:

- Chapter 3, Emissions and Air Quality Impacts, describes emission inventories and air quality modeling that are essential inputs into the benefits assessment.

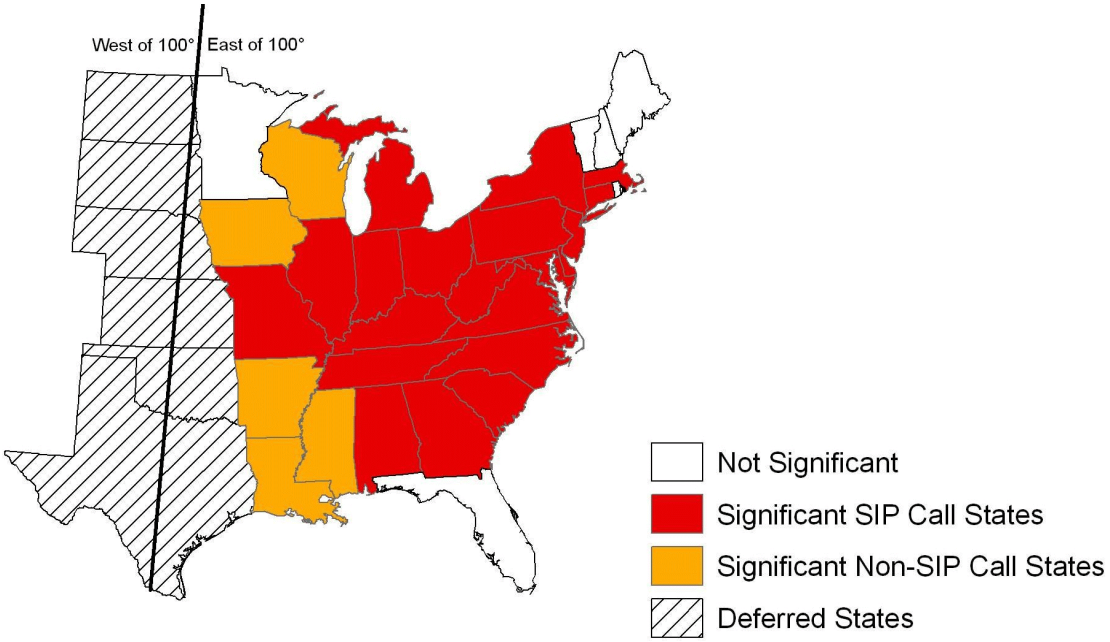


Figure 2-2. States Identified as Having Significant Contribution to Ozone

- Chapter 4, Benefits Analysis and Results, describes the methodology and results of the benefits analysis.
- Chapter 5, Qualitative Assessment of Nonmonetized Benefits, describes benefits that are not monetized for this rulemaking.
- Chapter 6, Comparison of Benefits and Costs, provides a comparison of the monetized benefits and estimated annual costs of the proposed control scenario.

SECTION 3

EMISSIONS AND AIR QUALITY IMPACTS

This chapter summarizes the emissions inventories and air quality modeling that serve as the inputs to the benefits analysis of this proposed rule as detailed in Chapter 4. In summary, given baseline and post-control emissions inventories for the emission species expected to impact ambient air quality, we use sophisticated photochemical air quality models to estimate baseline and post-control ambient concentrations of ozone and PM and deposition of nitrogen and sulfur for each year. The estimated changes in ambient concentrations are then combined with monitoring data to estimate population level exposures to changes in ambient concentrations for use in estimating health effects. Modeled changes in ambient data are also used to estimate changes in visibility and changes in other air quality statistics that are necessary to estimate welfare effects.

The initial section of this chapter provides a summary of the baseline emissions inventories and the emissions reductions that were modeled for this rule. The next section provides a summary of the methods for and results of estimating air quality for the 2010 and 2015 base cases and control scenarios for the purposes of the benefit analysis. There are separate sections for PM, ozone, and visibility.

3.1 Emissions Inventories and Estimated Emissions Reductions

The technical support document for emissions inventories discusses the development of the 2001, 2010 and 2015 baseline emissions inventories for the benefits analysis of this proposed rule. The emission sources and the basis for current and future-year inventories are listed in Table 3-1. Tables 3-2 and 3-3 summarize the baseline emissions of NO_X and SO_2 and the change in the emissions from EGUs that were used in modeling air quality changes.

Table 3-1. Emissions Sources and Basis for Current and Future-Year Inventories

Emissions Source	2001 Base Year	Future-Year Base Case Projections
Utilities	2001 CEM data	Integrated Planning Model (IPM)
Non-Utility Point and Area sources	Straight-line projections from 1996 NEI Version 3.12 (point) Version 3.11 (area)	BEA growth projections
Highway vehicles	MOBILE5b model with MOBILE6 adjustment factors for VOC and NOX; PART5 model for PM	VMT projection data
Nonroad engines (except locomotives, commercial marine vessels, and aircraft)	NONROAD2002 model	BEA and Nonroad equipment growth projections

Note: Full description of data, models, and methods applied for emissions inventory development and modeling are provided in Emissions Inventory TSD (EPA, 2003a).

3.2 Air Quality Impacts

This section summarizes the methods for and results of estimating air quality for the 2010 and 2015 base cases and control scenarios for the purposes of the benefit analysis. EPA has focused on the health, welfare, and ecological effects that have been linked to air quality changes. These air quality changes include the following:

1. Ambient particulate matter (PM_{10} and $PM_{2.5}$)–as estimated using a national-scale version of the REgional Modeling System for Aerosols and Deposition (REMSAD);

2. Ambient ozone–as estimated using regional-scale applications of the Comprehensive Air Quality Model with Extensions (CAMx); and

3. Visibility degradation (i.e., regional haze), as developed using empirical estimates of light extinction coefficients and efficiencies in combination with REMSAD modeled reductions in pollutant concentrations.

Table 3-2. Summary of Modeled Baseline Emissions for Lower 48 States

	Pollutant Emissions (tons)	
Source	**NO_X**	**SO_2**
2001 Baseline		
EGUs	4,824,967	10,714,558
Non-EGUs	3,180,835	3,696,048
Area	2,220,728	1,379,810
Mobile	8,694,038	261,526
Nonroad	4,059,278	531,203
Total, All Sources	22,979,846	16,583,145
2010 Base Case		
EGUs	3,943,438	9,856,926
Non-EGUs	3,228,201	3,799,163
Area	2,225,898	1,367,643
Mobile	4,931,947	29,790
Nonroad	3,404,962	236,446
Total, All Sources	17,734,447	15,289,969
2015 Base Case		
EGUs	4,008,241	9,222,097
Non-EGUs	3,307,415	3,893,813
Area	2,235,712	1,369,925
Mobile	3,458,279	32,551
Nonroad	2,903,048	232,644
Total, All Sources	15,912,695	14,751,030

Table 3-3. Summary of Modeled Emissions Changes for the Proposed Interstate Air Quality Rule: 2010 and 2015

Item	Pollutant	
	NO_X	SO_2
2010 Emission Changes[a]		
Absolute Tons	1,373,919	3,750,219
Percentage of EGU Emissions	34.8%	38.1%
Percentage of All Manmade Emissions	7.8%	24.5%
2015 Emission Changes[a]		
Absolute Tons	1,704,065	3,820,393
Percentage of EGU Emissions	42.5%	41.4%
Percentage of All Manmade Emissions	10.7%	25.9%

[a] Note that the emission changes only occur within the affected transport region; however, the percent reductions reflect the change as a share of baseline emissions for the lower 48 states as presented in Table 3-2.

The air quality estimates in this section are based on the emission changes summarized in the preceding section. These air quality results are in turn associated with human populations and ecosystems to estimate changes in health and welfare effects. In Section 3.2.1, we describe the estimation of PM air quality using REMSAD, and in Section 3.2.2, we cover the estimation of ozone air quality using CAMx. Lastly, in Section 3.2.3, we discuss the estimation of visibility degradation.

3.2.1 PM Air Quality Estimates

We use the emissions inputs summarized above with a national-scale version of the REgional Model System for Aerosols and Deposition (REMSAD) to estimate PM air quality in the contiguous U.S. REMSAD is a three-dimensional grid-based Eulerian air quality model designed to estimate annual particulate concentrations and deposition over large spatial scales (e.g., over the contiguous U.S.). Consideration of the different processes that affect primary (directly emitted) and secondary (formed by atmospheric processes) PM at the

regional scale in different locations is fundamental to understanding and assessing the effects of proposed pollution control measures that affect ozone, PM and deposition of pollutants to the surface.[1] Because it accounts for spatial and temporal variations as well as differences in the reactivity of emissions, REMSAD is useful for evaluating the impacts of the proposed rule on U.S. PM concentrations.

REMSAD was peer-reviewed in 1999 for EPA as reported in *"Scientific Peer-Review of the Regulatory Modeling System for Aerosols and Deposition*" (Seigneur et al., 1999). Earlier versions of REMSAD have been employed for the EPA's Prospective 812 Report to Congress, EPA's Heavy Duty (HD) Engine/Diesel Fuel rule, and EPA's air quality assessment of the Clear Skies Initiative. Version 7.06 of REMSAD was employed for this analysis and is fully described in the air quality modeling technical support document (EPA, 2003b). This version reflects updates in the following areas to improve performance and address comments from the 1999 peer-review:

1. Gas phase chemistry updates to "micro-CB4" mechanism including new treatment for the NO_3 and N_2O_5 species and the addition of several reactions to better account for the wide ranges in temperature, pressure, and concentrations that are encountered for regional and national applications.

2. PM chemistry updates to calculate particulate nitrate concentrations through use of the MARS-A equilibrium algorithm and internal calculation of secondary organic aerosols from both biogenic (terpene) and anthropogenic (estimated aromatic) VOC emissions.

3. Aqueous phase chemistry updates to incorporate the oxidation of SO_2 by O3 and O2 and to include the cloud and rain liquid water content from MM5 meteorological data directly in sulfate production and deposition calculations.

4. Calculation of the production of secondary organic aerosols (SOA) due to atmospheric chemistry processes has been added for both anthropogenic and biogenic organics.

[1]Given the focus of this rule on secondarily formed particles it is important to employ a Eulerian model such as REMSAD. The impact of secondarily formed pollutants typically involves primary precursor emissions from a multitude of widely dispersed sources, and chemical and physical processes of pollutants are best addressed using an air quality model that employs an Eulerian grid model design.

As discussed in the Air Quality Modeling TSD, the model tends to underestimate observed $PM_{2.5}$ concentrations nationwide.

Our analysis applies the modeling system to the entire U.S. for the six emissions scenarios: a 1996 baseline year for performance evaluation, a 2001 baseline projection, a 2010 baseline projection and a 2010 projection with controls, a 2015 baseline projection and a 2015 projection with controls. REMSAD simulates every hour of every day of the year and, thus, requires a variety of input files that contain information pertaining to the modeling domain and simulation period. These include gridded, 1-hour average emissions estimates and meteorological fields, initial and boundary conditions, and land-use information. As applied to the contiguous U.S., the model segments the area within the region into square blocks called grids (roughly equal in size to counties), each of which has several layers of air conditions. Using this data, REMSAD generates predictions of 1-hour average PM concentrations for every grid. As discussed in the Air Quality Modeling TSD, we use the relative predictions from the model by combining the 2001 base-year and each future-year scenario with speciated ambient air quality observations to determine the expected change in 2010 or 2015 concentrations due to the rule. After completing this process, we then calculated daily and seasonal PM air quality metrics as inputs to the health and welfare C-R functions of the benefits analysis. The following sections provide a more detailed discussion of each of the steps in this evaluation and a summary of the results.

3.2.1.1 Modeling Domain

The PM air quality analyses employed the modeling domain used previously in support of Clear Skies air quality assessment. As shown in Figure 3-1, the modeling domain encompasses the lower 48 States and extends from 126 degrees to 66 degrees west longitude and from 24 degrees north latitude to 52 degrees north latitude. The model contains horizontal grid-cells across the model domain of roughly 36 km by 36 km. There are 12 vertical layers of atmospheric conditions with the top of the modeling domain at 16,200 meters. The 36 by 36 km horizontal grid results in a 120 by 84 grid (or 10,080 grid-cells) for each vertical layer. Figure 3-2 illustrates the horizontal grid-cells for Maryland and surrounding areas.

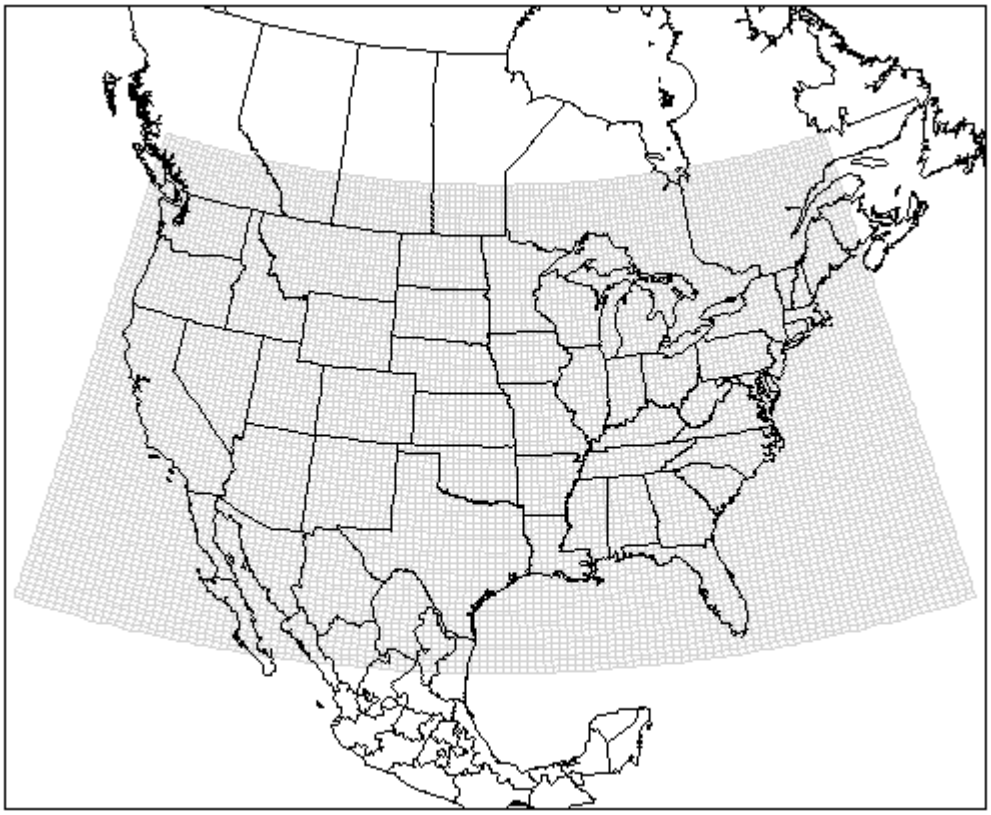


Figure 3-1. REMSAD Modeling Domain for Continental United States

Note: Gray markings define individual grid-cells in the REMSAD model.

3.2.1.2 Simulation Periods

For use in this benefits analysis, the simulation periods modeled by REMSAD included separate full-year application for each of the six emissions scenarios, i.e., 1996 and 2001 baseline years and the 2010 and 2015 base cases and control scenarios.

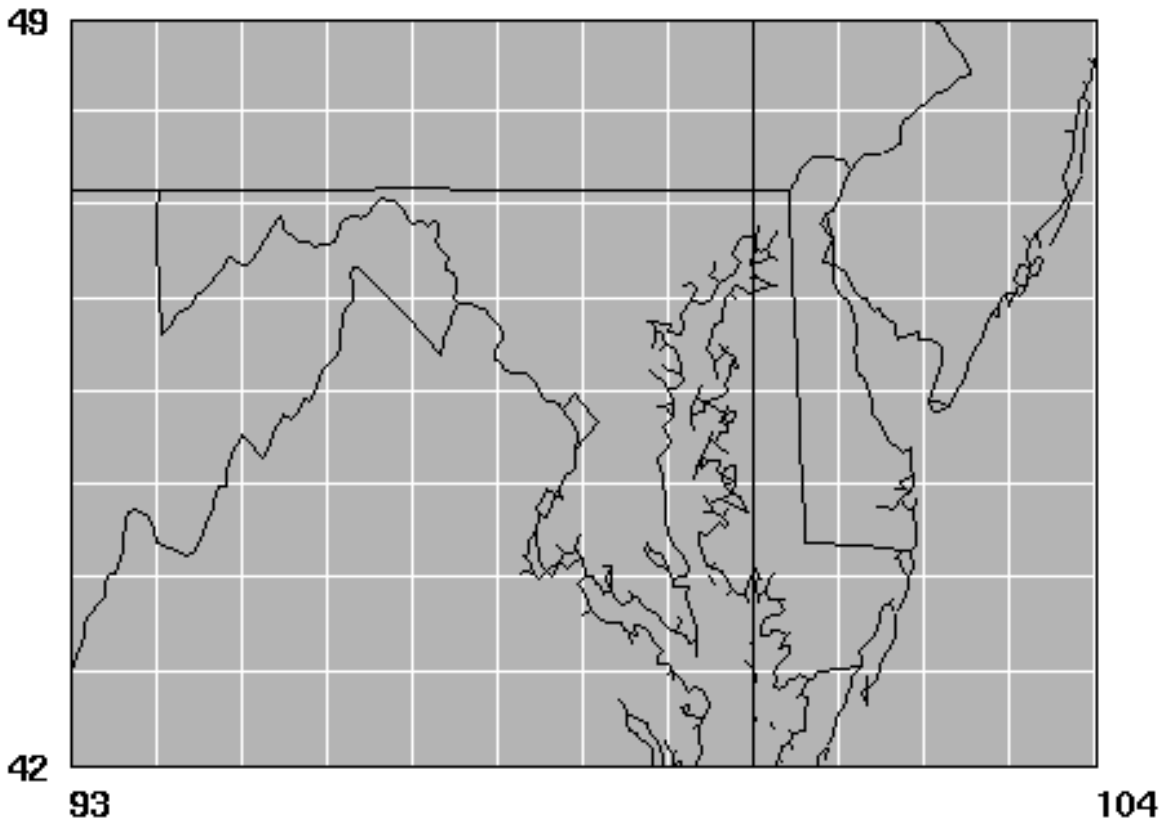


Figure 3-2. Example of REMSAD 36 x 36km Grid-cells for Maryland Area

3.2.1.3 Model Inputs

REMSAD requires a variety of input files that contain information pertaining to the modeling domain and simulation period. These include gridded, 1-hour average emissions estimates and meteorological fields, initial and boundary conditions, and land-use information. Separate emissions inventories were prepared for the 1996 and 2001 baseline years and each of the future-year base cases and control scenarios. All other inputs were specified for the 1996 baseline model application and remained unchanged for each future-year modeling scenario.

REMSAD requires detailed emissions inventories containing temporally allocated emissions for each grid-cell in the modeling domain for each species being simulated. The previously described annual emission inventories were preprocessed into model-ready inputs

through the SMOKE emissions preprocessing system. Details of the preprocessing of emissions through SMOKE as provided in the emissions inventory TSD. Meteorological inputs reflecting 1996 conditions across the contiguous U.S. were derived from Version 5 of the Mesoscale Model (MM5). These inputs included horizontal wind components (i.e., speed and direction), temperature, moisture, vertical diffusion rates, and rainfall rates for each grid cell in each vertical layer. Details of the annual 1996 MM5 modeling are provided in Olerud (2000).

A postprocessor called MM5REMSAD was developed to convert the MM5 data into the appropriate REMSAD grid coordinate systems and file formats. This postprocessor was used to develop the hourly average meteorological input files from the MM5 output. Documentation of the MM5REMSAD code and further details on the development of the input files are contained in Mansell (2000). A more detailed description of the development of the meteorological input data is provided in the Air Quality TSD, which is located in the docket for this rule.

The modeling specified initial species concentrations and lateral boundary conditions to approximate background concentrations of the species; for the lateral boundaries the concentrations varied (decreased parabolically) with height. These initial conditions reflect relatively clean background concentration values. Terrain elevations and land use information was obtained from the U.S. Geological Survey database at 10 km resolution and aggregated to the roughly 36 km horizontal resolution used for this REMSAD application. The development of model inputs is discussed in greater detail in the Air Quality TSD, which is available in the docket for this rule.

3.2.1.4 Model Performance for Particulate Matter (PM)

The purpose of the base year PM air quality modeling was to reproduce the atmospheric processes resulting in formation and dispersion of fine particulate matter across the U.S. An operational model performance evaluation for $PM_{2.5}$ and its related speciated components (e.g., sulfate, nitrate, elemental carbon etc.) for 1996 was performed in order to estimate the ability of the modeling system to replicate base year concentrations.

This evaluation is comprised principally of statistical assessments of model versus observed pairs. The robustness of any evaluation is directly proportional to the amount and quality of the ambient data available for comparison. Unfortunately, there are few $PM_{2.5}$ monitoring networks with available data for evaluation of the PM modeling. Critical limitations of the 1996 databases are a lack of urban monitoring sites with speciated

measurements and poor geographic representation of ambient concentration in the Eastern U.S.

The largest available ambient database for 1996 comes from the **I**nteragency **M**onitoring of **PRO**tected **V**isual **E**nvironments (IMPROVE) network. IMPROVE is a cooperative visibility monitoring effort between EPA, federal land management agencies, and state air agencies. Data is collected at Class I areas across the United States mostly at National Parks, National Wilderness Areas, and other protected pristine areas (IMPROVE 2000). There were approximately 60 IMPROVE sites that had complete annual $PM_{2.5}$ mass and/or $PM_{2.5}$ species data for 1996. Using the 100^{th} meridian to divide the eastern and western U.S., 42 sites were located in the West and 18 sites were in the East.

As presented in Table 3-4, the observed IMPROVE data used for the performance evaluation consisted of $PM_{2.5}$ total mass, sulfate ion, nitrate ion, elemental carbon, organic aerosols, and crustal material (soils). The REMSAD model output species were postprocessed in order to achieve compatibility with the observation species. The principal evaluation statistic used to evaluate REMSAD performance is the "ratio of the means." It is defined as the ratio of the average predicted values over the average observed values. The annual average ratio of the means was calculated for five individual PM2.5 species as well as for total PM2.5 mass. The metrics were calculated for all IMPROVE sites across the country as well as for the East and West individually. The following table shows the ratio of the annual means. Numbers greater than 1 indicate overpredictions compared to ambient observations (e.g., 1.23 is a 23 percent overprediction). Numbers less than 1 indicate underpredictions.

When considering annual average statistics (e.g., predicted versus observed), which are computed and aggregated over all sites and all days, REMSAD underpredicted fine particulate mass ($PM_{2.5}$), by 18 percent. $PM_{2.5}$ in the Eastern U.S. was underpredicted by 2 percent, while $PM_{2.5}$ in the West was underpredicted by 33 percent. All $PM_{2.5}$ component species were underpredicted in the west. In the East, nitrate and crustal material are overestimated. Elemental carbon shows neither over or underprediction in the east with a bias near 0 percent. Eastern sulfate is slightly underpredicted with a bias of 12 percent. Organic aerosols show little or no bias in the East and West.

Given the state of the science relative to PM modeling, it is inappropriate to judge PM model performance using criteria derived for other pollutants, like ozone. Still, the performance of the IAQR PM modeling is very encouraging, especially considering that the

Table 3-4. Model Performance Statistics for REMSAD $PM_{2.5}$ Species Predictions: 1996

	Ratio of the Means (annual average concentrations)		
IMPROVE PM Species	**Nationwide**	**Eastern U.S.**	**Western U.S.**
PM2.5, total mass	0.82	0.98	0.67
Sulfate ion	0.79	0.88	0.59
Nitrate ion	1.55	2.66	0.69
Elemental carbon	0.86	1.01	0.71
Organic aerosols	1.00	1.04	0.97
Soil/Other	1.33	3.08	0.81

Note: The dividing line between the West and East was defined as the 100th meridian.

results may be limited by our current knowledge of PM science and chemistry, by the emissions inventories for primary PM and secondary PM precursor pollutants, by the relatively sparse ambient data available for comparisons to model output, and by uncertainties in monitoring techniques. The model performance for sulfate is quite reasonable, which is key to the analysis due to the importance of SO_2 emissions reductions in the IAQR control strategy. Additional details, including comparisons to other monitoring networks, can be found in the Air Quality Modeling TSD.

3.2.1.5 Converting REMSAD Outputs to Benefits Inputs

REMSAD generates predictions of hourly PM concentrations for every grid. The particulate matter species modeled by REMSAD include a primary coarse fraction (corresponding to PM in the 2.5 to 10 micron size range), a primary fine fraction (corresponding to PM less than 2.5 microns in diameter), and several secondary particles (e.g., sulfates, nitrates, and organics). $PM_{2.5}$ is calculated as the sum of the primary fine fraction and all of the secondarily-formed particles. Future-year estimates of $PM_{2.5}$ were calculated using relative reduction factors (RRFs) applied to 2000-2002 $PM_{2.5}$ design values (EPA, 2003b). The procedures for determining the RRFs are similar to those in EPA's draft guidance for modeling the $PM_{2.5}$ standard (EPA, 1999a). The guidance recommends that model predictions be used in a relative sense to estimate changes expected to occur in each

major $PM_{2.5}$ species. These species are sulfate, nitrate, organic carbon, elemental carbon, crustal and un-attributed mass which is defined as the difference between measured $PM_{2.5}$ and the sum of the other five components. The procedure for calculating future year $PM_{2.5}$ design values is called the "Speciated Modeled Attainment Test (SMAT)". EPA previously used this procedure to estimate the ambient impact of the Clear Skies Act emissions controls.

The SMAT procedure was performed using the base year 2001 scenario and each of the future-year scenarios. The SMAT approach uses temporally scaled speciated PM2.5 monitor data from 2001-2002, reconstructed into total PM2.5 mass based on 2000-2002 design values, and kriged to 12 kilometer grids (nested within the standard 36 km REMSAD grid structure). Temporal scaling is based on ratios of future modeled REMSAD data to 2001 REMSAD model data, using REMSAD modeling conducted at the 36 km grid resolution. SMAT output files include both quarterly mean and annual mean PM2.5 mass results, which are then manipulated within SAS to produce a BenMAP input file containing 364 daily values (created by replicating the quarterly mean values for each day of the appropriate season).

3.2.1.6 PM Air Quality Results

Table 3-5 provides a summary of the predicted ambient $PM_{2.5}$ concentrations for the 2010 and 2015 base cases and changes associated with proposed rule. The REMSAD results indicate that the predicted change in PM concentrations is composed almost entirely of reductions in fine particulates ($PM_{2.5}$) with little or no reduction in coarse particles (PM_{10} less $PM_{2.5}$). Therefore, the observed changes in PM_{10} are composed primarily of changes in $PM_{2.5}$. In addition to the standard frequency statistics (e.g., minimum, maximum, average), we provide the population-weighted average which better reflects the baseline levels and predicted changes for more populated areas of the nation. This measure, therefore, better reflects the potential benefits of these predicted changes through exposure changes to these populations. As shown, the average annual mean concentrations of $PM_{2.5}$ across populated eastern U.S. grid-cells declines by roughly 5.6 percent (or 0.6 $\mu g/m^3$) and 7.5 percent (or 0.8 $\mu g/m^3$) in 2010 and 2015, respectively. The population-weighted average mean concentration declined by 6.1 percent (or 0.74 $\mu g/m^3$) in 2010 and 7.9 percent (or 0.94 $\mu g/m^3$) in 2015, which is much larger in absolute terms than the spatial average for both years. This indicates the proposed rule generates greater absolute air quality improvements in more populated, urban areas.

Table 3-5. Summary of Base Case PM Air Quality and Changes Due to Proposed Interstate Air Quality Rule: 2010 and 2015

	2010			2015		
Statistic	Base Case	Change[a]	Percent Change	Base Case	Change[a]	Percent Change
$PM_{2.5}$ ($\mu g/m^3$)						
Minimum Annual Mean	5.24	-0.33	-6.3%	5.13	-0.33	-6.4%
Maximum Annual Mean	16.88	-0.86	-5.1%	16.79	-1.19	-7.1%
Average Annual Mean	10.82	-0.61	-5.6%	10.67	-0.80	-7.5%
Pop-Weighted Average Annual Mean [b]	12.19	-0.74	-6.1%	11.99	-0.94	-7.9%

[a] The change is defined as the control case value minus the base case value.

[b] Calculated by summing the product of the projected REMSAD grid-cell population and the estimated PM concentration, for that grid-cell and then dividing by the total population.

Table 3-6 provides information on the populations in 2010 and 2015 that will experience improved PM air quality. There are significant populations that live in areas with meaningful reductions in annual mean $PM_{2.5}$ concentrations resulting from the proposed rule. As shown, in 2015, almost 40 percent of the U.S. population located in the eastern 37 state modeling domain are predicted to experience reductions of greater than 1.0 $\mu g/m^3$. This is an increase from the 20 percent of the U.S. population that are expected to experience such reductions in 2010. Furthermore, over 7 percent of this population will benefit from reductions in annual mean $PM_{2.5}$ concentrations of greater than 1.5 $\mu g/m^3$ and almost 2 percent will live in areas with reductions of greater than 1.75 $\mu g/m^3$.

3.2.2 Ozone Air Quality Estimates

We use the emissions inputs summarized earlier in this chapter with a regional-scale version of CAMx to estimate ozone air quality in the Eastern and Western U.S. CAMx is an Eulerian three-dimensional photochemical grid air quality model designed to calculate the concentrations of both inert and chemically reactive pollutants by simulating the physical and chemical processes in the atmosphere that affect ozone formation. Version 3.10 of the CAMx model was employed for these analyses. Because it accounts for spatial and temporal variations as well as differences in the reactivity of emissions, CAMx is useful for evaluating

the impacts of the proposed rule on U.S. ozone concentrations. Although the model tends to underestimate observed ozone, it exhibits less bias and error than any past regional ozone

Table 3-6. Distribution of PM2.5 Air Quality Improvements Over Population Due to Proposed Interstate Air Quality Rule: 2010 and 2015

Change in Annual Mean $PM_{2.5}$ Concentrations ($\mu g/m^3$)	2010 Population[b]		2015 Population	
	Number (millions)	Percent (%)	Number (millions)	Percent (%)
$0 > \Delta PM_{2.5}$ Conc ≤ 0.25	6.1	2.7%	0.0	0.0%
$0.25 > \Delta PM_{2.5}$ Conc ≤ 0.5	59.0	26.1%	29.9	12.8%
$0.5 > \Delta PM_{2.5}$ Conc ≤ 0.75	57.1	25.3%	52.9	22.6%
$0.75 > \Delta PM_{2.5}$ Conc ≤ 1.0	59.3	26.2%	60.6	25.9%
$1.0 > \Delta PM_{2.5}$ Conc ≤ 1.25	22.5	9.9%	34.6	14.8%
$1.25 > \Delta PM_{2.5}$ Conc ≤ 1.5	11.2	4.9%	38.0	16.2%
$1.5 > \Delta PM_{2.5}$ Conc ≤ 1.75	9.0	4.0%	13.9	5.9%
$\Delta PM_{2.5}$ Conc > 1.75	2.0	0.9%	4.2	1.8%

[a] The change is defined as the control case value minus the base case value.

[b] Population counts and percentages are for the fraction of the national population located in the eastern 37 state modeling domain considered in modeling health benefits for the rule.

modeling application conducted by EPA (i.e., OTAG, On-highway Tier-2, and HD Engine/Diesel Fuel).

Our analysis applies the modeling system separately to the Eastern U.S. for six emissions scenarios: a 1995 baseline projection, a 2001 baseline projection, a 2020 baseline projection and a 2020 projection with controls, a 2030 baseline projection and a 2030 projection with controls. The model was applied and evaluated over three episodes that occurred during the summer of 1995 base year. Subsequently, episodic ozone model runs were made for the 2001 base year scenario and the 2010 and 2015 base and control case scenarios for all episodes. Further discussion of this modeling, including evaluations of model performance relative to predicted future air quality, is provided in the air quality modeling TSD. As discussed in chapter 4, we use the relative predictions from the model by combining the 2001 base-year and each future-year scenario with current ambient air quality observations to determine the expected change in 2010 or 2015 ozone concentrations due to the rule (Abt Associates, 2003). These results are used solely in the benefits analysis.

The CAMx modeling system requires a variety of input files that contain information pertaining to the modeling domain and simulation period. These include gridded, day-specific emissions estimates and meteorological fields, initial and boundary conditions, and land-use information. As applied to the Eastern U.S., the model segments the area into square blocks called grids (roughly equal in size to counties), each of which has several layers of air conditions that are considered in the analysis. Using this data, the CAMx model generates predictions of hourly ozone concentrations for every grid. We then calibrate the results of this process to develop 2010 and 2015 ozone profiles at monitor sites by normalizing the observations to the observed ozone concentrations at each monitor site. For areas (grids) without ozone monitoring data, we interpolated ozone values using data from monitors surrounding the area. After completing this process, we calculated daily and seasonal ozone metrics to be used as inputs to the health and welfare C-R functions of the benefits analysis. The following sections provide a more detailed discussion of each of the steps in this evaluation and a summary of the results.

3.2.2.1 Modeling Domain

The modeling domain representing the Eastern U.S. is the same as that used previously for OTAG and the On-highway Tier-2 rulemaking. As shown in Figure 3-3, this domain encompasses most of the Eastern U.S. from the East coast to mid-Texas and consists of two grids with differing resolutions. The modeling domain extends from 99 degrees to 67 degrees west longitude and from 26 degrees to 47 degrees north latitude. The inner portion of the modeling domain shown in Figure 3-3 uses a relatively fine grid of 12 km consisting of nine vertical layers. The outer area has less horizontal resolution, as it uses a 36 km grid with the same nine vertical layers. The vertical height of the modeling domain is 4,000 meters above ground level for both areas.

3.2.2.2 Simulation Periods

For use in this benefits analysis, the simulation periods modeled by CAMx included several multi-day periods when ambient measurements recorded high ozone concentrations. A simulation period, or episode, consists of meteorological data characterized over a block of days that are used as inputs to the air quality model. A simulation period is selected to characterize a variety of ozone conditions including some days with high ozone concentrations in one or more portions of the U.S. and observed exceedances of the 1-hour NAAQS for ozone being recorded at monitors. We focused on the summer of 1995 for selecting the episodes to model because it is a recent time period for which we had model-

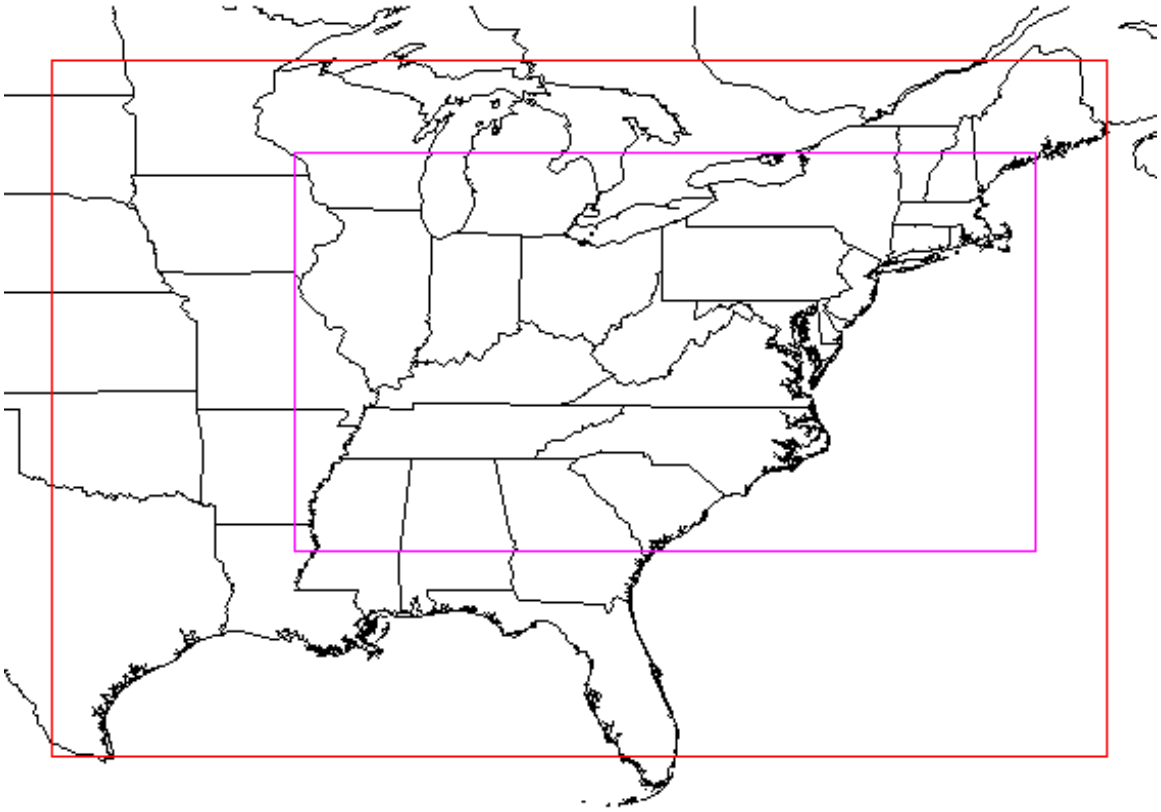


Figure 3-3. CAMx Eastern U.S. Modeling Domain

Note: The inner area represents fine grid modeling at 12 km resolution, while the outer area represents the coarse grid modeling at 36 km resolution.

ready meteorological inputs and this timeframe contained several periods of elevated ozone over the Eastern U.S. As detailed in the air quality modeling TSD, this analysis used three multi-day meteorological scenarios during the summer of 1995 for the model simulations over the eastern U.S.: June 12-24, July 5-15, and August 7-21. Each of the six emissions scenarios (1995 base year, 2001 base year, 2010 base and control, 2015 base and control) were simulated for the selected episodes. These episodes include a three day "ramp-up" period to initialize the model, but the results for these days are not used in this analysis.

3.2.2.3 Non-emissions Modeling Inputs

The meteorological data required for input into CAMx (wind, temperature, vertical mixing, etc.) were developed by separate meteorological models. The gridded

meteorological data for the three historical 1995 episodes were developed using the Regional Atmospheric Modeling System (RAMS), version 3b. This model provided needed data at every grid cell on an hourly basis. These meteorological modeling results were evaluated against observed weather conditions before being input into CAMx and it was concluded that the model fields were adequate representations of the historical meteorology. A more detailed description of the settings and assorted input files employed in these applications is provided in the Air Quality TSD, which is located in the docket for this rule.

The modeling assumed background pollutant levels at the top and along the periphery of the domain as in Tier 2. Additionally, initial conditions were assumed to be relatively clean as well. Given the ramp-up days and the expansive domains, it is expected that these assumptions will not affect the modeling results, except in areas near the boundary (e.g., Dallas-Fort Worth TX). The other non-emission CAMx inputs (land use, photolysis rates, etc.) were developed using procedures employed in the Tier 2/OTAG regional modeling. The development of model inputs is discussed in greater detail in the Air Quality TSD, which is available in the docket for this rule.

3.2.2.4 Model Performance for Photochemical Ozone

The purpose of the 1995 base year photochemical ozone modeling was to reproduce the atmospheric processes resulting in the observed ozone concentrations over these domains and episodes. One of the fundamental assumptions in air quality modeling is that a model which adequately replicates observed pollutant concentrations in the base year can be used to assess the effects of future year emissions controls. A series of performance statistics was calculated for the Eastern U.S. domain as well as the four quadrants and multiple subregions. The model performance evaluation consisted solely of comparisons against ambient surface ozone data. There was insufficient data available in terms of ozone precursors or ozone aloft to allow for a more complete assessment of model performance. Three primary statistical metrics were used to assess the overall accuracy of the base year modeling simulations.

- Mean normalized bias is defined as the average difference between the hourly model predictions and observations (paired in space and time) at each monitoring location, normalized by the magnitude of the observations.
- Mean normalized gross error is defined as the average absolute difference between the hourly model predictions and observations (paired in space and time) at each monitoring location, normalized by the magnitude of the observations.

- Average accuracy of the peak is defined as the average difference between peak daily model predictions and observations at each monitoring location, normalized by the magnitude of the observations.

In general, the model tends to underestimate observed ozone. When all hourly observed ozone values greater than a 60 ppb threshold are compared to their model counterparts for the 30 episode modeling days in the eastern domain, the mean normalized bias is -1.1 percent and the mean normalized gross error is 20.5 percent. As shown in Table 3-7, the model generally underestimates observed ozone values for the June and July episodes, but predicts higher than observed amounts for the August episode.

Table 3-7. Model Performance Statistics for Hourly Ozone in the Eastern U.S. CAMx Ozone Simulations: 1995 Base Case

Episode	Average Accuracy of the Peak	Mean Normalized Bias	Mean Normalized Gross Error
June 1995	–7.3	–8.8	19.6
July 1995	–3.3	–5.0	19.1
August 1995	9.6	8.6	23.3

At present, there are no guidance criteria by which one can determine if a regional ozone modeling exercise is exhibiting adequate model performance. These base case simulations were determined to be acceptable based on comparisons to previously completed model rulemaking analyses (e.g., OTAG, Tier-2, and Heavy-Duty Engine). The modeling completed for this proposal exhibits less bias and error than any past regional ozone modeling application done by EPA. Thus, the model is considered appropriate for use in projecting changes in future year ozone concentrations and the resultant health/economic benefits due to the proposed emissions reductions.

In addition, the CAMx modeling results were also evaluated at a "local" level to ensure that areas determined to need the emissions reductions based on projected exceedances of the ozone standard were not unduly influenced by local overestimation of ozone in the model base year. As detailed in the Air Quality Modeling TSD, performance statistics were computed for each of 51 local subregions within the modeling domain. These performance statistics were compared to the recommended performance ranges for urban attainment modeling (EPA, 1991). The results indicate that model performance for the June episode was

within the recommended ranges for 69 percent of the local areas examined. For the July and August episodes, the percent of local areas with performance within the recommended ranges was 80 percent and 61 percent, respectively.

3.2.2.5 Converting CAMx Outputs to Full-Season Profiles for Benefits Analysis

This study extracted hourly, surface-layer ozone concentrations for each grid-cell from the standard CAMx output file containing hourly average ozone values. These model predictions are used in conjunction with the observed concentrations obtained from the Aerometric Information Retrieval System (AIRS) to generate ozone concentrations for the entire ozone season.[2,3] The predicted changes in ozone concentrations from the future-year base case to future-year control scenario serve as inputs to the health and welfare C-R functions of the benefits analysis, i.e., the Environmental Benefits Mapping and Analysis Program (BenMAP).

In order to estimate ozone-related health and welfare effects for the contiguous U.S., full-season ozone data are required for every BenMAP grid-cell. Given available ozone monitoring data, we generated full-season ozone profiles for each location in the contiguous 48 States in two steps: (1) we combine monitored observations and modeled ozone predictions to interpolate hourly ozone concentrations to a grid of 8 km by 8 km population grid-cells, and (2) we converted these full-season hourly ozone profiles to an ozone measure of interest, such as the daily average.[4,5] These methods are described in detail in the benefits analysis technical support document (Abt Associates, 2003).

3.2.2.6 Ozone Air Quality Results

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[2]The ozone season for this analysis is defined as the 5-month period from May to September; however, to estimate certain crop yield benefits, the modeling results were extended to include months outside the 5-month ozone season.

[3]Based on AIRS, there were 961 ozone monitors with sufficient data, i.e., 50 percent or more days reporting at least 9 hourly observations per day (8 am to 8 pm) during the ozone season.

[4]The 8 km grid squares contain the population data used in the health benefits analysis model, BenMAP. See Chapter 4 for a discussion of this model.

[5]This approach is a generalization of planar interpolation that is technically referred to as enhanced Voronoi Neighbor Averaging (EVNA) spatial interpolation (See Abt Associates (2003) for a more detailed description).

This section provides a summary the predicted ambient ozone concentrations from the CAMx model for the 2010 and 2015 base cases and changes associated with the proposed rule. Table 3-8 provides those ozone metrics for grid-cells in the Eastern U.S. that enter the concentration response functions for health benefits endpoints. The population-weighted average reflects the baseline levels and predicted changes for more populated areas of the

Table 3-8. Summary of CAMx Derived Population-Weighted Ozone Air Quality Metrics for Health Benefits Endpoints Due to Proposed Interstate Air Quality Rule: Eastern U.S.

	2010			2015		
Statistic [a]	Base Case	Change [b]	Percent Change [b]	Base Case	Change [b]	Percent Change [b]
Population-Weighted Average (ppb) [d]						
Daily 1-Hour Maximum Concentration	53.32	–0.51	–0.95%	52.10	–1.05	–2.02%
Daily 5-Hour Average Concentration	44.51	–0.42	–0.93%	43.65	–0.87	–2.00%
Daily 8-Hour Average Concentration	43.81	–0.41	–0.93%	42.97	–0.86	–1.99%
Daily 12-Hour Average Concentration	41.28	–0.38	–0.92%	40.56	–0.80	–1.98%
Daily 24-Hour Average Concentration	31.20	–0.28	–0.89%	30.83	–0.59	–1.91%

[a] These ozone metrics are calculated at the CAMX grid-cell level for use in health effects estimates based on the results of spatial and temporal Voronoi Neighbor Averaging. Except for the daily 24-hour average, these ozone metrics are calculated over relevant time periods during the daylight hours of the "ozone season," i.e., May through September. For the 5-hour average, the relevant time period is 10 am to 3 pm; for the 8-hr average, it is 9 am to 5 pm; and, for the 12-hr average it is 8 am to 8 pm.

[b] The change is defined as the control case value minus the base case value. The percent change is the "Change" divided by the "Base Case," and then multiplied by 100 to convert the value to a percentage.

[d] Calculated by summing the product of the projected CAMx grid-cell population and the estimated CAMx grid-cell seasonal ozone concentration, and then dividing by the total population.

nation. This measure, therefore, will better reflect the potential benefits of these predicted changes through exposure changes to these populations.

3.2.3 Visibility Degradation Estimates

Visibility degradation is often directly proportional to decreases in light transmittal in the atmosphere. Scattering and absorption by both gases and particles decrease light transmittance. To quantify changes in visibility, our analysis computes a light-extinction coefficient, based on the work of Sisler (1996), which shows the total fraction of light that is decreased per unit distance. This coefficient accounts for the scattering and absorption of light by both particles and gases, and accounts for the higher extinction efficiency of fine

particles compared to coarse particles. Fine particles with significant light-extinction efficiencies include sulfates, nitrates, organic carbon, elemental carbon (soot), and soil (Sisler, 1996).

Based upon the light-extinction coefficient, we also calculated a unitless visibility index, called a "deciview," which is used in the valuation of visibility. The deciview metric provides a scale for perceived visual changes over the entire range of conditions, from clear to hazy. Under many scenic conditions, the average person can generally perceive a change of one deciview. The higher the deciview value, the worse the visibility. Thus, an improvement in visibility is a decrease in deciview value.

Table 3-9. Distribution of Populations Experiencing Visibility Improvements due to Proposed Interstate Air Quality Rule: 2010 and 2015

Improvements in Visibility[a] (annual average deciviews)	2010 Population		2015 Population	
	Number (millions)	Percent (%)	Number (millions)	Percent (%)
0 > Δ Deciview ≤ 0.2	75.6	24.9%	74.8	23.6%
0.2 > Δ Deciview ≤ 0.4	24.1	7.9%	15.2	4.8%
0.4 > Δ Deciview ≤ 0.6	46.5	15.3%	25.3	8.0%
0.6 > Δ Deciview ≤ 0.8	87.7	28.8%	64.7	20.4%
0.8 > Δ Deciview ≤ 1.0	56.0	18.4%	57.8	18.2%
Δ Deciview > 1.0	14.3	4.7%	79.1	25.0%

Table 3-9 provides the distribution of visibility improvements across 2010 and 2015 populations resulting from this proposed rule. The majority of the 2015 U.S. population live in areas with predicted improvement in annual average visibility of greater than 0.6 deciviews resulting from the proposed rule. As shown, almost 72 percent of the 2015 U.S. population are predicted to experience improved annual average visibility of greater than 0.4 deciviews. Furthermore, roughly 25 percent of the 2015 U.S. population will benefit from reductions in annual average visibility of greater than 1 deciviews.

Because the visibility benefits analysis distinguishes between general regional visibility degradation and that particular to Federally-designated Class I areas (i.e., national parks, forests, recreation areas, wilderness areas, etc.), we separated estimates of visibility degradation into "residential" and "recreational" categories.[6] The estimates of visibility degradation for the "recreational" category apply to Federally-designated Class I areas, while estimates for the "residential" category apply to non-Class I areas. Deciview estimates are estimated using outputs from REMSAD for the 2010 and 2015 base cases and control scenarios.

3.2.3.1 Residential Visibility Improvements

Air quality modeling results predict that the proposed Interstate Air Quality Rule will create improvements in visibility through the country. In Table 3-10, we summarize residential visibility improvements across the Eastern U.S. in 2010 and 2015. The baseline annual average visibility for eastern U.S. counties is 21.61 deciviews in 2010. The mean improvement across eastern U.S. counties is 0.69 deciviews, or almost 3.2 percent. In urban areas with a population of 250,000 or more, the mean improvement in annual visibility was similar at 0.71 deciviews in 2010 and ranged from 0.17 to 1.64 deciviews. In rural areas, the mean improvement in visibility was 0.68 deciviews in 2010 and ranged from 0.19 to 1.69 deciviews.

[6] The visibility calculations presented in this section are changes in the annual average visibility for the purpose of generating monetized benefits. There improvements in visibility should not be confused with the requirements under the Regional Haze rule to show "reasonable progress" for the 20% best and 20% worst days to each Class I area. Example Regional Haze calculations for the 20% best and worst days are contained in the AQMTSD.

Table 3-10. Summary of Baseline Residential Visibility and Changes by Region: 2010 and 2015 (annual average deciviews)

	2010			2015		
Regions[a]	Base Case	Change[b]	Percent Change	Base Case	Change[b]	Percent Change
Eastern U.S.	21.61	0.69	3.17%	21.31	0.85	3.95%
Urban	22.78	0.71	3.14%	22.50	0.88	3.95%
Rural	21.14	0.68	3.18%	20.84	0.84	3.96%

[a] The dividing line between the Eastern and Western U.S. was defined as the 100th meridian.

[b] An improvement in visibility is a decrease in deciview value. The change is defined as the control case deciview level minus the base case deciview level.

3.2.3.2 Recreational Visibility Improvements

In Table 3-11, we summarize recreational visibility improvements in 2010 and 2015 in Federal Class I areas located in the eastern U.S. These recreational visibility regions are shown in Figure 3-4. As shown, the improvement in visibility for Federal Class I areas in the Eastern U.S. increases from 3.8 percent, or 0.77 deciviews, in 2010. The predicted absolute improvement of 0.94 deciviews in 2015 reflects a 4.6 percent change from 2015 baseline visibility of 20.38 deciviews.

Table 3-11. Summary of Baseline Recreational Visibility and Changes by Region: 2010 and 2015 (annual average deciviews)

	2010			2015		
Class I Visibility Regions[a]	Base Case	Change[b]	Percent Change	Base Case	Change[b]	Percent Change
Eastern U.S.	20.59	0.77	3.75%	20.38	0.94	4.61%
Southeast	22.04	0.91	4.11%	21.80	1.17	5.35%
Northeast/Midwest	19.28	0.65	3.38%	19.11	0.74	3.85%

[a] Regions are pictured in Figure VI-5 and are defined in the technical support document (see Abt Associates, 2003).

[b] An improvement in visibility is a decrease in deciview value. The change is defined as the control case deciview level minus the base case deciview level.

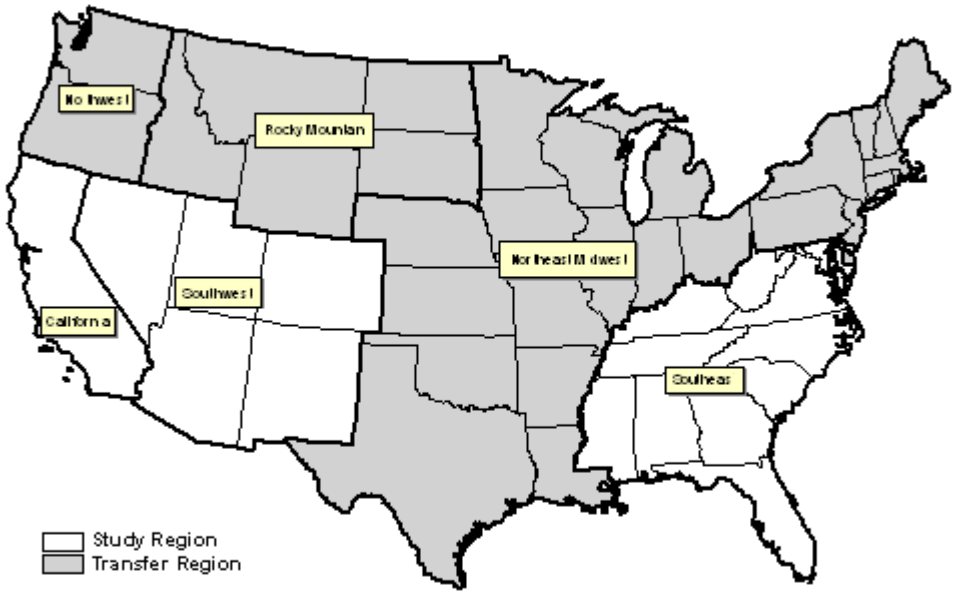


Figure 3-4. Recreational Visibility Regions for Continental U.S.

Note: Study regions were represented in the Chestnut and Rowe (1990a, 1990b) studies used in evaluating the benefits of visibility improvements, while transfer regions used extrapolated study results.

SECTION 4

BENEFITS ANALYSIS AND RESULTS

This chapter reports the EPA's analysis of a subset of the public health and welfare impacts and associated monetized benefits to society of the proposed IAQR. The EPA is required by Executive Order 12866 to estimate the benefits and costs of major new pollution control regulations. Accordingly, the analysis presented here attempts to answer three questions: 1) what are the physical health and welfare effects of changes in ambient air quality resulting from reductions in precursors to particulate matter (PM) including (NOx) and sulfur dioxide (SO_2) emissions? 2) how much are the changes in these effects attributable to the proposed rule worth to U.S. citizens as a whole in monetary terms? and 3) how do the monetized benefits compare to the costs? It constitutes one part of the EPA's thorough examination of the relative merits of this proposed regulation.

The analysis presented in this chapter uses a methodology generally consistent with benefits analyses performed for the recent analysis of Nonroad Diesel Engines Tier 4 Standards and the proposed Clear Skies Act of 2003 (EPA, 2003). The benefits analysis relies on three major modeling components:

1) Calculation of the impact that a set of preliminary emissions standards for EGUs based on a state-level cap and trade program would have on the national inventory of precursors to PM including SO_2 and NOx.

2) Air quality modeling for 2010 and 2015 to determine changes in ambient concentrations of ozone and particulate matter, reflecting baseline and post-control emissions inventories.

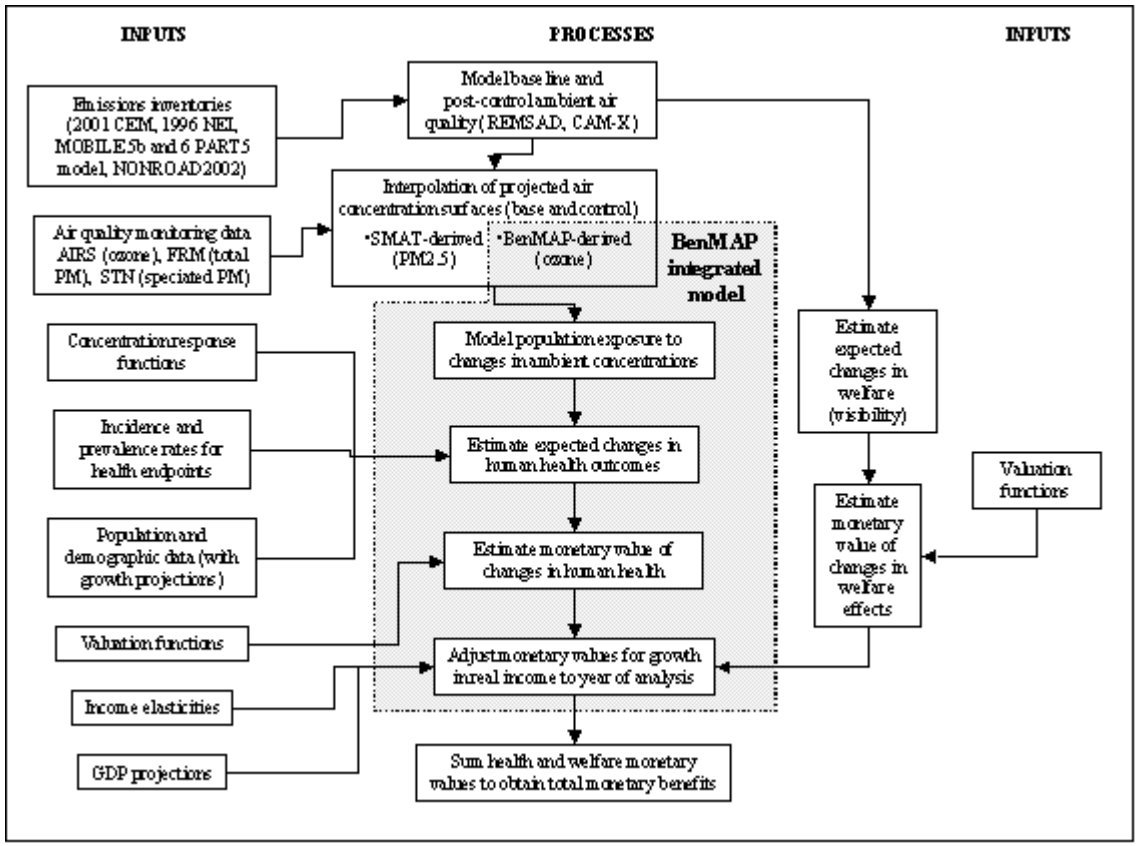
3) A benefits analysis to determine the changes in human health and welfare, both in terms of physical effects and monetary value, that result from the projected changes in ambient concentrations of various pollutants for the modeled standards.

A wide range of human health and welfare effects are linked to the emissions of NOx and SOx from EGUs and the resulting impact on ambient concentrations of ozone and PM. Potential human health effects linked to PM2.5 range from mortality linked to long-term exposure to PM, to a range of morbidity effects linked to long-term (chronic) and shorter-term (acute) exposures (e.g., respiratory and cardiovascular symptoms resulting in hospital admissions, asthma exacerbations, and acute and chronic bronchitis [CB]). Exposure to ozone has also been linked to a variety of respiratory effects including hospital admissions and illnesses resulting in school absences.[7] Welfare effects potentially linked to PM include materials damage and visibility impacts, while ozone can adversely affect the agricultural and forestry sectors by decreasing yields of crops and forests. Although methods exist for quantifying the benefits associated with many of these human health and welfare categories, not all can be evaluated at this time due to limitations in methods and/or data. Table 4-1 lists the full complement of human health and welfare effects associated with PM and ozone and identifies those effects that are quantified for the primary estimate, are quantified as part of the sensitivity analysis (to be completed for the supplemental analysis), and remain unquantified because of to current limitations in methods or available data.

Figure 4-1 illustrates the major steps in the benefits analysis. Given baseline and post-control emissions inventories for the emission species expected to affect ambient air quality, we use sophisticated photochemical air quality models to estimate baseline and post-control ambient concentrations of ozone and PM, and deposition of nitrogen and sulfur for each year. The estimated changes in ambient concentrations are then combined with monitoring data to estimate population-level exposures to changes in ambient concentrations for use in estimating health effects. Modeled changes in ambient data are also used to estimate changes in visibility, and changes in other air quality statistics that are necessary to estimate welfare effects. Changes in population

[7]Short-term exposure to ambient ozone has also been linked to premature death. The EPA is currently evaluating the epidemiological literature examining the relationship between ozone and premature mortality, sponsoring three independent meta-analyses of the literature. Once this evaluation has been completed and peer-reviewed, the EPA will consider including ozone-related premature mortality in the primary benefits analysis for the final rule.

Figure 4-1. Key Steps in Air Quality Modeling Based Benefits Analysis



exposure to ambient air pollution are then input to impact functions[8] to generate changes in incidence of health effects, or changes in other exposure metrics are input to dose-response functions to generate changes in welfare effects. The resulting effects changes are then assigned monetary values, taking into account adjustments to values for growth in real income out to the year of analysis (values for health and welfare effects are in general positively related to real income levels). Finally, values for individual health and welfare effects are summed to obtain an estimate of the total monetary value of the changes in emissions.

On September 26, 2002, the National Academy of Sciences (NAS) released a report on its review of the Agency's methodology for analyzing the health benefits of measures taken to reduce air pollution. The report focused on the EPA's approach for estimating the health benefits of regulations designed to reduce concentrations of airborne PM.

In its report, the NAS said that the EPA has generally used a reasonable framework for analyzing the health benefits of PM-control measures. It recommended, however, that the Agency take a number of steps to improve its benefits analysis. In particular, the NAS stated that the Agency should

- include benefits estimates for a range of regulatory options;
- estimate benefits for intervals, such as every 5 years, rather than a single year;
- clearly state the projected baseline statistics used in estimating health benefits, including those for air emissions, air quality, and health outcomes;
- examine whether implementation of proposed regulations might cause unintended impacts on human health or the environment;

[8]The term "impact function" as used here refers to the combination of (a) an effect estimate obtained from the epidemiological literature, (b) the baseline incidence estimate for the health effect of interest in the modeled population, (c) the size of that modeled population, and (d) the change in the ambient air pollution metric of interest. These elements are combined in the impact function to generate estimates of changes in incidence of the health effect. The impact function is distinct from the concentration response (C-R) function, which strictly refers to the estimated equation from the epidemiological study relating incidence of the health effect and ambient pollution. We refer to the specific value of the relative risk or estimated coefficients in the epidemiological study as the "effect estimate." In referencing the functions used to generate changes in incidence of health effects for this RIA, we use the term impact function rather than C-R function because "impact function" includes all key input parameters used in the incidence calculation.

- when appropriate, use data from non-U.S. studies to broaden age ranges to which current estimates apply and to include more types of relevant health outcomes; and

- begin to move the assessment of uncertainties from its ancillary analyses into its base analyses by conducting probabilistic, multiple-source uncertainty analyses. This assessment should be based on available data and expert judgment.

Although the NAS made a number of recommendations for improvement in the EPA's approach, it found that the studies selected by the Agency for use in its benefits analysis were generally reasonable choices. In particular, the NAS agreed with the EPA's decision to use cohort studies for estimating premature mortality benefits. It also concluded that the Agency's selection of the American Cancer Society (ACS) study for the evaluation of PM-related premature mortality was reasonable, although it noted the publication of new cohort studies that the Agency should evaluate. Since the publication of the NAS report, the EPA has reviewed new cohort studies, including reanalyses of the ACS study data and has carefully considered these new study data in developing the analytical approach for the IAQR (see below).

In addition to the NAS report, the EPA has also received technical guidance and input regarding its methodology for conducting PM- and ozone-related benefits analysis from two additional sources, including the Health Effects Subgroup (HES) of the SAB Council reviewing the 812 blueprint (SAB-HES, 2003) and the Office of Management and Budget (OMB) through ongoing discussions regarding methods used in conducting regulatory impact analyses (RIAs). The SAB HES recommendations include the following (SAB-HES, 2003):

- use of the updated ACS Pope et al. (2002) study rather than the ACS Krewski et al. study to estimate mortality for the primary analysis;

- dropping the alternative estimate used in earlier RIAs and instead including a primary estimate that incorporates consideration of uncertainty in key effects categories such as mortality directly into the estimates (e.g., use of the standard errors from the Pope et al. [2002] study in deriving confidence bounds for the adult mortality estimates);

- addition of infant mortality (children under the age of one) into the primary estimate, based on supporting evidence from the World Health Organization Global Burden of Disease study and other published studies that strengthen the evidence for a relationship between PM exposure and respiratory inflamation and infection in children leading to death;

- inclusion of asthma exacerbations for children in the primary estimate;
- expansion of the age groups evaluated for a range of morbidity effects beyond the narrow band of the studies to the broader (total) age group (e.g., expanding a study population for 7 to 11 year olds to cover the entire child age range of 6 to 18 years).
- inclusion of new endpoints (school absences [ozone], nonfatal heart attacks in adults [PM], hospital admissions for children under two [ozone]), and suggestion of a new meta-analysis of hospital admissions (PM10) rather than using a few PM2.5 studies;[9] and
- updating of populations and baseline incidences.

Recommendations from OMB regarding RIA methods have focused on the approach used to characterize uncertainty in the benefits estimates generated for RIAs, as well as the approach used to value mortality estimates. The EPA is currently in the process of developing a comprehensive integrated strategy for characterizing the impact of uncertainty in key elements of the benefits modeling process (e.g., emissions modeling, air quality modeling, health effects incidence estimation, valuation) on the results that are generated. A subset of this effort, which is currently underway, involves an expert elicitation designed to characterize uncertainty in the estimation of PM-related mortality resulting from both short-term and longer-term exposure. The EPA will be evaluating the results of this elicitation to determine its usefulness in characterizing uncertainty in our estimates of PM-related mortality benefits. As elements of this uncertainty analysis strategy are finalized, it may be possible to integrate them into later iterations of the analysis completed for the IAQR (e.g., the supplemental analysis and final rule).

We are also altering the value of a statistical life (VSL) used in the analysis to reflect new information in the ongoing academic debate over the appropriate characterization of the value of reducing the risk of premature mortality. In previous analyses, we used a distribution of VSL based on 26 VSL estimates from the economics literature. For this analysis, we are characterizing the VSL distribution in a more general fashion, based on two recent meta-

[9]Note that the SAB-HES comments were made in the context of a review of the methods for the Section 812 analysis of the costs and benefits of the Clean Air Act. This context is pertinent to our interpretation of the SAB- HES comments on the selection of effect estimates for hospital admissions associated with PM (SAB-HES, 2003). The Section 812 analysis is focused on a broad set of air quality changes, including both the coarse and fine fractions of PM10. As such, impact functions that focus on the full impact of PM10 are appropriate. However, for the IAQR, which is expected to affect primarily the fine fraction (PM2.5) of PM10, impact functions that focus primarily on PM2.5 are more appropriate.

analyses of the wage-risk-based VSL literature. The new distribution is assumed to be normal, with a mean of $5.5 million and a 95 percent confidence interval between $1 and $10 million. The EPA welcomes public comment on the appropriate methodology for valuing reductions in the risk of premature death.

The EPA has addressed many of the comments received from the NAS, the SAB-HES, and OMB in developing the analytical approach for the IAQR. We have also reflected advances in data and methods in air quality modeling, epidemiology, and economics in developing this analysis. Updates to the assumptions and methods used in estimating PM 2.5-related and ozone-related benefits since completion of the Proposed Nonroad Diesel Rule include the following:

Air Quality

- Use of the Simulated Modeled Attainment Test (SMAT) approach for developing PM2.5 air modeling results. The nonroad diesel rule used spatially and temporally scaled total PM2.5 mass based on monitoring data from 1999 to 2001 (averaged by season). For the nonroad diesel rule, spatial scaling was based on 1996 modeled REMSAD data at a 36 km grid resolution, while temporal scaling was based on the ratios of future modeled REMSAD data to 1996 modeled REMSAD data. All scaling was conducted internally by BenMAP (see below) using the monitor and model relative grid creation option. Resulting gridded outputs were for binned daily PM2.5 averages. For the IAQR, we used the SMAT approach, which uses temporally scaled speciated PM2.5 monitor data from 2001-2002, reconstructed into total PM2.5 mass based on 2000-2002 design values and kriged to 12 kilometer grids (nested within the standard 36 km REMSAD grid structure). Temporal scaling is based on ratios of future modeled REMSAD data to 2001 REMSAD model data, using REMSAD modeling conducted at the 36 km grid resolution. SMAT output files include both quarterly mean and annual mean PM2.5 mass results, which are then manipulated within SAS to produce a BenMAP input file containing 364 daily values (created by replicating the quarterly mean values for each day of the appropriate season). For more details on the SMAT approach and REMSAD modeling, see the air quality chapter of this document.

- For both PM and ozone, the interstate air quality analysis domain will include only the eastern United States, focusing on 37 States believed to contribute significantly to the long-range transport of precursors in the formation of PM2.5.

Health Endpoints

- Incorporation of updated impact functions to reflect updated time-series studies of hospital admissions to correct for errors in application of the generalized additive model (GAM) functions in S-plus. More information on this issue is available at http://www.healtheffects.org.
- The primary analysis will use an all cause mortality effect estimate based on the Pope et al. (2002) reanalysis of the ACS study data. In addition, we will provide a breakout for two major cause of death categories—cardiopulmonary and lung cancer.
- Infant mortality will be included in the primary analysis.
- Asthma exacerbations are incorporated into the primary analysis. Although the Nonroad Diesel Rule included asthma exacerbations as a separate endpoint outside of the base case analysis, for the IAQR, we will include asthma exacerbations in children 6 to 18 years of age as part of the primary analysis.

Valuation

- In generating the monetized benefits for mortality in the primary analysis, the VSL will be entered as a mean (best estimate) of 5.5 million. Unlike the Nonroad Diesel Rule, the IAQR will not include a value of statistical life year (VSLY) estimate.

In response to comments from the SAB-HES as well as the NAS panel, rather than including an alternative estimate in the IAQR, the EPA will investigate the impact of key assumptions on mortality and morbidity estimates through a series of sensitivity analyses (to be completed for the supplemental analysis).

The benefits estimates generated for the Proposed IAQR are subject to a number of assumptions and uncertainties, which are discussed throughout the document. For example, key assumptions underlying the primary estimate for the mortality category include the following:

(1) Inhalation of fine particles is causally associated with premature death at concentrations near those experienced by most Americans on a daily basis. Although biological mechanisms for this effect have not yet been definitively established, the weight of the available epidemiological evidence supports an assumption of causality.

(2) All fine particles, regardless of their chemical composition, are equally potent in causing premature mortality. This is an important assumption, because PM produced via transported precursors emitted from EGUs may differ significantly from direct PM released from automotive engines and other industrial sources, but no clear scientific grounds exist for supporting differential effects estimates by particle type.

(3) The C-R function for fine particles is approximately linear within the range of ambient concentrations under consideration. Thus, the estimates include health benefits from reducing fine particles in areas with varied concentrations of PM, including both regions that are in attainment with fine particle standard and those that do not meet the standard.

(4) The forecasts for future emissions and associated air quality modeling are valid. Although recognizing the difficulties, assumptions, and inherent uncertainties in the overall enterprise, these analyses are based on peer-reviewed scientific literature and up-to-date assessment tools, and we believe the results are highly useful in assessing this proposal.

In addition to the quantified and monetized benefits summarized above, a number of additional categories are not currently amenable to quantification or valuation. These include reduced acid and particulate deposition damage to cultural monuments and other materials, reduced ozone effects on forested ecosystems, and environmental benefits due to reductions of impacts of acidification in lakes and streams and eutrophication in coastal areas. Additionally, we have not quantified a number of known or suspected health effects linked with PM and ozone for which appropriate health impact functions are not available or which do not provide easily interpretable outcomes (i.e., changes in forced expiratory volume [FEV1]). As a result, monetized benefits generated for the primary estimate may underestimate the total benefits attributable to the proposed regulatory option.

Benefits estimates for the Proposed IAQR were generated using BenMAP, which is a computer program developed by the EPA that integrates a number of the modeling elements used in previous RIAs (e.g., interpolation functions, population projections, health impact functions, valuation functions, analysis and pooling methods) to translate modeled air concentration estimates into health effects incidence estimates and monetized benefits estimates. BenMAP provides estimates of both the mean impacts and the distribution of impacts.

Table 4-1. Estimated Monetized Benefits of the Proposed IAQR

	Total Benefits[a, b] (billions 1999$)	
	2010	2015
Using a 3% discount rate	$58+B	$84+B
Using a 7% discount rate	$54+B	$79+B

[a] For notational purposes, unquantified benefits are indicated with a "B" to represent the sum of additional monetary benefits and disbenefits. A detailed listing of unquantified health and welfare effects is provided in Table 4-2.

[b] Results reflect the use of two different discount rates: a 3 percent rate, which is recommended by the EPA's *Guidelines for Preparing Economic Analyses* (EPA, 2000c), and 7 percent, which is recommended by OMB Circular A-94 (OMB, 1992). Results are rounded to two significant digits.

In general, the chapter is organized around the steps illustrated in Figure 4-1. In Section 4.1, we provide an overview of the data and methods that are used to quantify and value health and welfare endpoints and discuss how we incorporate uncertainty into our analysis. In Section 4.2, we report the results of the analysis for human health and welfare effects (the overall benefits estimated for the Proposed IAQR are summarized in Table 4-1). Details on the emissions inventory and air modeling are presented in Chapter 3.0.

4.1 Benefit Analysis- Data and Methods

Environmental and health economists have a number of methods for estimating the economic value of improvements in (or deterioration of) environmental quality. The method used in any given situation depends on the nature of the effect and the kinds of data, time, and resources that are available for investigation and analysis. This section provides an overview of the methods we selected to quantify and monetize the benefits included in this RIA.

Given changes in environmental quality (ambient air quality, visibility, nitrogen, and sulfate deposition), the next step is to determine the economic value of those changes. We follow a "damage-function" approach in calculating total benefits of the modeled changes in environmental quality. This approach estimates changes in individual health and welfare

endpoints (specific effects that can be associated with changes in air quality) and assigns values to those changes assuming independence of the individual values. Total benefits are calculated simply as the sum of the values for all nonoverlapping health and welfare endpoints. This imposes no overall preference structure and does not account for potential income or substitution effects (i.e., adding a new endpoint will not reduce the value of changes in other endpoints). The "damage-function" approach is the standard approach for most cost-benefit analyses of environmental quality programs and has been used in several

Table 4-2. Human Health and Welfare Effects of Pollutants Affected by the Proposed IAQR

	Quantified and Monetized in Base	Quantified and/or Monetized Effects in	
	Hospital admissions: respiratory Emergency room visits for asthma Minor restricted activity days[a] School loss days	Asthma attacks Cardiovascular emergency room visits **Sensitivity Analyses** Acute respiratory symptoms	Increased airway responsiveness to stimuli Inflammation in the lung **Unquantified Effects** Chronic respiratory damage Premature aging of the lungs Acute inflammation and respiratory cell damage Increased susceptibility to respiratory infection Nonasthma respiratory emergency room visits
	Decreased outdoor worker productivity	Decreased yields for commercial crops (selected species) Decreased eastern commercial forest productivity (selected species)	Decreased western commercial forest productivity Decreased eastern commercial forest productivity (other species) Decreased yields for fruits and vegetables Decreased yields for other commercial and noncommercial crops Damage to urban ornamental plants Impacts on recreational demand from damaged forest aesthetics Damage to ecosystem functions

Table 4-2. Human Health and Welfare Effects of Pollutants Affected by the Proposed IAQR (continued)

	Quantified and Monetized in Base	Quantified and/or Monetized Effects in	
	Premature mortality: long-term exposures Bronchitis: chronic and acute Hospital admissions: respiratory and cardiovascular Emergency room visits for asthma Non-fatal heart attacks (myocardial infarction) Lower and upper respiratory illness Minor restricted activity days Work loss days Asthma exacerbations (asthmatic population) Respiratory symptoms (asthmatic population) Infant mortality	Premature mortality: short-term exposures **Sensitivity Analyses**	Low birth weight Changes in pulmonary function **Unquantified Effects** Chronic respiratory diseases other than chronic bronchitis Morphological changes Altered host defense mechanisms Nonasthma respiratory emergency room visits
	Visibility in Southeastern Class I areas	Visibility in northeastern and Midwestern Class I areas Visibility in residential and non-Class I areas Household soiling	Visibility in western U.S. Class I areas

Table 4-2. Human Health and Welfare Effects of Pollutants Affected by the Proposed IAQR (continued)

	Quantified and Monetized in Base	Quantified and/or Monetized Effects in	
Nitrogen and Sulfate Deposition/ Welfare	[a]	**Sensitivity Analyses**	Impacts of acidic sulfate and nitrate deposition on commercial forests **Unquantified Effects** Impacts of acidic deposition on commercial freshwater fishing Impacts of acidic deposition on recreation in terrestrial ecosystems Impacts of nitrogen deposition on commercial fishing, agriculture, and forests Impacts of nitrogen deposition on recreation in estuarine ecosystems Reduced existence values for currently healthy ecosystems
			Hospital admissions for respiratory and cardiac diseases Respiratory symptoms in asthmatics
			Lung irritation Lowered resistance to respiratory infection Hospital admissions for respiratory and cardiac diseases

Table 4-2. Human Health and Welfare Effects of Pollutants Affected by the Proposed IAQR (continued)

	Quantified and Monetized in Base	Quantified and/or Monetized Effects in	
Mercury Deposition/ Health	[a]	**Sensitivity Analyses**	Neurological disorders Learning disabilities **Unquantified Effects** Retarded development Potential cardiovascular effects * Altered blood pressure regulation * Increased heart rate variability * Myocardial infarctions * Potential reproductive effects *
Mercury Deposition/ Welfare			Impacts on birds and mammals (e.g., reproductive effects) Impacts to commercial, subsistence, and recreational fishing Reduced existence values for currently healthy ecosystems

a more complete discussion of presentation of benefits estimates.

have a significant effect on daily mortality rates, independent of exposure to PM. The EPA is currently conducting a series of meta-analyses of the ozone mortality epidemiology literature and will reevaluate inclusion of ozone-related mortality in the primary analysis once the meta-analyses have been completed.

recent published analyses (Banzhaf et al., 2002; Levy et al., 2001; Levy et al., 1999; Ostro and Chestnut, 1998).

To assess economic value in a damage-function framework, the changes in environmental quality must be translated into effects on people or on the things that people value. In some cases, the changes in environmental quality can be directly valued, as is the case for changes in visibility. In other cases, such as for changes in ozone and PM, a health and welfare impact analysis must first be conducted to convert air quality changes into effects that can be assigned dollar values.

For the purposes of this RIA, the health impacts analysis is limited to those health effects that are directly linked to ambient levels of air pollution and specifically to those linked to ozone and PM. There may be other, indirect health impacts associated with implementing controls to meet the preliminary control options, such as occupational health impacts for equipment operators. These impacts may be positive or negative, but in general, for this set of control options, they are expected to be small relative to the direct air pollution-related impacts.

The welfare impacts analysis is limited to changes in the environment that have a direct impact on human welfare. For this analysis, we are limited by the available data to examining impacts of changes in visibility. We also provide qualitative discussions of the impact of changes in other environmental and ecological effects, for example, changes in deposition of nitrogen and sulfur to terrestrial and aquatic ecosystems, but we are unable to place an economic value on these changes.

We note at the outset that the EPA rarely has the time or resources to perform extensive new research to measure either the health outcomes or their values for this analysis. Thus, similar to Kunzli et al. (2000) and other recent health impact analyses, our estimates are based on the best available methods of benefits transfer. Benefits transfer is the science and art of adapting primary research from similar contexts to obtain the most accurate measure of benefits for the environmental quality change under analysis. Where appropriate, adjustments are made for the level of environmental quality change, the sociodemographic and economic characteristics of the affected population, and other factors to improve the accuracy and robustness of benefits estimates.

4.1.1 Valuation Concepts

In valuing health impacts, we note that reductions in ambient concentrations of air pollution generally lower the risk of future adverse health affects by a fairly small amount for a large population. The appropriate economic measure is therefore willingness to pay (WTP) for changes in risk prior to the regulation (Freeman, 1993). In general, economists tend to view an individual's WTP for an improvement in environmental quality as the appropriate measure of the value of a risk reduction. An individual's willingness to accept (WTA) compensation for not receiving the improvement is also a valid measure. However, WTP is generally considered to be a more readily available and conservative measure of benefits. Adoption of WTP as the measure of value implies that the value of environmental quality improvements depends on the individual preferences of the affected population and that the existing distribution of income (ability to pay) is appropriate. For some health effects, such as hospital admissions, WTP estimates are generally not available. In these cases, we use the cost of treating or mitigating the effect as a primary estimate. These cost of illness (COI) estimates generally understate the true value of reductions in risk of a health effect, reflecting the direct expenditures related to treatment but not the value of avoided pain and suffering from the health effect (Harrrington and Portnoy, 1987; Berger, 1987).

For many goods, WTP can be observed by examining actual market transactions. For example, if a gallon of bottled drinking water sells for $1, it can be observed that at least some people are willing to pay $1 for such water. For goods not exchanged in the market, such as most environmental "goods," valuation is not as straightforward. Nevertheless, a value may be inferred from observed behavior, such as sales and prices of products that result in similar effects or risk reductions (e.g., nontoxic cleaners or bike helmets). Alternatively, surveys can be used in an attempt to directly elicit WTP for an environmental improvement.

One distinction in environmental benefits estimation is between use values and nonuse values. Although no general agreement exists among economists on a precise distinction between the two (see Freeman [1993]), the general nature of the difference is clear. Use values are those aspects of environmental quality that affect an individual's welfare more or less directly. These effects include changes in product prices, quality, and availability; changes in the quality of outdoor recreation and outdoor aesthetics; changes in health or life expectancy; and the costs of actions taken to avoid negative effects of environmental quality changes.

Nonuse values are those for which an individual is willing to pay for reasons that do not relate to the direct use or enjoyment of any environmental benefit but might relate to existence values and bequest values. Nonuse values are not traded, directly or indirectly, in markets. For this reason, the measurement of nonuse values has proved to be significantly more difficult than the measurement of use values. The air quality changes produced by the IAQR cause changes in both use and nonuse values, but the monetary benefit estimates are almost exclusively for use values.

More frequently than not, the economic benefits from environmental quality changes are not traded in markets, so direct measurement techniques cannot be used. There are three main nonmarket valuation methods used to develop values for endpoints considered in this analysis: stated preference (or contingent valuation [CV]), indirect market (e.g., hedonic wage), and avoided cost methods.

The stated preference or CV method values endpoints by using carefully structured surveys to ask a sample of people what amount of compensation is equivalent to a given change in environmental quality. There is an extensive scientific literature and body of practice on both the theory and technique of stated preference-based valuation. The EPA believes that well-designed and well-executed stated preference studies are valid for estimating the benefits of air quality regulations.[10] Stated preference valuation studies form the basis for valuing a number of health and welfare endpoints, including the value of mortality risk reductions, CB risk reductions, minor illness risk reductions, and visibility improvements.

Indirect market methods can also be used to infer the benefits of pollution reduction. The most important application of this technique for our analysis is the calculation of the VSL for use in estimating benefits from mortality risk reductions. No market exists where changes in the probability of death are directly exchanged. However, people make decisions about

[10]Concerns about the reliability of value estimates from CV studies arose because research has shown that bias can be introduced easily into these studies if they are not carefully conducted. Accurately measuring WTP for avoided health and welfare losses depends on the reliability and validity of the data collected. There are several issues to consider when evaluating study quality, including but not limited to 1) whether the sample estimates of WTP are representative of the population WTP; 2) whether the good to be valued is comprehended and accepted by the respondent; 3) whether the WTP elicitation format is designed to minimize strategic responses; 4) whether WTP is sensitive to respondent familiarity with the good, to the size of the change in the good, and to income; 5) whether the estimates of WTP are broadly consistent with other estimates of WTP for similar goods; and 6) the extent to which WTP responses are consistent with established economic principles.

occupation, precautionary behavior, and other activities associated with changes in the risk of death. By examining these risk changes and the other characteristics of people's choices, it is possible to infer information about the monetary values associated with changes in mortality risk (see Section 4.1.5.5.1).

Avoided cost methods are ways to estimate the costs of pollution by using the expenditures made necessary by pollution damage. For example, if buildings must be cleaned or painted more frequently as levels of PM increase, then the appropriately calculated increment of these costs is a reasonable lower-bound estimate (under most conditions) of true economic benefits when PM levels are reduced. Avoided costs methods are also used to estimate some of the health-related benefits related to morbidity, such as hospital admissions (see Section 4.1.5).

4.1.2 Growth in WTP Reflecting National Income Growth Over Time

Our analysis accounts for expected growth in real income over time. Economic theory argues that WTP for most goods (such as environmental protection) will increase if real incomes increase. There is substantial empirical evidence that the income elasticity[11] of WTP for health risk reductions is positive, although there is uncertainty about its exact value. Thus, as real income increases, the WTP for environmental improvements also increases. Although many analyses assume that the income elasticity of WTP is unit elastic (i.e., 10 percent higher real income level implies a 10 percent higher WTP to reduce risk changes), empirical evidence suggests that income elasticity is substantially less than one and thus relatively inelastic. As real income rises, the WTP value also rises but at a slower rate than real income.

The effects of real income changes on WTP estimates can influence benefit estimates in two different ways: through real income growth between the year a WTP study was conducted and the year for which benefits are estimated, and through differences in income between study populations and the affected populations at a particular time. Empirical evidence of the effect of real income on WTP gathered to date is based on studies examining the former. The Environmental Economics Advisory Committee (EEAC) of the SAB advised the EPA to adjust WTP for increases in real income over time but not to adjust WTP to account for cross-sectional income differences "because of the sensitivity of making such

[11]Income elasticity is a common economic measure equal to the percentage change in WTP for a 1 percent change in income.

distinctions, and because of insufficient evidence available at present" (EPA-SAB-EEAC-00-013).

Based on a review of the available income elasticity literature, we adjust the valuation of human health benefits upward to account for projected growth in real U.S. income. Faced with a dearth of estimates of income elasticities derived from time-series studies, we applied estimates derived from cross-sectional studies in our analysis. Details of the procedure can be found in Kleckner and Neumann (1999). An abbreviated description of the procedure we used to account for WTP for real income growth between 1990 and 2010 and 2015 is presented below.

Reported income elasticities suggest that the severity of a health effect is a primary determinant of the strength of the relationship between changes in real income and WTP. As such, we use different elasticity estimates to adjust the WTP for minor health effects, severe and chronic health effects, and premature mortality. We also expect that the WTP for improved visibility in Class I areas would increase with growth in real income. The elasticity values used to adjust estimates of benefits in 2010 and 2015 are presented in Table 4-3.

Table 4-3. Elasticity Values Used to Account for Projected Real Income Growth[a]

Benefit Category	Central Elasticity Estimate
Minor Health Effect	0.14
Severe and Chronic Health Effects	0.45
Premature mortality	0.40
Visibility[b]	0.90

[a] Derivation of estimates can be found in Kleckner and Neumann (1999) and Chestnut (1997). COI estimates are assigned an adjustment factor of 1.0.

[b] No range was applied for visibility because no ranges were available in the current published literature.

In addition to elasticity estimates, projections of real gross domestic product (GDP) and populations from 1990 to 2010 and 2015 are needed to adjust benefits to reflect real per capita income growth. For consistency with the emissions and benefits modeling, we use national population estimates for the years 1990 to 1999 based on U.S. Census Bureau

estimates (Hollman, Mulder and Kallan, 2000). These population estimates are based on application of a cohort-component model applied to 1990 U.S. Census data projections (U.S. Bureau of Census, 2000).[12] For the years between 2000 and 2015, we applied growth rates based on the U.S. Census Bureau projections to the U.S. Census estimate of national population in 2000. We use projections of real GDP provided in Kleckner and Neumann (1999) for the years 1990 to 2010.[13] We use projections of real GDP (in chained 1996 dollars) provided by Standard and Poor's[14] (2000) for the years 2010 to 2015.[15]

Using the method outlined in Kleckner and Neumann (1999) and the population and income data described above, we calculate WTP adjustment factors for each of the elasticity estimates listed in Table 4-4. Benefits for each of the categories (minor health effects, severe and chronic health effects, premature mortality, and visibility) will be adjusted by multiplying the unadjusted benefits by the appropriate adjustment factor. Table 4-4 lists the estimated adjustment factors. Note that, for premature mortality, we apply the income adjustment factor *ex post* to the present discounted value of the stream of avoided mortalities occurring over the lag period. Also note that no adjustments will be made to benefits based on the COI approach or to work loss days and worker productivity. This assumption will also lead us to underpredict benefits in future years because it is likely that increases in real U.S. income would also result in increased COI (due, for example, to increases in wages paid to medical workers) and increased cost of work loss days and lost worker productivity (reflecting that if worker incomes are higher, the losses resulting from reduced worker production would also be higher).

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[12]U.S. Bureau of Census. Annual Projections of the Total Resident Population, Middle Series, 1999-2100. (Available on the internet at http://www.census.gov/population/www/projections/natsum-T1.html)

[13]U.S. Bureau of Economic Analysis, Table 2A (1992$). (Available on the internet at http://www.bea.doc.gov/bea/dn/0897nip2/tab2a.htm) and U.S. Bureau of Economic Analysis, Economics and Budget Outlook. Note that projections for 2007 to 2010 are based on average GDP growth rates between 1999 and 2007.

[14]Standard and Poor's. 2000. "The U.S. Economy: The 25 Year Focus." Winter.

[15]In previous analyses, we used the Standard and Poor's projections of GDP directly. This led to an apparent discontinuity in the adjustment factors between 2010 and 2011. We refined the method by applying the relative growth rates for GDP derived from the Standard and Poor's projections to the 2010 projected GDP based on the Bureau of Economic Analysis projections.

Table 4-4. Adjustment Factors Used to Account for Projected Real Income Growth[a]

Benefit Category	2010	2015
Minor Health Effect	1.034	1.073
Severe and Chronic Health Effects	1.113	1.254
Premature Mortality	1.100	1.222
Visibility	1.239	1.581

[a] Based on elasticity values reported in Table 4-3, U.S. Census population projections, and projections of real gross domestic product per capita

4.1.3 Methods for Describing Uncertainty

In any complex analysis using estimated parameters and inputs from numerous models, there are likely to be many sources of uncertainty. This analysis is no exception. As outlined both in this and preceding chapters, many inputs are used to derive the final estimate of benefits, including emission inventories, air quality models (with their associated parameters and inputs), epidemiological health effect estimates, estimates of values (both from WTP and COI studies), population estimates, income estimates, and estimates of the future state of the world (i.e., regulations, technology, and human behavior). Each of these inputs may be uncertain and, depending on their location in the benefits analysis, may have a disproportionately large impact on final estimates of total benefits. For example, emissions estimates are used in the first stage of the analysis. As such, any uncertainty in emissions estimates will be propagated through the entire analysis. When compounded with uncertainty in later stages, small uncertainties in emission levels can lead to much larger impacts on total benefits.

Some key sources of uncertainty in each stage of the benefits analysis are the following:

- gaps in scientific data and inquiry;
- variability in estimated relationships, such as epidemiological effect estimates, introduced through differences in study design and statistical modeling;

- errors in measurement and projection for variables such as population growth rates;
- errors due to misspecification of model structures, including the use of surrogate variables, such as using PM_{10} when $PM_{2.5}$ is not available, excluded variables, and simplification of complex functions; and
- biases due to omissions or other research limitations.

Some of the key uncertainties in the benefits analysis are presented in Table 4-5. Given the wide variety of sources for uncertainty and the potentially large degree of uncertainty about any primary estimate, it is necessary for us to address this issue in several ways, based on the following types of uncertainty:

Table 4-5. Primary Sources of Uncertainty in the Benefit Analysis

1. Uncertainties Associated With Impact Functions
– The value of the ozone or PM effect estimate in each impact function.
– Application of a single impact function to pollutant changes and populations in all locations.
– Similarity of future year impact functions to current impact functions.
– Correct functional form of each impact function.
– Extrapolation of effect estimates beyond the range of ozone or PM concentrations observed in the source epidemiological study.
– Application of impact functions only to those subpopulations matching the original study population.
2. Uncertainties Associated With Ozone and PM Concentrations
– Responsiveness of the models to changes in precursor emissions resulting from the control policy.
– Projections of future levels of precursor emissions, especially ammonia and crustal materials.
– Model chemistry for the formation of ambient nitrate concentrations.
– Lack of ozone monitors in rural areas requires extrapolation of observed ozone data from urban to rural areas.
– Use of separate air quality models for ozone and PM does not allow for a fully integrated analysis of pollutants and their interactions.
– Full ozone season air quality distributions are extrapolated from a limited number of simulation days.
– Comparison of model predictions of particulate nitrate with observed rural monitored nitrate levels indicates that REMSAD overpredicts nitrate in some parts of the Eastern US
3. Uncertainties Associated with PM Mortality Risk
– Limited scientific literature supporting a direct biological mechanism for observed epidemiological evidence.
– Direct causal agents within the complex mixture of PM have not been identified.
– The extent to which adverse health effects are associated with low level exposures that occur many times in the year versus peak exposures.
– The extent to which effects reported in the long-term exposure studies are associated with historically higher levels of PM rather than the levels occurring during the period of study.
– Reliability of the limited ambient $PM_{2.5}$ monitoring data in reflecting actual $PM_{2.5}$ exposures.
4. Uncertainties Associated With Possible Lagged Effects
– The portion of the PM-related long-term exposure mortality effects associated with changes in annual PM levels would occur in a single year is uncertain as well as the portion that might occur in subsequent years.
5. Uncertainties Associated With Baseline Incidence Rates
– Some baseline incidence rates are not location-specific (e.g., those taken from studies) and may therefore not accurately represent the actual location-specific rates.
– Current baseline incidence rates may not approximate well baseline incidence rates in 2015.
– Projected population and demographics may not represent well future-year population and demographics.
6. Uncertainties Associated With Economic Valuation
– Unit dollar values associated with health and welfare endpoints are only estimates of mean WTP and therefore have uncertainty surrounding them.
– Mean WTP (in constant dollars) for each type of risk reduction may differ from current estimates due to differences in income or other factors.
7. Uncertainties Associated With Aggregation of Monetized Benefits
– Health and welfare benefits estimates are limited to the available impact functions. Thus, unquantified or unmonetized benefits are not included.

a. *Quantifiable uncertainty in benefits estimates.* For some parameters or inputs it may be possible to provide a statistical representation of the underlying

uncertainty distribution. Quantitative uncertainty may include measurement uncertainty or variation in estimates across or within studies. For example, the variation in VSL results across available meta-analyses provides a source of uncertainty that can be characterized in calculating monetized benefits. Methods typically used to evaluate the impact of these quantifiable sources of uncertainty on benefits and incidence estimates center on Monte Carlo-based probabilistic simulation. This technique allows uncertainty in key inputs to be propagated through the model to generate a single distribution of results reflecting the combined impact of multiple sources of uncertainty. Variability can also be considered along with uncertainty using nested two-stage Monte Carlo simulation.

b. *Uncertainty in the basis for quantified estimates*. Often it is possible to identify a source of uncertainty (e.g., an ongoing debate over the proper method to estimate premature mortality) that is not readily addressed through traditional uncertainty analysis. In these cases, it is possible to characterize the potential impact of this uncertainty on the overall benefits estimates through sensitivity analyses.

c. *Nonquantifiable uncertainty.* Uncertainties may also result from omissions of known effects from the benefits calculation, perhaps owing to a lack of data or modeling capability. For example, in this analysis we were unable to quantify the benefits of avoided airborne nitrogen deposition on aquatic and terrestrial ecosystems.

It should be noted that, even for individual endpoints, there is usually more than one source of uncertainty. This makes it difficult to provide an overall quantified uncertainty estimate for individual endpoints or for total benefits, without conducting a comprehensive uncertainty analysis that considers the aggregate impact of multiple sources of uncertainty on benefits estimates.

The NAS report on the EPA's benefits analysis methodology highlighted the need for the EPA to conduct rigorous quantitative analysis of uncertainty in its benefits estimates. In response to these comments, the EPA has initiated the development of a comprehensive methodology for characterizing the aggregate impact of uncertainty in key modeling elements on both health incidence and benefits estimates. This methodology will begin by identifying those modeling elements that have a significant impact on benefits due to either the magnitude of their uncertainty or other factors such as nonlinearity within the modeling framework. A combination of influence analysis and sensitivity analysis methods may be

used to focus the analysis of uncertainty on these key sources of uncertainty. A probabilistic simulation approach based on Monte Carlo methods will be developed for propagating the impact of these sources of uncertainty through the modeling framework. Issues such as correlation between input parameters and the identification of reasonable upper and lower bounds for input distributions characterizing uncertainty will be addressed in developing the approach.

One component of the EPA's uncertainty analysis methodology that is currently underway is an expert elicitation intended to characterize uncertainty in the effect estimates used to estimate mortality resulting from both short-term (timer series studies) and longer-term (cohort studies) exposure to PM. This expert elicitation is aimed at evaluating uncertainty in both the form of the mortality impact function (e.g., threshold versus linear models) and the fit of a specific model to the data (e.g., confidence bounds for specific percentiles of the mortality effect estimates). Additional issues such as the ability of longer-term cohort studies to capture mortality resulting from short-term peak PM exposures is also being addressed in the expert elicitation.

EPA will consider incorporating elements of this uncertainty analysis methodology, including information from the expert elicitation addressing the mortality estimate, into subsequent analysis conducted for the IAQR (e.g., the Supplemental Analysis and Final Rule) as they become available. For the Proposed IAQR, EPA has addressed key sources of uncertainty through a series of sensitivity analyses (to be completed for the supplemental analysis) examining the impact of alternate assumptions on the benefits estimates that are generated.

Our estimate of total benefits should be viewed as an approximate result because of the sources of uncertainty discussed above (see Table 4-5). Uncertainty about specific aspects of the health and welfare estimation models are discussed in greater detail in the following sections. The total benefits estimate may understate or overstate actual benefits of the rule.

In considering the monetized benefits estimates, the reader should remain aware of the many limitations of conducting these analyses mentioned throughout this RIA. One significant limitation of both the health and welfare benefits analyses is the inability to quantify many of the serious effects listed in Table 4-1. For many health and welfare effects, such as changes in ecosystem functions and PM-related materials damage, reliable impact functions and/or valuation functions are not currently available. In general, if it were possible to monetize these benefits categories, the benefits estimates presented in this analysis would increase. Unquantified benefits are qualitatively discussed in the health and welfare effects sections. In addition to unquantified benefits, there may also be environmental costs that we are unable to quantify. These endpoints are qualitatively discussed in the health and welfare effects sections as well. The net effect of excluding benefit and disbenefit categories from the estimate of total benefits depends on the relative magnitude of the effects.

4.1.4 Demographic Projections

Quantified and monetized human health impacts depend critically on the demographic characteristics of the population, including age, location, and income. In previous analyses, we have used simple projections of total population that did not take into account changes in demographic composition over time. In the current analysis, we use more sophisticated projections based on economic forecasting models developed by Woods and Poole, Inc. The Woods and Poole (WP) database contains county-level projections of population by age, sex, and race out to 2025. Projections in each county are determined simultaneously with every other county in the United States to take into account patterns of economic growth and migration. The sum of growth in county-level populations is constrained to equal a previously determined national population growth, based on Bureau of Census estimates (Hollman, Mulder and Kallan, 2000). According to WP, linking county-level growth projections together and constraining to a national-level total growth avoids potential errors introduced by forecasting each county independently. County projections are developed in a four-stage process. First, national-level variables such as income, employment, populations, etc. are forecasted. Second, employment projections are made for 172 economic areas defined by the Bureau of Economic Analysis, using an "export-base" approach, which relies on linking industrial sector production of nonlocally consumed production items, such as outputs from mining, agriculture, and manufacturing with the national economy. The export-base approach requires estimation of demand equations or calculation of historical growth rates for output and employment by sector. Third, population is projected for each economic area based on net migration rates derived from employment opportunities and following a

cohort-component method based on fertility and mortality in each area. Fourth, employment and population projections are repeated for counties, using the economic region totals as bounds. The age, sex, and race distributions for each region or county are determined by aging the population by single year of age by sex and race for each year through 2015 based on historical rates of mortality, fertility, and migration.

The WP projections of county-level population are based on historical population data from 1969-1999 and do not include the 2000 Census results. Given the availability of detailed 2000 Census data, we constructed adjusted county-level population projections for each future year using a two-stage process. First, we constructed ratios of the projected WP populations in a future year to the projected WP population in 2000 for each future year by age, sex, and race. Second, we multiplied the block level 2000 Census population data by the appropriate age-, sex-, and race-specific WP ratio for the county containing the census block, for each future year. This results in a set of future population projections that is consistent with the most recent detailed census data.

As noted above, values for environmental quality improvements are expected to increase with growth in real per capita income. Accounting for real income growth over time requires projections of both real GDP and total U.S. populations. For consistency with the emissions and benefits modeling, we use national population estimates based on the U.S. Census Bureau projections.

4.1.5 Health Benefits Assessment Methods

The most significant monetized benefits of reducing ambient concentrations of PM and ozone are attributable to reductions in health risks associated with air pollution. The EPA's Criteria Documents for ozone and PM list numerous health effects known to be linked to ambient concentrations of these pollutants (EPA, 1996a and 1996b). As illustrated in Figure 4-1, quantification of health impacts requires several inputs, including epidemiological effect estimates, baseline incidence and prevalence rates, potentially affected populations, and estimates of changes in ambient concentrations of air pollution. Previous sections have described the population and air quality inputs. This section describes the effect estimates and baseline incidence and prevalence inputs and the methods used to quantify and monetize changes in the expected number of incidences of various health effects.

4.1.5.1 Selecting Health Endpoints and Epidemiological Effect Estimates

Quantifiable health benefits of the proposal may be related to ozone only, PM only, or both pollutants. Decreased worker productivity, respiratory hospital admissions for children under two, and school absences are related to ozone but not PM. PM-only health effects include premature mortality, nonfatal heart attacks, CB, acute bronchitis, upper and lower respiratory symptoms, asthma exacerbations, and work loss days.[16] Health effects related to both PM and ozone include hospital admissions, emergency room visits for asthma, and minor restricted activity days.

We relied on the available published scientific literature to ascertain the relationship between PM and ozone exposure and adverse human health effects. We evaluated studies using the selection criteria summarized in Table 4-6. These criteria include consideration of whether the study was peer reviewed, the match between the pollutant studied and the pollutant of interest, the study design and location, and characteristics of the study population, among other considerations. The selection of C-R functions for the benefits analysis is guided by the goal of achieving a balance between comprehensiveness and scientific defensibility.

Recently, the Health Effects Institute (HEI) reported findings by health researchers at Johns Hopkins University and others that have raised concerns about aspects of the statistical methods used in a number of recent time-series studies of short-term exposures to air pollution and health effects (Greenbaum, 2002). The estimates derived from the long-term exposure studies, which account for a major share of the economic benefits described in

[16]Evidence has been found linking ozone exposures with premature mortality independent of PM exposures. A recent analysis by Thurston and Ito (2001) reviewed previously published time-series studies of the effect of daily ozone levels on daily mortality and found that previous EPA estimates of the short-term mortality benefits of the ozone NAAQS (EPA, 1997) may have been underestimated by up to a factor of two, even when PM is controlled for in the models. In its September 2001 advisory on the draft analytical blueprint for the second Section 812 prospective analysis, the SAB cited the Thurston and Ito study as a significant advance in understanding the effects of ozone on daily mortality and recommended re-evaluation of the ozone mortality endpoint for inclusion in the next prospective study (EPA-SAB-COUNCIL-ADV-01-004, 2001). In addition, a recent World Health Organization (WHO) report found that "recent epidemiological studies have strengthened the evidence that there are short-term O3 effects on mortality and respiratory morbidity and provided further information on exposure-response relationships and effect modification." (WHO, 2002). Based on these new analyses and recommendations, the EPA is currently reevaluating ozone-related mortality for inclusion in the primary benefits analysis. The EPA is sponsoring three independent meta-analyses of the ozone-mortality epidemiology literature to inform a determination on inclusion of this important health endpoint. Upon completion and peer review of the meta-analyses, the EPA will make its determination on whether benefits of reductions in ozone-related mortality will be included in the benefits analysis for the final IAQR.

this chapter, are not affected. Similarly, the time-series studies employing generalized linear models (GLMs) or other parametric methods, as well as case-crossover studies, are not affected. As discussed in HEI materials provided to the EPA and to CASAC (Greenbaum, 2002), researchers working on the National Morbidity, Mortality, and Air Pollution Study (NMMAPS) found problems in the default "convergence criteria" used in Generalized Additive Models (GAM) and a separate issue first identified by Canadian investigators about the potential to underestimate standard errors in the same statistical package. Following identification of the GAM issue, a number of time-series studies were reanalyzed using alternative methods, typically GAM with more stringent convergence criteria and an alternative model such as generalized linear models (GLM) with natural smoothing splines, and the results of the reanalyses have been compiled and reviewed in a recent HEI publication (HEI, 2003a). In most, but not all, of the reanalyzed studies, it was found that risk estimates were reduced and confidence intervals increased with the use of GAM with more stringent convergence criteria or GLM analyses; however, the reanalyses generally did not substantially change the findings of the original studies, and the changes in risk estimates with alternative analysis methods were much smaller than the variation in effects across studies. The HEI review committee concluded the following:

a. Although the number of studies showing an association of PM with mortality was slightly smaller, the PM association persisted in the majority of studies.

b. In some of the large number of studies in which the PM association persisted, the estimates of PM effect were substantially smaller.

c. In the few studies in which investigators performed further sensitivity analyses, some showed marked sensitivity of the PM effect estimate to the degree of smoothing and/or the specification of weather (HEI, 2003b, p. 269)

Examination of the original studies used in our benefits analysis found that the health endpoints that are potentially affected by the GAM issues include reduced hospital admissions and reduced lower respiratory symptoms. For the IAQR, we have incorporated a

Table 4-6. Summary of Considerations Used in Selecting C-R Functions

Consideration	Comments
Peer reviewed research	Peer reviewed research is preferred to research that has not undergone the peer review process.
Study type	Among studies that consider chronic exposure (e.g., over a year or longer) prospective cohort studies are preferred over cross-sectional studies because they control for important individual-level confounding variables that cannot be controlled for in cross-sectional studies.
Study period	Studies examining a relatively longer period of time (and therefore having more data) are preferred, because they have greater statistical power to detect effects. More recent studies are also preferred because of possible changes in pollution mixes, medical care, and life style over time. However, when there are only a few studies available, studies from all years will be included.
Population attributes	The most technically appropriate measures of benefits would be based on impact functions that cover the entire sensitive population, but allow for heterogeneity across age or other relevant demographic factors. In the absence of effect estimates specific to age, sex, preexisting condition status, or other relevant factors, it may be appropriate to select effect estimates that cover the broadest population, to match with the desired outcome of the analysis, which is total national-level health impacts.
Study size	Studies examining a relatively large sample are preferred because they generally have more power to detect small magnitude effects. A large sample can be obtained in several ways, either through a large population, or through repeated observations on a smaller population, i.e. through a symptom diary recorded for a panel of asthmatic children.
Study location	U.S. studies are more desirable than non-U.S. studies because of potential differences in pollution characteristics, exposure patterns, medical care system, population behavior and life style.
Pollutants included in model	When modeling the effects of ozone and PM (or other pollutant combinations) jointly, it is important to use properly specified impact functions that include both pollutants. Use of single pollutant models in cases where both pollutants are expected to affect a health outcome can lead to double-counting when pollutants are correlated.
Measure of PM	For this analysis, impact functions based on $PM_{2.5}$ are preferred to PM_{10} because the IAQR will regulate emissions of PM2.5 precursors and air quality modeling was conducted for this size fraction of PM. Where $PM_{2.5}$ functions are not available, PM_{10} functions are used as surrogates, recognizing that there will be potential downward (upward) biases if the fine fraction of PM_{10} is more (less) toxic than the coarse fraction.
Economically valuable health effects	Some health effects, such as forced expiratory volume and other technical measurements of lung function, are difficult to value in monetary terms. These health effects are not quantified in this analysis.
Non-overlapping endpoints	Although the benefits associated with each individual health endpoint may be analyzed separately, care must be exercised in selecting health endpoints to include in the overall benefits analysis because of the possibility of double counting of benefits.

number of studies that have been updated to correct for the GAM issue, including Ito et al. (2003) for respiratory-related hospital admissions (COPD and pneumonia), Shepard et al. (2003) for respiratory-related hospital admissions (asthma), Moolgavkar (2003) for cardiovascular-related hospital admissions (ICD codes 390-429), and Ito et al. (2003) for cardiovascular-related hospital admissions (ischemic heart disease, dysrhythmia, and heart failure). Several additional hospital admissions-related studies have not yet been formally updated to correct for the GAM issue. These include the lower respiratory symptoms study and hospital admissions for respiratory and cardiovascular causes in populations aged 20 to 64. However, as discussed above, available evidence suggests that the errors introduced into effect estimates due to the GAM issue should not significantly affect incidence results.

It is important to reiterate that the estimates derived from the long-term exposure studies, which account for a major share of the economic benefits described in this chapter, are not affected by the GAM issue. Similarly, the time-series studies employing GLMs or other parametric methods, as well as case-crossover studies, are not affected.

Although a broad range of serious health effects has been associated with exposure to elevated ozone and PM levels (as noted for example in Table 4-1 and described more fully in the ozone and PM Criteria Documents (EPA, 1996a, 1996b)), we include only a subset of health effects in this quantified benefit analysis. Health effects are excluded from this analysis for three reasons: the possibility of double counting (such as hospital admissions for specific respiratory diseases); uncertainties in applying effect relationships based on clinical studies to the affected population; or a lack of an established relationship between the health effect and pollutant in the published epidemiological literature.

In general, the use of results from more than a single study can provide a more robust estimate of the relationship between a pollutant and a given health effect. However, there are often differences between studies examining the same endpoint, making it difficult to pool the results in a consistent manner. For example, studies may examine different pollutants or different age groups. For this reason, we consider very carefully the set of studies available examining each endpoint and select a consistent subset that provides a good balance of population coverage and match with the pollutant of interest. In many cases, either because of a lack of multiple studies, consistency problems, or clear superiority in the quality or comprehensiveness of one study over others, a single published study is selected as the basis of the effect estimate.

When several effect estimates for a pollutant and a given health endpoint have been selected, they are quantitatively combined or pooled to derive a more robust estimate of the relationship. The benefits Technical Support Document (TSD) completed for the nonroad diesel rulemaking provides details of the procedures used to combine multiple impact functions (Abt Associates, 2003). In general, we use fixed or random effects models to pool estimates from different studies of the same endpoint. Fixed effects pooling simply weights each study's estimate by the inverse variance, giving more weight to studies with greater statistical power (lower variance). Random effects pooling accounts for both within-study variance and between-study variability, due, for example, to differences in population susceptibility. We use the fixed effects model as our null hypothesis and then determine whether the data suggest that we should reject this null hypothesis, in which case we would use the random effects model.[17] Pooled impact functions are used to estimate hospital admissions (PM), school absence days (ozone), lower respiratory symptoms (PM), asthma exacerbations (PM), and asthma-related emergency room visits (ozone). For more details on methods used to pool incidence estimates, see the benefits TSD for the nonroad diesel rulemaking (Abt Associates, 2003).

Effect estimates for a pollutant and a given health endpoint are applied consistently across all locations nationwide. This applies to both impact functions defined by a single effect estimate and those defined by a pooling of multiple effect estimates. Although the effect estimate may, in fact, vary from one location to another (e.g., due to differences in population susceptibilities or differences in the composition of PM), location-specific effect estimates are generally not available.

The specific studies from which effect estimates for the primary analysis are drawn are included in Table 4-7.

Premature Mortality. Both long- and short-term exposures to ambient levels of air pollution have been associated with increased risk of premature mortality. The size of the mortality risk estimates from these epidemiological studies, the serious nature of the effect itself, and the high monetary value ascribed to prolonging life make mortality risk reduction the most important health endpoint quantified in this analysis.

[17]The fixed effects model assumes that there is only one pollutant coefficient for the entire modeled area. The random effects model assumes that different studies are estimating different parameters; therefore, there may be a number of different underlying pollutant coefficients.

Epidemiological analyses have consistently linked air pollution, especially PM, with excess mortality. Although a number of uncertainties remain to be addressed by continued research (NRC, 1998), a substantial body of published scientific literature documents the correlation between elevated PM concentrations and increased mortality rates. Community epidemiological studies that have used both short-term and long-term exposures and response have been used to estimate PM/ mortality relationships. Short-term studies use a time-series approach to relate short-term (often day-to-day) changes in PM concentrations and changes in daily mortality rates up to several days after a period of elevated PM concentrations. Long-term studies examine the potential relationship between community-level PM exposures over multiple years and community-level annual mortality rates.

Table 4-7. Endpoints and Studies Used to Calculate Total Monetized Health Benefits

Endpoint	Pollutant	Study	Study Population
Premature Mortality			
Premature Mortality— Long-term exposure, all-cause	$PM_{2.5}$	Pope et al. (2002)	>29 years
Premature Mortality— Long-term exposure, all-cause	$PM_{2.5}$	Woodruff et al., 1997	Infant (<1 yr)
Chronic Illness			
Chronic Bronchitis	$PM_{2.5}$	Abbey, et al. (1995)	> 26 years
Non-fatal Heart Attacks	$PM_{2.5}$	Peters et al. (2001)	Adults
Hospital Admissions			
Respiratory	Ozone	Pooled estimate: Schwartz (1995) - ICD 460-519 (all resp) Schwartz (1994a, 1994b) - ICD 480-486 (pneumonia) Moolgavkar et al. (1997) - ICD 480-487 (pneumonia) Schwartz (1994b) - ICD 491-492, 494-496 (COPD) Moolgavkar et al (1997) - ICD 490-496 (COPD)	> 64 years
	Ozone	Burnett et al. (2001)	< 2 years
	$PM_{2.5}$	Pooled estimate: Moolgavkar (2003) - ICD 490-496 (COPD) Ito (2003) - ICD 490-496 (COPD)	> 64 years
	$PM_{2.5}$	Moolgavkar (2000) - ICD 490-496 (COPD)	20-64 years
	$PM_{2.5}$	Ito (2003) - ICD 480-486 (pneumonia)	> 64 years
	$PM_{2.5}$	Sheppard, et al. (2003) - ICD 493 (asthma)	< 65 years
Cardiovascular	$PM_{2.5}$	Pooled estimate: Moolgavkar (2003) - ICD 390-429 (all cardiovascular) Ito (2003) - ICD 410-414, 427-428 (ischemic heart disease, dysrhythmia, heart failure)	> 64 years
	$PM_{2.5}$	Moolgavkar (2000) - ICD 390-429 (all cardiovascular)	20-64 years
Asthma-Related ER Visits	Ozone	Pooled estimate: Weisel et al. (1995), Cody et al. (1992), Stieb et al. (1996)	All ages
	$PM_{2.5}$	Norris et al. (1999)	0-18 years

(continued)

Table 4-7. Endpoints and Studies Used to Calculate Total Monetized Health Benefits (continued)

Endpoint	Pollutant	Study	Study Population
Other Health Endpoints			
Acute Bronchitis	$PM_{2.5}$	Dockery et al. (1996)	8-12 years
Upper Respiratory Symptoms	PM_{10}	Pope et al. (1991)	Asthmatics, 9-11 years
Lower Respiratory Symptoms	$PM_{2.5}$	Schwartz and Neas (2000)	7-14 years
Asthma Exacerbations	$PM_{2.5}$	Pooled estimate: Ostro et al. (2001) (cough, wheeze and shortness of breath) Vedal et al. (1998) Cough	6-18 years[a]
Work Loss Days	$PM_{2.5}$	Ostro (1987)	18-65 years
School Absence Days	Ozone	Pooled estimate: Gilliland et al (2001) Chen et al (2000)	 9-10 years 6-11 years
Worker Productivity	Ozone	Crocker and Horst (1981)	Outdoor workers, 18-65
Minor Restricted Activity Days	$PM_{2.5}$, Ozone	Ostro and Rothschild (1989)	18-65 years

[a] The original study populations were 8 to 13 for the Ostro et al (2001) study and 6 to 13 for the Vedal et al. (1998) study. Based on advice from the SAB-HES, we have extended the applied population to 6 to 18, reflecting the common biological basis for the effect in children in the broader age group.

Researchers have found statistically significant associations between PM and premature mortality using both types of studies. In general, the risk estimates based on the long-term exposure studies are larger than those derived from short-term studies. Cohort analyses are better able to capture the full public health impact of exposure to air pollution over time

(Kunzli, 2001; NRC, 2002). This section discusses some of the issues surrounding the estimation of premature mortality.

Over a dozen studies have found significant associations between various measures of long-term exposure to PM and elevated rates of annual mortality, beginning with Lave and Seskin (1977). Most of the published studies found positive (but not always statistically significant) associations with available PM indices such as total suspended particles (TSP), however, exploration of alternative model specifications sometimes raised questions about causal relationships (e.g., Lipfert, [1989]). These early "cross-sectional" studies (e.g., Lave and Seskin [1977]; Ozkaynak and Thurston [1987]) were criticized for a number of methodological limitations, particularly for inadequate control at the individual level for variables that are potentially important in causing mortality, such as wealth, smoking, and diet. More recently, several long-term studies have been published that use improved approaches and appear to be consistent with the earlier body of literature. These new "prospective cohort" studies reflect a significant improvement over the earlier work because they include individual-level information with respect to health status and residence. The most extensive study and analyses has been based on data from two prospective cohort groups, often referred to as the Harvard "Six-City Study" (Dockery et al., 1993) and the "American Cancer Society or ACS study" (Pope et al., 1995); these studies have found consistent relationships between fine particle indicators and premature mortality across multiple locations in the United States. A third major data set comes from the California based 7th Day Adventist Study (e.g., Abbey et al, 1999), which reported associations between long-term PM exposure and mortality in men. Results from this cohort, however, have been inconsistent and the air quality results are not geographically representative of most of the United States. More recently, a cohort of adult male veterans diagnosed with hypertension has been examined (Lipfert et al., 2000). The characteristics of this group differ from the cohorts in the ACS, Six-Cities, and 7th Day Adventist studies with respect to income, race, health status, and smoking status. Unlike previous long-term analyses, this study found some associations between mortality and ozone but found inconsistent results for PM indicators. Because of the selective nature of the population in the veteran's cohort, which may have resulted in estimates of relative risk that are biased relative to a relative risk for the general

population, we have chosen not to include any effect estimates from the Lipfert et al. (2000) study in our benefits assessment.[18]

Given their consistent results and broad geographic coverage, the Six-City and ACS data have been particularly important in benefits analyses. The credibility of these two studies is further enhanced by the fact that they were subject to extensive reexamination and reanalysis by an independent team of scientific experts commissioned by HEI (Krewski et al., 2000). The final results of the reanalysis were then independently peer reviewed by a Special Panel of the HEI Health Review Committee. The results of these reanalyses confirmed and expanded those of the original investigators. This intensive independent reanalysis effort was occasioned both by the importance of the original findings as well as concerns that the underlying individual health effects information has never been made publicly available.

The HEI re-examination lends credibility to the original studies and highlights sensitivities concerning the relative impact of various pollutants, the potential role of education in mediating the association between pollution and mortality, and the influence of spatial correlation modeling. Further confirmation and extension of the overall findings using more recent air quality and a longer follow-up period for the ACS cohort was recently published in the *Journal of the American Medical Association* (Pope et al., 2002).

In developing and improving the methods for estimating and valuing the potential reductions in mortality risk over the years, the EPA has consulted with the SAB-HES. That panel recommended use of long-term prospective cohort studies in estimating mortality risk reduction (EPA-SAB-COUNCIL-ADV-99-005, 1999). This recommendation has been

[18]The EPA recognizes that the ACS cohort also is not completely representative of the demographic mix in the general population. The ACS cohort is almost entirely white, and has higher income and education levels relative to the general population. The EPA's approach to this problem is to match populations based on the potential for demographic characteristics to modify the effect of air pollution on mortality risk. Thus, for the various ACS-based models, we are careful to apply the effect estimate only to ages matching those in the original studies, because age has a potentially large modifying impact on the effect estimate, especially when younger individuals are excluded from the study population. For the Lipfert analysis, the applied population should be limited to that matching the sample used in the analysis. This sample was all male, veterans, and diagnosed hypertensive. There are also a number of differences between the composition of the sample and the general population, including a higher percentage of African Americans (35 percent), and a much higher percentage of smokers (81 percent former smokers, 57 percent current smokers) than the general population (12 percent African American, 24 percent current smokers). These composition differences cannot be controlled for, but should be recognized as adding to the potential extrapolation bias. The EPA recognizes the difficulty in controlling for composition of income and education levels. However, in or out criterion such as age, veteran status, hypertension, race and sex are all controllable by applying filters to the population data. The EPA has traditionally only controlled for age, because the ACS study used only age as a screen.

confirmed by a recent report from the National Research Council, which stated that "it is essential to use the cohort studies in benefits analysis to capture all important effects from air pollution exposure" (NAS, 2002, p. 108). More specifically, the SAB recommended emphasis on the ACS study because it includes a much larger sample size and longer exposure interval and covers more locations (e.g., 50 cities compared to the Six Cities Study) than other studies of its kind. As explained in the regulatory impact analysis for the Heavy-Duty Engine/Diesel Fuel rule (EPA, 2000a), more recent EPA benefits analyses have relied on an improved specification of the ACS cohort data that was developed in the HEI reanalysis (Krewski et al., 2000). The latest reanalysis of the ACS cohort data (Pope et al., 2002), provides additional refinements to the analysis of PM-related mortality by (a) extending the follow-up period for the ACS study subjects to 16 years, which triples the size of the mortality data set; (b) substantially increasing exposure data, including consideration for cohort exposure to PM2.5 following implementation of PM2.5 standard in 1999; (c) controlling for a variety of personal risk factors including occupational exposure and diet; and (d) using advanced statistical methods to evaluate specific issues that can adversely affect risk estimates including the possibility of spatial autocorrelation of survival times in communities located near each other. Because of these refinements, the SAB- HES recommends using the Pope et al. (2002) study as the basis for the primary mortality estimate for adults and suggests that alternate estimates of mortality generated using other cohort and time series studies could be included as part of the sensitivity analysis (SAB-HES, 2003).

The SAB-HES also recommended using the estimated relative risks from the Pope et al. (2002) study based on the average exposure to PM2.5, measured by the average of two PM2.5 measurements, over the periods 1979-1983, and 1999-2000. In addition to relative risks for all-cause mortality, the Pope et al. (2002) study provides relative risks for cardiopulmonary, lung cancer, and all other cause mortality. Because of concerns regarding the statistical reliability of the all-other cause mortality relative risk estimates, we calculate mortality impacts for the primary analysis based on the all-cause relative risk. However, we provide separate estimates of cardiopulmonary and lung cancer deaths to show how these important causes of death are affected by reductions in PM2.5.

In previous RIAs, infant mortality has not been evaluated as part of the primary analysis. Instead, benefits estimates related to reduced infant mortality have been included as part of the sensitivity analysis for RIAs. However, recently published studies have strengthened the case for an association between PM exposure and respiratory inflamation and infection leading to premature mortality in children under 5 years of age. Specifically, the SAB- HES

noted the release of the World Health Organization Global Burden of Disease Study focusing on ambient air, which cites several recently published time-series studies relating daily PM exposure to mortality in children (SAB-HES, 2003). The SAB-HES also cites the study by Belanger et al. (2003) as corroborating findings linking PM exposure to increased respiratory inflamation and infections in children. Recently, a study by Chay and Greenstone (2003) found that reductions in TSP caused by the recession of 1981-1982 were related to reductions in infant mortality at the county level. With regard to the cohort study conducted by Woodruff et al. (1997), the SAB- HES notes several strengths of the study, including the use of a larger cohort drawn from a large number of metropolitan areas and efforts to control for a variety of individual risk factors in infants (e.g., maternal educational level, maternal ethnicity, parental marital status, and maternal smoking status). Based on these findings, the SAB-HES recommends that the EPA incorporate infant mortality into the primary benefits estimate and that infant mortality be evaluated using a impact function developed from the Woodruff et al. (1997) study (SAB-HES, 2003).

Chronic Bronchitis. CB is characterized by mucus in the lungs and a persistent wet cough for at least 3 months a year for several years in a row. CB affects an estimated 5 percent of the U.S. population (American Lung Association, 1999). A limited number of studies have estimated the impact of air pollution on new incidences of CB. Schwartz (1993) and Abbey et al.(1995) provide evidence that long-term PM exposure gives rise to the development of CB in the United States. Because the Inter-State Air Quality regulations are expected to reduce primarily $PM_{2.5}$, this analysis uses only the Abbey et al (1995) study, because it is the only study focusing on the relationship between $PM_{2.5}$ and new incidences of CB.

Nonfatal Myocardial Infarctions (heart attacks). Nonfatal heart attacks have been linked with short-term exposures to PM2.5 in the United States (Peters et al., 2001) and other countries (Poloniecki et al. ,1997). We use a recent study by Peters et al. (2001) as the basis for the impact function estimating the relationship between PM2.5 and nonfatal heart attacks. Peters et al. is the only available U.S. study to provide a specific estimate for heart attacks. Other studies, such as Samet et al. (2000) and Moolgavkar et al. (2000), show a consistent relationship between all cardiovascular hospital admissions, including for nonfatal heart attacks, and PM. Given the lasting impact of a heart attack on longer-term health costs and earnings, we choose to provide a separate estimate for nonfatal heart attacks based on the single available U.S. effect estimate. The finding of a specific impact on heart attacks is consistent with hospital admission and other studies showing relationships between fine

particles and cardiovascular effects both within and outside the United States. These studies provide a weight of evidence for this type of effect. Several epidemiologic studies (Liao et al., 1999; Gold et al., 2000; Magari et al., 2001) have shown that heart rate variability (an indicator of how much the heart is able to speed up or slow down in response to momentary stresses) is negatively related to PM levels. Heart rate variability is a risk factor for heart attacks and other coronary heart diseases (Carthenon et a.l, 2002; Dekker et al., 2000; Liao et al., 1997, Tsuji et al., 1996). As such, significant impacts of PM on heart rate variability are consistent with an increased risk of heart attacks.

Hospital and Emergency Room Admissions. Because of the availability of detailed hospital admission and discharge records, there is an extensive body of literature examining the relationship between hospital admissions and air pollution. Because of this, many of the hospital admission endpoints use pooled impact functions based on the results of a number of studies. In addition, some studies have examined the relationship between air pollution and emergency room (ER) visits. Because most ER visits do not result in an admission to the hospital (the majority of people going to the ER are treated and return home), we treat hospital admissions and ER visits separately, taking account of the fraction of ER visits that are admitted to the hospital.

Hospital admissions require the patient to be examined by a physician and, on average, may represent more serious incidents than ER visits. The two main groups of hospital admissions estimated in this analysis are respiratory admissions and cardiovascular admissions. There is not much evidence linking ozone or PM with other types of hospital admissions. The only type of ER visits that have been consistently linked to ozone and PM in the United States are asthma-related visits.

To estimate avoided incidences of cardiovascular hospital admissions associated with PM2.5, we use studies by Moolgavkar (2003) and Ito et al. (2003). There are additional published studies showing a statistically significant relationship between PM10 and cardiovascular hospital admissions. However, given that the preliminary control options we are analyzing are expected to reduce primarily PM2.5, we have chosen to focus on the two studies focusing on PM2.5. Both of these studies provide an effect estimate for populations over 65, allowing us to pool the impact functions for this age group. Only Moolgavkar

(2000) provided a separate effect estimate for populations 20 to 64.[19] Total cardiovascular hospital admissions are thus the sum of the pooled estimate for populations over 65 and the single study estimate for populations 20 to 64. Cardiovascular hospital admissions include admissions for myocardial infarctions. To avoid double counting benefits from reductions in myocardial infarctions when applying the impact function for cardiovascular hospital admissions, we first adjusted the baseline cardiovascular hospital admissions to remove admissions for myocardial infarctions.

To estimate total avoided incidences of respiratory hospital admissions, we use impact functions for several respiratory causes, including chronic obstructive pulmonary disease (COPD), pneumonia, and asthma. As with cardiovascular admissions, there are additional published studies showing a statistically significant relationship between PM10 and respiratory hospital admissions. We use only those focusing on PM2.5. Both Moolgavkar (2000) and Ito et al. (2003) provide effect estimates for COPD in populations over 65, allowing us to pool the impact functions for this group. Only Moolgavkar (2000) provided a separate effect estimate for populations 20 to 64[20]. Total COPD hospital admissions are thus the sum of the pooled estimate for populations over 65 and the single study estimate for populations 20 to 64. Only Ito et al (2003) estimated pneumonia, and only for the population 65 and older. In addition, Sheppard et al. (2003) provided an effect estimate for asthma hospital admissions for populations under age 65. Total avoided incidences of PM-related respiratory-related hospital admissions is the sum of COPD, pneumonia, and asthma admissions.

To estimate the effects of PM air pollution reductions on asthma-related ER visits, we use the effect estimate from a study of children 18 and under by Norris et al. (1999). As noted earlier, there is another study by Schwartz examining a broader age group (less than 65), but the Schwartz study focused on PM10 rather than PM2.5. We selected the Norris et al. (1999)

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[19]Note that the Moolgavkar (2000) study has not been updated to reflect the more stringent GAM convergence criteria. However, given that no other estimates are available for this age group, we have chosen to use the existing study. Given the very small (<5 percent) difference in the effect estimates for 65 and older cardiovascular hospital admissions between the original and reanalyzed results, we do not expect there to be much bias introduced by this choice.

[20]Note that the Moolgavkar (2000) study has not been updated to reflect the more stringent GAM convergence criteria. However, given that no other estimates are available for this age group, we have chosen to use the existing study. Given the very small (<10 percent) difference in the effect estimates for 65 and older COPD hospital admissions between the original and reanalyzed results, we do not expect there to be much bias introduced by this choice.

effect estimate because it better matched the pollutant of interest. Because children tend to have higher rates of hospitalization for asthma relative to adults under 65, we will likely capture the majority of the impact of PM2.5 on asthma ER visits in populations under 65, although there may still be significant impacts in the adult population under 65.

To estimate avoided incidences of respiratory hospital admissions associated with ozone, we use a number of studies examining hospital admissions for a range of respiratory illnesses, including pneumonia and COPD. Two age groups, adults over 65 and children under 2, are examined. For adults over 65, Schwartz (1995) provides effect estimates for two different cities relating ozone and hospital admissions for all respiratory causes (defined as ICD codes 460-519). Impact functions based on these studies are pooled first before being pooled with other studies. Two studies (Moolgavkar et al., 1997; Schwartz, 1994a) examined ozone and pneumonia hospital admissions in Minneapolis. One additional study (Schwartz, 1994b) examined ozone and pneumonia hospital admissions in Detroit. The impact functions for Minneapolis are pooled together first, and the resulting impact function is then pooled with the impact function for Detroit. This avoids assigning too much weight to the information coming from one city. For COPD hospital admissions, there are two available studies, Moolgavkar et al. (1997), conducted in Minneapolis, and Schwartz (1994b), conducted in Detroit. These two studies are pooled together. To estimate total respiratory hospital admissions for adults over 65, COPD admissions are added to pneumonia admissions, and the result is pooled with the Schwartz (1995) estimate of total respiratory admissions. Burnett et al. (2001) is the only study providing an effect estimate for respiratory hospital admissions in children under 2.

Acute Health Events and School/Work Loss Days. As indicated in Table 4-1, in addition to mortality, chronic illness, and hospital admissions, a number of acute health effects not requiring hospitalization are associated with exposure to ambient levels of ozone and PM. The sources for the effect estimates used to quantify these effects are described below.

Around 4 percent of U.S. children between ages 5 and 17 experience episodes of acute bronchitis annually (American Lung Association, 2002). Acute bronchitis is characterized by coughing, chest discomfort, slight fever, and extreme tiredness, lasting for a number of days. According to the MedlinePlus medical encyclopedia,[21] with the exception of cough, most acute bronchitis symptoms abate within 7 to 10 days. Incidence of episodes of acute

[21]See http://www.nlm.nih.gov/medlineplus/ency/article/000124.htm, accessed January 2002.

bronchitis in children between the ages of 5 and 17 are estimated using an effect estimate developed from Dockery et al. (1996).

Incidences of lower respiratory symptoms (e.g., wheezing, deep cough) in children aged 7 to 14 are estimated using an effect estimate from Schwartz and Neas (2000).

Because asthmatics have greater sensitivity to stimuli (including air pollution), children with asthma can be more susceptible to a variety of upper respiratory symptoms (e.g., runny or stuffy nose; wet cough; and burning, aching, or red eyes). Research on the effects of air pollution on upper respiratory symptoms has thus focused on effects in asthmatics. Incidences of upper respiratory symptoms in asthmatic children aged 9 to 11 are estimated using an effect estimate developed from Pope et al. (1991).

Health effects from air pollution can also result in missed days of work (either from personal symptoms or from caring for a sick family member). Work loss days due to PM2.5 are estimated using an effect estimate developed from Ostro (1987). Children may also be absent from school due to respiratory or other diseases caused by exposure to air pollution. Most studies examining school absence rates have found little or no association with PM2.5, but several studies have found a significant association between ozone levels and school absence rates. We use two recent studies, Gilliland et al. (2001) and Chen et al. (2000), to estimate changes in absences (school loss days) due to changes in ozone levels. The Gilliland et al. study estimated the incidence of new periods of absence, while the Chen et al. study examined absence on a given day. We convert the Gilliland estimate to days of absence by multiplying the absence periods by the average duration of an absence. We estimate an average duration of school absence of 1.6 days by dividing the average daily school absence rate from Chen et al. (2000) and Ransom and Pope (1992) by the episodic absence rate from Gilliland et al. (2001). This provides estimates from Chen et al. (2000) and Gilliland et al. (2000), which can be pooled to provide an overall estimate.

Minor restricted activity days (MRAD) result when individuals reduce most usual daily activities and replace them with less strenuous activities or rest, yet not to the point of missing work or school. For example, a mechanic who would usually be doing physical work most of the day will instead spend the day at a desk doing paper and phone work due to difficulty breathing or chest pain. The effect of PM2.5 and ozone on MRAD is estimated using an effect estimate derived from Ostro and Rothschild (1989).

In previous RIAs, we have not included estimates of asthma exacerbations in the asthmatic population in the primary analysis because of concerns over double counting of

benefits and difficulties in differentiating asthma symptoms for purposes of first developing impact functions that cover distinct endpoints and then establishing the baseline incidence estimates required for predicting incidence reductions. Concerns over double counting stem from the fact that studies of the general population also include asthmatics, so estimates based solely on the asthmatic population cannot be directly added to the general population numbers without double counting. In one specific case (upper respiratory symptoms in children), the only study available was limited to asthmatic children, so this endpoint can be readily included in the calculation of total benefits. However, other endpoints, such as lower respiratory symptoms and MRADs, are estimated for the total population that includes asthmatics. Therefore, to simply add predictions of asthma-related symptoms generated for the population of asthmatics to these total population-based estimates could result in double counting, especially if they evaluate similar endpoints. The SAB-HES, in commenting on the analytical blueprint for 812 acknowledged these challenges in evaluating asthmatic symptoms and appropriately adding them into the primary analysis (SAB-HES, 2003). However, despite these challenges, the SAB-HES recommends the addition of asthma-related symptoms (i.e., asthma exacerbations) to the primary analysis, provided that the studies use the panel study approach and that they have comparable design and baseline frequencies in both asthma prevalence and exacerbation rates. Note also, that the SAB-HES, while supporting the incorporation of asthma exacerbation estimates, does not believe that the association between ambient air pollution, including ozone and PM, and the new onset of asthma is sufficiently strong to support inclusion of this asthma-related endpoint in the primary estimate. For the IAQR, we have followed the SAB-HES recommendations regarding asthma exacerbations in developing the primary estimate. To prevent double counting, we are focusing the estimation on asthma exacerbations occurring in children and are excluding adults from the calculation. Asthma exacerbations occurring in adults are assumed to be captured in the general population endpoints such as work loss days and MRADs. Consequently, if we had included an adult-specific asthma exacerbation estimate, we would likely double count incidence for this endpoint. However, because the general population endpoints do not cover children (with regard to asthmatic effects), an analysis focused specifically on asthma exacerbations for children (6 to 18 years of age) could be conducted without concern for double counting.

To characterize asthma exacerbations in children, we selected two studies (Ostro et al., 2001 and Vedal et al., 1998) that followed panels of asthmatic children. Ostro et al. (2001) followed a group of 138 African-American children in Los Angeles for 13 weeks, recording daily occurrences of respiratory symptoms associated with asthma exacerbations (e.g.,

shortness of breath, wheeze, and cough). This study found a statistically significant association between PM2.5, measured as a 12-hour average, and the daily prevalence of shortness of breath and wheeze endpoints. Although the association was not statistically significant for cough, the results were still positive and close to significance; consequently, we decided to include this endpoint, along with shortness of breath and wheeze, in generating incidence estimates (see below). Vedal et al. (1998) followed a group of elementary school children, including 74 asthmatics, located on the west coast of Vancouver Island for 18 months including measurements of daily peak expiratory flow (PEF) and the tracking of respiratory symptoms (e.g., cough, phlegm, wheeze, chest tightness) through the use of daily diaries. Association between PM10 and respiratory symptoms for the asthmatic population was only reported for two endpoints: cough and PEF. Because it is difficult to translate PEF measures into clearly defined health endpoints that can be monetized, we only included the cough-related effect estimate from this study in quantifying asthma exacerbations. We employed the following pooling approach in combining estimates generated using effect estimates from the two studies to produce a single asthma exacerbation incidence estimate. First, we pooled the separate incidence estimates for shortness of breath, wheeze, and cough generated using effect estimates from the Ostro et al study, because each of these endpoints is aimed at capturing the same overall endpoint (asthma exacerbations) and there could be overlap in their predictions. The pooled estimate from the Ostro et al. study is then pooled with the cough-related estimate generated using the Vedal study. The rationale for this second pooling step is similar to the first; both studies are attempting to quantify the same overall endpoint (asthma exacerbations).

Additional epidemiological studies are available for characterizing asthma-related health endpoints (the full list of epidemiological studies considered for modeling asthma-related incidence are presented in Table 4-8). However, based on recommendations from the SAB-HES, we decided not to use these additional studies in generating the primary estimate. In particular, the Yu et al. (2000) estimates show a much higher baseline incidence rate than other studies, which may lead to an overstatement of the expected impacts in the overall asthmatic population. The Whittemore and Korn (1980) study did not use a well-defined endpoint, instead focusing on a respondent-defined "asthma attack." Other studies looked at respiratory symptoms in asthmatics but did not focus on specific exacerbations of asthma.

4.1.5.2 Uncertainties Associated with Health Impact Functions

Within-Study Variation. Within-study variation refers to the precision with which a given study estimates the relationship between air quality changes and health effects. Health effects

studies provide both a "best estimate" of this relationship plus a measure of the statistical uncertainty of the relationship. This size of this uncertainty depends on factors such as the number of subjects studied and the size of the effect being measured. The results of even the most well-designed epidemiological studies are characterized by this type of uncertainty, though well-designed studies typically report narrower uncertainty bounds around the best estimate than do studies of lesser quality. In selecting health endpoints, we generally focus on endpoints where a statistically significant relationship has been observed in at least some studies, although we may pool together results from studies with both statistically significant and insignificant estimates to avoid selection bias.

Across-Study Variation. Across-study variation refers to the fact that different published studies of the same pollutant/health effect relationship typically do not report identical findings; in some instances the differences are substantial. These differences can exist even between equally reputable studies and may result in health effect estimates that vary considerably. Across-study variation can result from two possible causes. One possibility is that studies report different estimates of the single true relationship between a given pollutant and a health effect due to differences in study design, random chance, or other factors. For example, a hypothetical study conducted in New York and one conducted in Seattle may report different C-R functions for the relationship between PM and mortality, in part because of differences between these two study populations (e.g., demographics,

Table 4-8. Studies Examining Health Impacts in the Asthmatic Population Evaluated for Use in the Benefits Analysis

Endpoint	Definition	Pollutant	Study	Study Population
Asthma Attack Indicators[1]				
Shortness of breath	Prevalence of shortness of breath; incidence of shortness of breath	$PM_{2.5}$	Ostro et al. (2001)	African-American asthmatics, 8-13
Cough	Prevalence of cough; incidence of cough	$PM_{2.5}$	Ostro et al. (2001)	African-American asthmatics, 8-13
Wheeze	Prevalence of wheeze; incidence of wheeze	$PM_{2.5}$	Ostro et al. (2001)	African-American asthmatics, 8-13
Asthma exacerbation	≥ 1 mild asthma symptom: wheeze, cough, chest tightness, shortness of breath)	PM_{10}, $PM_{1.0}$	Yu et al. (2000)	Asthmatics, 5-13
Cough	Prevalence of cough	PM_{10}	Vedal et al. (1998)	Asthmatics, 6-13
Other symptoms/illness endpoints				
Upper respiratory symptoms	≥ 1 of the following: runny or stuffy nose; wet cough; burning, aching, or red eyes	PM_{10}	Pope et al. (1991)	Asthmatics 9-11
Moderate or worse asthma	Probability of moderate (or worse) rating of overall asthma status	$PM_{2.5}$	Ostro et al. (1991)	Asthmatics, all ages
Acute bronchitis	≥ 1 episodes of bronchitis in the past 12 months	$PM_{2.5}$	McConnell et al. (1999)	Asthmatics, 9-15*
Phlegm	"Other than with colds, does this child usually seem congested in the chest or bring up phlegm?"	$PM_{2.5}$	McConnell et al. (1999)	Asthmatics, 9-15*
Asthma attacks	Respondent-defined asthma attack	PM2.5, ozone	Whittemore and Korn (1980)	Asthmatics, all ages

activity patterns). Alternatively, study results may differ because these two studies are in fact estimating different relationships; that is, the same reduction in PM in New York and Seattle may result in different reductions in premature mortality. This may result from a number of factors, such as differences in the relative sensitivity of these two populations to PM pollution and differences in the composition of PM in these two locations. In either case, where we identified multiple studies that are appropriate for estimating a given health effect, we generated a pooled estimate of results from each of those studies.

Application of C-R Relationship Nationwide. Regardless of the use of impact functions based on effect estimates from a single epidemiological study or multiple studies, each impact function was applied uniformly throughout the United States to generate health benefit estimates. However, to the extent that pollutant/health effect relationships are region-specific, applying a location-specific impact function at all locations in the United States may result in overestimates of health effect changes in some locations and underestimates of health effect changes in other locations. It is not possible, however, to know the extent or direction of the overall effect on health benefit estimates introduced by application of a single impact function to the entire United States. This may be a significant uncertainty in the analysis, but the current state of the scientific literature does not allow for a region-specific estimation of health benefits.[22]

Extrapolation of Impact Functions Across Populations. Epidemiological studies often focus on specific age ranges, either due to data availability limitations (e.g., most hospital admission data come from Medicare records, which are limited to populations 65 and older), or to simplify data collection (e.g., some asthma symptom studies focus on children at summer camps, which usually have a limited age range). We have assumed for the primary analysis that most impact functions should be applied only to those populations with ages that strictly match the populations in the underlying epidemiological studies. However, in many cases, there is no biological reason why the observed health effect would not also occur in other populations within a reasonable range of the studied population. For example, Dockery et al. (1996) examined acute bronchitis in children aged 8 to 12. There is no biological reason to expect a very different response in children aged 6 or 14. By excluding populations outside the range in the studies, we may be underestimating the health impact in the overall population. In response to recommendations from the SAB-HES, where there appears to be a

[22]Although we are not able to use region-specific effect estimates, we use region-specific baseline incidence rates where available. This allows us to take into account regional differences in health status, which can have a significant impact on estimated health benefits.

reasonable physiological basis for expanding the age group associated with a specific effect estimate beyond the study population to cover the full age group (e.g., expanding from a study population of 7 to 11 year olds to the full 6to 18 year child age group), we have done so and used those expanded incidence estimates in the primary analysis.

Uncertainties in the PM Mortality Relationship. Health researchers have consistently linked air pollution, especially PM, with excess mortality. A substantial body of published scientific literature recognizes a correlation between elevated PM concentrations and increased mortality rates. However, much about this relationship is still uncertain. These uncertainties include the following:

- Causality: A substantial number of published epidemiological studies find an association between elevated PM concentrations and increased mortality rates; however, these epidemiological studies are not designed to definitively prove causation. For the analysis of the IAQ rulemaking, we assumed a causal relationship between exposure to elevated PM and premature mortality, based on the consistent evidence of a correlation between PM and mortality reported in the substantial body of published scientific literature.

- Other Pollutants: PM concentrations are correlated with the concentrations of other criteria pollutants, such as ozone and CO, and it is unclear how much each of these pollutants may influence mortality rates. Recent studies (see Thurston and Ito [2001]) have explored whether ozone may have mortality effects independent of PM, but we do not view the evidence as conclusive at this time. The EPA is currently evaluating the epidemiological literature on the relationship between ozone and mortality and will determine whether to include ozone mortality as a separate impact in the analysis of the final IAQR based on the results of our evaluation. To the extent that the C-R functions we use to evaluate the preliminary control options in fact capture mortality effects of other criteria pollutants besides PM, we may be overestimating the benefits of reductions in PM. However, we are not providing separate estimates of the mortality benefits from the ozone and CO reductions likely to occur due to the preliminary control options.

- Shape of the C-R Function: The shape of the true PM mortality C-R function is uncertain, but this analysis assumes the C-R function to have a log-linear form (as derived from the literature) throughout the relevant range of exposures. If this is not the correct form of the C-R function, or if certain scenarios predict concentrations well above the range of values for which the C-R function was fitted, avoided mortality may be mis-estimated.

- Regional Differences: As discussed above, significant variability exists in the results of different PM/mortality studies. This variability may reflect regionally specific C-R

functions resulting from regional differences in factors such as the physical and chemical composition of PM. If true regional differences exist, applying the PM/mortality C-R function to regions outside the study location could result in mis-estimation of effects in these regions.

- Exposure/Mortality Lags: There is a potential time lag between changes in PM exposures and changes in mortality rates. For the chronic PM/mortality relationship, the length of the lag is unknown and may be dependent on the kind of exposure. The existence of such a lag is important for the valuation of premature mortality incidence because economic theory suggests that benefits occurring in the future should be discounted. There is no specific scientific evidence of the existence or structure of a PM effects lag. However, current scientific literature on adverse health effects similar to those associated with PM (e.g., smoking-related disease) and the difference in the effect size between chronic exposure studies and daily mortality studies suggests that all incidences of premature mortality reduction associated with a given incremental change in PM exposure probably would not occur in the same year as the exposure reduction. The smoking-related literature also implies that lags of up to a few years or longer are plausible. Adopting the lag structure used in the Tier 2/Gasoline Sulfur and Heavy-Duty Engine/Diesel Fuel RIAs and endorsed by the SAB (EPA-SAB-COUNCIL-ADV-00-001, 1999), we assume a 5-year lag structure.[23] This approach assumes that 25 percent of PM-related premature deaths occur in each of the first 2 years after the exposure and the rest occur in equal parts (approximately 17 percent) in each of the ensuing 3 years.

- Cumulative Effects: As a general point, we attribute the PM/mortality relationship in the underlying epidemiological studies to cumulative exposure to PM. However, the relative roles of PM exposure duration and PM exposure level in inducing premature mortality remain unknown at this time.

4.1.5.3 Baseline Health Effect Incidence Rates

The epidemiological studies of the association between pollution levels and adverse health effects generally provide a direct estimate of the relationship of air quality changes to the relative risk of a health effect, rather than an estimate of the absolute number of avoided cases. For example, a typical result might be that a 10 $\mu g/m^3$ decrease in daily $PM_{2.5}$ levels might decrease hospital admissions by 3 percent. The baseline incidence of the health effect is necessary to convert this relative change into a number of cases. The baseline incidence

[23] The SAB-HES has recently recommended that EPA rethink the use of a 5-year lag. They recommend that a more complex lag structure be considered incorporation components dealing with short-term (0-6 months), intermediate (1-2 years) and long-term (15-25 years) exposures. EPA is evaluating techniques for characterizing lag structures and will incorporate new methods as they become available.

rate provides an estimate of the incidence rate (number of cases of the health effect per year, usually per 10,000 or 100,000 general population) in the assessment location corresponding to baseline pollutant levels in that location. To derive the total baseline incidence per year, this rate must be multiplied by the corresponding population number (e.g., if the baseline incidence rate is number of cases per year per 100,000 population, it must be multiplied by the number of 100,000s in the population).

Some epidemiological studies examine the association between pollution levels and adverse health effects in a specific subpopulation, such as asthmatics or diabetics. In these cases, it is necessary to develop not only baseline incidence rates, but also prevalence rates for the defining condition (e.g., asthma). For both baseline incidence and prevalence data, we use age-specific rates where available. Impact functions are applied to individual age groups and then summed over the relevant age range to provide an estimate of total population benefits.

In most cases, because of a lack of data or methods, we have not attempted to project incidence rates to future years, instead assuming that the most recent data on incidence rates is the best prediction of future incidence rates. In recent years, better data on trends in incidence and prevalence rates for some endpoints, such as asthma, have become available. We are working to develop methods to use these data to project future incidence rates. However, for our primary benefits analysis of the proposed IAQR, we will continue to use current incidence rates. We will examine the impact of using projected mortality rates and asthma prevalence in sensitivity analyses.

Table 4-9 summarizes the baseline incidence data and sources used in the benefits analysis. In most cases, a single national incidence rate is used, due to a lack of more spatially disaggregated data. We used national incidence rates whenever possible, because these data are most applicable to a national assessment of benefits. However, for some studies, the only available incidence information comes from the studies themselves; in these cases, incidence in the study population is assumed to represent typical incidence at the national level. However, for hospital admissions, regional rates are available, and for premature mortality, county-level data are available.

Age, cause, and county-specific mortality rates were obtained from the U.S. Centers for Disease Control (CDC) for the years 1996 through 1998. CDC maintains an online data repository of health statistics, CDC Wonder, accessible at http://wonder.cdc.gov/. The mortality

Table 4-9. Baseline Incidence Rates and Population Prevalence Rates for Use in Impact Functions, General Population

Endpoint	Parameter	Rates	
		Value	Source[a]
Mortality	Daily or annual mortality rate	Age, cause, and county-specific rate	CDC Wonder (1996-1998)
Hospitalizations	Daily hospitalization rate	Age, region, cause-specific rate	1999 NHDS public use data files[b]
Asthma ER visits	Daily asthma ER visit rate	Age, Region specific visit rate	2000 NHAMCS public use data files[c]; 1999 NHDS public use data files[b]
Chronic Bronchitis	Annual prevalence rate per person Age 18-44 Age 45-64 Age 65 and older	 0.0367 0.0505 0.0587	1999 HIS (American Lung Association, 2002b, Table 4)
	Annual incidence rate per person	0.00378	Abbey et al. (1993, Table 3)
Nonfatal MI (heart attacks)	Daily nonfatal myocardial infarction incidence rate per person, 18+ Northeast Midwest South West	 0.0000159 0.0000135 0.0000111 0.0000100	1999 NHDS public use data files[b]; adjusted by 0.93 for prob. of surviving after 28 days (Rosamond et al., 1999)
Asthma Exacerbations	Incidence (and prevalence) among asthmatic African American children - daily wheeze - daily cough - daily dyspnea	 0.076 (0.173) 0.067 (0.145) 0.037 (0.074)	Ostro et al. (2001)
	Prevalence among asthmatic children - daily wheeze - daily cough - daily dyspnea	 0.038 0.086 0.045	Vedal et al. (1998)
Acute Bronchitis	Annual bronchitis incidence rate, children	0.043	American Lung Association (2002a, Table 11)

(continued)

Table 4-9. Baseline Incidence Rates and Population Prevalence Rates for Use in Impact Functions, General Population (continued)

Endpoint	Parameter	Rates	
		Value	Source[a]
Lower Respiratory Symptoms	Daily lower respiratory symptom incidence among children[d]	0.0012	Schwartz (1994, Table 2)
Upper Respiratory Symptoms	Daily upper respiratory symptom incidence among asthmatic children	0.3419	Pope et al. (1991, Table 2)
Work Loss Days	Daily WLD incidence rate per person (18-65) Age 18-24 Age 25-44 Age 45-64	 0.00540 0.00678 0.00492	1996 HIS (Adams et al., 1999, Table 41); U.S. Bureau of the Census (2000)
Minor Restricted Activity Days	Daily MRAD incidence rate per person	0.02137	Ostro and Rothschild (1989, p. 243)
School Loss Days[e]	Daily school absence rate per person	0.055	National Center for Education Statistics (1996)
	Daily illness-related school absence rate per person[e] Northeast Midwest South Southwest	 0.0136 0.0146 0.0142 0.0206	1996 HIS (Adams et al., 1999, Table 47); estimate of 180 school days per year
	Daily *respiratory* illness-related school absence rate per person Northeast Midwest South West	 0.0073 0.0092 0.0061 0.0124	1996 HIS (Adams et al., 1999, Table 47); estimate of 180 school days per year

[a] The following abbreviations are used to describe the national surveys conducted by the National Center for Health Statistics: HIS refers to the National Health Interview Survey; NHDS—National Hospital Discharge Survey; NHAMCS—National Hospital Ambulatory Medical Care Survey.

[b] See ftp://ftp.cdc.gov/pub/Health_Statistics/NCHS/Datasets/NHDS/

[c] See ftp://ftp.cdc.gov/pub/Health_Statistics/NCHS/Datasets/NHAMCS/

[d] Lower Respiratory Symptoms are defined as ≥2 of the following: cough, chest pain, phlegm, wheeze

[e] The estimate of daily illness-related school absences excludes school loss days associated with injuries to match the

rates provided are derived from U.S. death records and U.S. Census Bureau postcensal population estimates. Mortality rates were averaged across 3 years (1996 through 1998) to provide more stable estimates. When estimating rates for age groups that differed from the CDC Wonder groupings, we assumed that rates were uniform across all ages in the reported age group. For example, to estimate mortality rates for individuals ages 30 and up, we scaled the 25- to 34-year old death count and population by one-half and then generated a population-weighted mortality rate using data for the older age groups. Note that we have not projected any changes in mortality rates over time. We are aware that the U.S. Census projections of total and age-specific mortality rates used in our population projections are based on projections of declines in mortality rates for younger populations and increases in mortality rates for older populations over time. We are evaluating the most appropriate way to incorporate these projections of changes in overall national mortality rates into our database of county-level cause-specific mortality rates. In the interim, we have not attempted to adjust future mortality rates. This will lead to an overestimate of mortality benefits in future years, with the overestimation bias increasing the further benefits are projected into the future. We do not at this time have a quantified estimate of the magnitude of the potential bias in the years analyzed for this rule (2010 and 2015).

For the set of endpoints affecting the asthmatic population, in addition to baseline incidence rates, prevalence rates of asthma in the population are needed to define the applicable population. Table 4-9 lists the baseline incidence rates and their sources for asthma symptom endpoints. Table 4-10 lists the prevalence rates used to determine the applicable population for asthma symptom endpoints. Note that these reflect current asthma prevalence and assume no change in prevalence rates in future years. As noted above, we are investigating methods for projecting asthma prevalence rates in future years.

4.1.5.4 Accounting for Potential Health Effect Thresholds

When conducting clinical (chamber) and epidemiological studies, functions may be estimated with or without explicit thresholds. Air pollution levels below the threshold are assumed to have no associated adverse health effects. When a threshold is not assumed, as is often the case in epidemiological studies, any exposure level is assumed to pose a nonzero risk of response to at least one segment of the population.

The possible existence of an effect threshold is a very important scientific question and issue for policy analyses such as this one. The EPA SAB Advisory Council for Clean Air Compliance, which provides advice and review of the EPA's methods for assessing the

benefits and costs of the Clean Air Act under Section 812 of the Clean Air Act, has advised the EPA that there is currently no scientific basis for selecting a threshold of 15 $\mu g/m^3$ or any other specific threshold for the PM-related health effects considered in typical benefits analyses (EPA-SAB-Council-ADV-99-012, 1999). This is supported by the recent literature on health effects of PM exposure (Daniels et al., 2000; Pope, 2000; Rossi et al., 1999; Schwartz, 2000) that finds in most cases no evidence of a nonlinear relationship between PM

Table 4-10. Asthma Prevalence Rates Used to Estimate Asthmatic Populations in Impact Functions

Population Group	Asthma Prevalence Rates	
	Value	Source
All Ages	0.0386	American Lung Association (2002c, Table 7)—based on 1999 HIS
<18	0.0527	American Lung Association (2002c, Table 7)—based on 1999 HIS
5-17	0.0567	American Lung Association (2002c, Table 7)—based on 1999 HIS
18-44	0.0371	American Lung Association (2002c, Table 7)—based on 1999 HIS
45-64	0.0333	American Lung Association (2002c, Table 7)—based on 1999 HIS
65+	0.0221	American Lung Association (2002c, Table 7)—based on 1999 HIS
Male, 27+	0.021	2000 HIS public use data files[a]
African-American, 5 to 17	0.0726	American Lung Association (2002c, Table 9)—based on 1999 HIS
African-American, <18	0.0735	American Lung Association (2002c, Table 9)—based on 1999 HIS

[a] See ftp://ftp.cdc.gov/pub/Health_Statistics/NCHS/Datasets/NHIS/2000/

and health effects and certainly does not find a distinct threshold. The most recent draft of the EPA Air Quality Criteria for Particulate Matter (EPA, 2002) reports only one study, analyzing data from Phoenix, AZ, that reported even limited evidence suggestive of a possible threshold for PM2.5 (Smith et al., 2000).

Recent cohort analyses by HEI (Krewski et al., 2000) and Pope et al. (2002) provide additional evidence of a quasi-linear relationship between long-term exposures to $PM_{2.5}$ and mortality. According to the latest draft PM criteria document, Krewski et al. (2000) found a "visually near-linear relationship between all-cause and cardiopulmonary mortality residuals and mean sulfate concentrations, near-linear between cardiopulmonary mortality and mean $PM_{2.5}$, but a somewhat nonlinear relationship between all-cause mortality residuals and mean $PM_{2.5}$ concentrations that flattens above about 20 $\mu g/m^3$. The confidence bands around the fitted curves are very wide, however, neither requiring a linear relationship nor precluding a nonlinear relationship if suggested by reanalyses."

The Pope et al. (2002) analysis, which represented an extension to the Krewski et al. analysis, found that the functions relating PM2.5 and mortality "were not significantly different from linear associations."

Daniels et al. (2000) examined the presence of thresholds in PM_{10} C-R relationships for daily mortality using the largest 20 U.S. cities for 1987-1994. The results of their models suggest that the linear model was preferred over spline and threshold models. Thus, these results suggest that linear models without a threshold may well be appropriate for estimating the effects of PM_{10} on the types of mortality of main interest. Schwartz and Zanobetti (2000) investigated the presence of threshold by simulation and actual data analysis of 10 U.S. cities. In the analysis of real data from 10 cities, the combined C-R curve did not show evidence of a threshold in the PM_{10}-mortality associations. Schwartz, Laden, and Zanobetti (2002) investigated thresholds by combining data on the PM2.5-mortality relationships for six cities and found an essentially linear relationship down to 2 $\mu g/m^3$, which is at or below anthropogenic background in most areas. They also examined just traffic-related particles and again found no evidence of a threshold. The Smith et al. (2000) study of associations between daily total mortality and $PM_{2.5}$ and $PM_{10-2.5}$ in Phoenix, AZ, (during 1995-1997) also investigated the possibility of a threshold using a piecewise linear model and a cubic spline model. For both the piecewise linear and cubic spline models, the analysis suggested a threshold of around 20 to 25 $\mu g/m^3$. However, the C-R curve for $PM_{2.5}$ presented in this publication suggests more of a U- or V-shaped relationship than the usual "hockey stick" threshold relationship.

Based on the recent literature and advice from the SAB, we assume there are no thresholds for modeling health effects. Although not included in the primary analysis, the potential impact of a health effects threshold on avoided incidences of PM-related premature mortality is explored as a key sensitivity analysis and is presented in Appendix 9-B (to be completed for the supplemental analysis).

Our assumptions regarding thresholds are supported by the National Research Council in its recent review of methods for estimating the public health benefits of air pollution regulations. In their review, the National Research Council concluded that there is no evidence for any departure from linearity in the observed range of exposure to PM_{10} or $PM_{2.5}$, nor any indication of a threshold. They cite the weight of evidence available from both short- and long-term exposure models and the similar effects found in cities with low and high ambient concentrations of PM.

4.1.5.5 Selecting Unit Values for Monetizing Health Endpoints

The appropriate economic value of a change in a health effect depends on whether the health effect is viewed *ex ante* (before the effect has occurred) or *ex post* (after the effect has occurred). Reductions in ambient concentrations of air pollution generally lower the risk of future adverse health affects by a fairly small amount for a large population. The appropriate economic measure is therefore *ex ante* WTP for changes in risk. However, epidemiological studies generally provide estimates of the relative risks of a particular health effect avoided due to a reduction in air pollution. A convenient way to use this data in a consistent framework is to convert probabilities to units of avoided statistical incidences. This measure is calculated by dividing individual WTP for a risk reduction by the related observed change in risk. For example, suppose a measure is able to reduce the risk of premature mortality from 2 in 10,000 to 1 in 10,000 (a reduction of 1 in 10,000). If individual WTP for this risk reduction is $100, then the WTP for an avoided statistical premature mortality amounts to $1 million ($100/0.0001 change in risk). Using this approach, the size of the affected population is automatically taken into account by the number of incidences predicted by epidemiological studies applied to the relevant population. The same type of calculation can produce values for statistical incidences of other health endpoints.

For some health effects, such as hospital admissions, WTP estimates are generally not available. In these cases, we use the cost of treating or mitigating the effect as a primary estimate. For example, for the valuation of hospital admissions we use the avoided medical costs as an estimate of the value of avoiding the health effects causing the admission. These COI estimates generally understate the true value of reductions in risk of a health effect. They tend to reflect the direct expenditures related to treatment but not the value of avoided pain and suffering from the health effect. Table 4-11 summarizes the value estimates per health effect that we used in this analysis. Values are presented both for a 1990 base income level and adjusted for income growth in the two future analysis years, 2010 and 2015. Note that the unit values for hospital admissions are the weighted averages of the ICD-9 code-specific values for the group of ICD-9 codes included in the hospital admission categories. A discussion of the valuation methods for premature mortality and CB is provided here because of the relative importance of these effects. Discussions of the methods used to value nonfatal myocardial infarctions (heart attacks) and school absence days are provided because these endpoints have only recently been added to the analysis and the valuation methods are still under development. We welcome comment on these valuation methods. In the following

discussions, unit values are presented at 1990 levels of income for consistency with previous analyses. Equivalent future year values can be obtained from Table 4-11.

Table 4-11. Unit Values Used for Economic Valuation of Health Endpoints (1999$)

	Central Estimate of Value Per Statistical Incidence			
	1990 Income	**2010 Income**	**2015 Income**	
Premature Mortality (Value of a Statistical Life) **Health Endpoint**	**Level**	**Level**	**Level**	Point estimate is the mean of a normal distribution with a 95 percent confidence interval between $1 and $10 million. Confidence interval is based on two meta-analyses of the wage-risk VSL literature. $1 million represents the lower end of the interquartile range from the Mrozek and Taylor (2000) meta-analysis. $10 million represents the upper end of the interquartile range from the Viscusi and Aldy (2003) meta-analysis. The VSL represents the value of a small change in mortality risk aggregated over the affected population.
Nonfatal Myocardial Infarction (heart attack)				Point estimate is the mean of a generated distribution of WTP to avoid a case of pollution-related CB. WTP to avoid a case of pollution-related CB is derived by adjusting WTP (as described in Viscusi et al., 1991) to avoid a severe case of CB for the difference in severity and taking into account the elasticity of WTP with respect to severity of CB. Age specific cost-of-illness values reflecting lost earnings and direct medical costs over a 5 year period following a non-fatal MI. Lost earnings estimates based on Cropper and Krupnick (1990). Direct medical costs based on simple average of estimates from Russell et al. (1998) and Wittels et al. (1990).
	$66,902	$66,902	$66,902	:
	$74,676	$74,676	$74,676	Cropper and Krupnick (1990). Present discounted value of 5 yrs of lost earnings:
	$78,834	$78,834	$78,834	
	$140,649	$140,649	$140,649	at 7%
	$66,902	$66,902	$66,902	25-44 $8,774 $7,855 45-54 $12,932 $11,578 55-65 $74,746 $66,920
	$65,293	$65,293	$65,293	
	$73,149	$73,149	$73,149	: An average of:
	$76,871	$76,871	$76,871	1. Wittels et al., 1990 ($102,658 – no discounting)
	$132,214	$132,214	$132,214	2. Russell et al., 1998, 5-yr period. ($22,331 at 3% discount rate; $21,113 at 7% discount rate)
	~~$65,293~~	~~$65,293~~	~~$65,293~~	

Table 4-11. Unit Values Used for Economic Valuation of Health Endpoints (1999$) (continued)

Health Endpoint	Central Estimate of Value Per Statistical Incidence			Derivation of Estimates
	1990 Income	2010 Income	2015 Income	
Hospital Admissions	**Level**	**Level**	**Level**	
Chronic Obstructive Pulmonary Disease (COPD) (ICD codes 490-492, 494-496)				The COI estimates (lost earnings plus direct medical costs) are based on ICD-9 code level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total COPD category illnesses) reported in Agency for Healthcare Research and Quality, 2000 (www.ahrq.gov).
Pneumonia (ICD codes 480-487)				The COI estimates (lost earnings plus direct medical costs) are based on ICD-9 code level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total pneumonia category illnesses) reported in Agency for Healthcare Research and Quality, 2000 (www.ahrq.gov). The COI estimates (lost earnings plus direct medical costs) are based on ICD-9 code level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total asthma category illnesses) reported in Agency for Healthcare Research and Quality, 2000 (www.ahrq.gov).
All Cardiovascular (ICD codes 390-429)				The COI estimates (lost earnings plus direct medical costs) are based on ICD-9 code level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total cardiovascular category illnesses) reported in Agency for Healthcare Research and Quality, 2000 (www.ahrq.gov).
				Simple average of two unit COI values: (1) $311.55, from Smith et al., 1997, and (2) $260.67, from Stanford et al., 1999.

Table 4-11. Unit Values Used for Economic Valuation of Health Endpoints (1999$) (continued)

	Central Estimate of Value Per Statistical Incidence			
	1990 Income	2010 Income	2015 Income	
Respiratory Ailments Not Requiring Hospitalization	Level	Level	Level	
Health Endpoint				Combinations of the 3 symptoms for which WTP estimates are available that closely match those listed by Pope, et al. result in 7 different "symptom clusters," each describing a "type" of URS. A dollar value was derived for each type of URS, using mid-range estimates of WTP (IEc, 1994) to avoid each symptom in the cluster and assuming additivity of WTPs. The dollar value for URS is the average of the dollar values for the 7 different types of URS. **Derivation of Estimates**
				Combinations of the 4 symptoms for which WTP estimates are available that closely match those listed by Schwartz, et al. result in 11 different "symptom clusters," each describing a "type" of LRS. A dollar value was derived for each type of LRS, using mid-range estimates of WTP (IEc, 1994) to avoid each symptom in the cluster and assuming additivity of WTPs. The dollar value for LRS is the average of the dollar values for the 11 different types of LRS.
				Asthma exacerbations are valued at $42 per incidence, based on the mean of average WTP estimates for the four severity definitions of a "bad asthma day," described in Rowe and Chestnut (1986). This study surveyed asthmatics to estimate WTP for avoidance of a "bad asthma day," as defined by the subjects. For purposes of valuation, an asthma attack is assumed to be equivalent to a day in which asthma is moderate or worse as reported in the Rowe and Chestnut (1986) study.
				Assumes a 6 day episode, with daily value equal to the average of low and high values for related respiratory symptoms recommended in Neumann, et al. 1994.

Table 4-11. Unit Values Used for Economic Valuation of Health Endpoints (1999$) (continued)

	Central Estimate of Value Per Statistical Incidence			
	1990 Income	2010 Income	2015 Income	
Restricted Activity and Work/School Loss Days	Level	Level	Level	
Health Endpoint	Variable (national median =)			County-specific median annual wages divided by 50 (assuming 2 weeks of vacation) and then by 5 – to get median daily wage. U.S. Year 2000 Census, compiled by Geolytics, Inc.
				Based on expected lost wages from parent staying home with child. Estimated daily lost wage (if a mother must stay at home with a sick child) is based on the median weekly wage among women age 25 and older in 2000 (U.S. Census Bureau, Statistical Abstract of the United States: 2001, Section 12: Labor Force, Employment, and Earnings, Table No. 621). This median wage is $551. Dividing by 5 gives an estimated median daily wage of $103. **Derivation of Estimates** The expected loss in wages due to a day of school absence in which the mother would have to stay home with her child is estimated as the probability that the mother is in the workforce times the daily wage she would lose if she missed a day = 72.85% of $103, or $75.
	$0.95 per worker per 10% change in ozone per day	$0.95 per worker per 10% change in ozone per day	$0.95 per worker per 10% change in ozone per day	Based on $68 – median daily earnings of workers in farming, forestry and fishing – from Table 621, Statistical Abstract of the United States ("Full-Time Wage and Salary Workers – Number and Earnings: 1985 to 2000") (Source of data in table: U.S. Bureau of Labor Statistics, Bulletin 2307 and Employment and Earnings, monthly).
Minor Restricted Activity Days (MRADs)				Median WTP estimate to avoid one MRAD from Tolley, et al. (1986) .

4.1.5.5.1 Valuing Reductions in Premature Mortality Risk. We estimate the monetary benefit of reducing premature mortality risk using the "value of statistical lives saved" (VSL) approach, which is a summary measure for the value of small changes in mortality risk experienced by a large number of people. The VSL approach applies information from several published value-of-life studies to determine a reasonable benefit of preventing premature mortality. The mean value of avoiding one statistical death is assumed to be $5.5 million in 1999 dollars. This represents a central value consistent with the range of values suggested by recent meta-analyses of the wage-risk VSL literature. The distribution of VSL is characterized by a confidence interval from $1 to $10 million, based on two meta-analyses of the wage-risk VSL literature. The $1 million lower confidence limit represents the lower end of the interquartile range from the Mrozek and Taylor (2000) meta-analysis. The $10 million upper confidence limit represents the upper end of the interquartile range from the Viscusi and Aldy (2003) meta-analysis.

In previous analyses, we used an estimate of mean VSL equal to $6.3 million, based on a distribution fitted to the estimates from 26 value-of-life studies identified in the Section 812 reports as "applicable to policy analysis." The EPA welcomes comments on the departure from this approach for the current analysis.

As indicated in the previous section on quantification of premature mortality benefits, we assume for this analysis that some of the incidences of premature mortality related to PM exposures occur in a distributed fashion over the 5 years following exposure. To take this into account in the valuation of reductions in premature mortality, we apply an annual 3 percent discount rate to the value of premature mortality occurring in future years.[24]

The economics literature concerning the appropriate method for valuing reductions in premature mortality risk is still developing. The adoption of a value for the projected reduction in the risk of premature mortality is the subject of continuing discussion within the economics and public policy analysis community. Regardless of the theoretical economic considerations, the EPA prefers not to draw distinctions in the monetary value assigned to the

[24]The choice of a discount rate, and its associated conceptual basis, is a topic of ongoing discussion within the federal government. The EPA adopted a 3 percent discount rate for its base estimate in this case to reflect reliance on a "social rate of time preference" discounting concept. We have also calculated benefits and costs using a 7 percent rate consistent with an "opportunity cost of capital" concept to reflect the time value of resources directed to meet regulatory requirements. In this case, the benefit and cost estimates were not significantly affected by the choice of discount rate. Further discussion of this topic appears in the EPA's *Guidelines for Preparing Economic Analyses* (in press).

lives saved even if they differ in age, health status, socioeconomic status, gender, or other characteristic of the adult population.

Following the advice of the EEAC of the SAB, the EPA currently uses the VSL approach in calculating the primary estimate of mortality benefits, because we believe this calculation provides the most reasonable single estimate of an individual's willingness to trade off money for reductions in mortality risk (EPA-SAB-EEAC-00-013). Although there are several differences between the labor market studies the EPA uses to derive a VSL estimate and the PM air pollution context addressed here, those differences in the affected populations and the nature of the risks imply both upward and downward adjustments. Table 4-12 lists some of these differences and the expected effect on the VSL estimate for air pollution-related mortality. In the absence of a comprehensive and balanced set of adjustment factors, the EPA believes it is reasonable to continue to use the $5.5 million value while acknowledging the significant limitations and uncertainties in the available literature.

Table 4-12. Expected Impact on Estimated Benefits of Premature Mortality Reductions of Differences Between Factors Used in Developing Applied VSL and Theoretically Appropriate VSL

Attribute	Expected Direction of Bias
Age	Uncertain, perhaps overestimate
Life expectancy/health status	Uncertain, perhaps overestimate
Attitudes toward risk	Underestimate
Income	Uncertain
Voluntary vs. Involuntary	Uncertain, perhaps underestimate
Catastrophic vs. protracted death	Uncertain, perhaps underestimate

Some economists emphasize that the VSL is not a single number relevant for all situations. Indeed, the VSL estimate of $5.5 million (1999 dollars) is itself the central tendency of a number of estimates of the VSL for some rather narrowly defined populations. When there are significant differences between the population affected by a particular health

risk and the populations used in the labor market studies, as is the case here, some economists prefer to adjust the VSL estimate to reflect those differences.

The SAB-EEAC advised the EPA "continue to use a wage-risk-based VSL as its primary estimate, including appropriate sensitivity analyses to reflect the uncertainty of these estimates," and that "the only risk characteristic for which adjustments to the VSL can be made is the timing of the risk" (EPA-SAB-EEAC-00-013, EPA, 2000b). In developing our primary estimate of the benefits of premature mortality reductions, we have followed this advice and discounted over the lag period between exposure and premature mortality.

Uncertainties Specific to Premature Mortality Valuation. The economic benefits associated with premature mortality are the largest category of monetized benefits of the proposed IAQR. In addition, in prior analyses, the EPA has identified valuation of mortality benefits as the largest contributor to the range of uncertainty in monetized benefits (see EPA [1999]). Because of the uncertainty in estimates of the value of premature mortality avoidance, it is important to adequately characterize and understand the various types of economic approaches available for mortality valuation. Such an assessment also requires an understanding of how alternative valuation approaches reflect that some individuals may be more susceptible to air pollution-induced mortality or reflect differences in the nature of the risk presented by air pollution relative to the risks studied in the relevant economics literature.

The health science literature on air pollution indicates that several human characteristics affect the degree to which mortality risk affects an individual. For example, some age groups appear to be more susceptible to air pollution than others (e.g., the elderly and children). Health status prior to exposure also affects susceptibility. An ideal benefits estimate of mortality risk reduction would reflect these human characteristics, in addition to an individual's WTP to improve one's own chances of survival plus WTP to improve other individuals' survival rates. The ideal measure would also take into account the specific nature of the risk reduction commodity that is provided to individuals, as well as the context in which risk is reduced. To measure this value, it is important to assess how reductions in air pollution reduce the risk of dying from the time that reductions take effect onward, and how individuals value these changes. Each individual's survival curve, or the probability of surviving beyond a given age, should shift as a result of an environmental quality improvement. For example, changing the current probability of survival for an individual also shifts future probabilities of that individual's survival. This probability shift will differ

across individuals because survival curves depend on such characteristics as age, health state, and the current age to which the individual is likely to survive.

Although a survival curve approach provides a theoretically preferred method for valuing the benefits of reduced risk of premature mortality associated with reducing air pollution, the approach requires a great deal of data to implement. The economic valuation literature does not yet include good estimates of the value of this risk reduction commodity. As a result, in this study we value avoided premature mortality risk using the VSL approach.

Other uncertainties specific to premature mortality valuation include the following:

- Across-study variation: There is considerable uncertainty as to whether the available literature on VSL provides adequate estimates of the VSL saved by air pollution reduction. Although there is considerable variation in the analytical designs and data used in the existing literature, the majority of the studies involve the value of risks to a middle-aged working population. Most of the studies examine differences in wages of risky occupations, using a wage-hedonic approach. Certain characteristics of both the population affected and the mortality risk facing that population are believed to affect the average WTP to reduce the risk. The appropriateness of a distribution of WTP based on the current VSL literature for valuing the mortality-related benefits of reductions in air pollution concentrations therefore depends not only on the quality of the studies (i.e., how well they measure what they are trying to measure), but also on the extent to which the risks being valued are similar and the extent to which the subjects in the studies are similar to the population affected by changes in pollution concentrations.

- Level of risk reduction: The transferability of estimates of the VSL from the wage-risk studies to the context of the Interstate Air Quality Rulemaking analysis rests on the assumption that, within a reasonable range, WTP for reductions in mortality risk is linear in risk reduction. For example, suppose a study estimates that the average WTP for a reduction in mortality risk of 1/100,000 is $50, but that the actual mortality risk reduction resulting from a given pollutant reduction is 1/10,000. If WTP for reductions in mortality risk is linear in risk reduction, then a WTP of $50 for a reduction of 1/100,000 implies a WTP of $500 for a risk reduction of 1/10,000 (which is 10 times the risk reduction valued in the study). Under the assumption of linearity, the estimate of the VSL does not depend on the particular amount of risk reduction being valued. This assumption has been shown to be reasonable provided the change in the risk being valued is within the range of risks evaluated in the underlying studies (Rowlatt et al., 1998).

- Voluntariness of risks evaluated: Although job-related mortality risks may differ in several ways from air pollution-related mortality risks, the most important difference may be that job-related risks are incurred voluntarily, or generally assumed to be, whereas air pollution-related risks are incurred involuntarily. Some evidence suggests that people will pay more to reduce involuntarily incurred risks than risks incurred voluntarily. If this is the case, WTP estimates based on wage-risk studies may understate WTP to reduce involuntarily incurred air pollution-related mortality risks.

- Sudden versus protracted death: A final important difference related to the nature of the risk may be that some workplace mortality risks tend to involve sudden, catastrophic events, whereas air pollution-related risks tend to involve longer periods of disease and suffering prior to death. Some evidence suggests that WTP to avoid a risk of a protracted death involving prolonged suffering and loss of dignity and personal control is greater than the WTP to avoid a risk (of identical magnitude) of sudden death. To the extent that the mortality risks addressed in this assessment are associated with longer periods of illness or greater pain and suffering than are the risks addressed in the valuation literature, the WTP measurements employed in the present analysis would reflect a downward bias.

- Self-selection and skill in avoiding risk. Recent research (Shogren et al., 2002) suggests that VSL estimates based on hedonic wage studies may overstate the average value of a risk reduction. This is based on the fact that the risk-wage tradeoff revealed in hedonic studies reflects the preferences of the marginal worker (i.e., that worker who demands the highest compensation for his risk reduction). This worker must have either higher risk, lower risk tolerance, or both. However, the risk estimate used in hedonic studies is generally based on average risk, so the VSL may be upwardly biased because the wage differential and risk measures do not match.

4.1.5.5.2 Valuing Reductions in the Risk of Chronic Bronchitis. The best available estimate of WTP to avoid a case of CB comes from Viscusi et al. (1991). The Viscusi et al. study, however, describes a severe case of CB to the survey respondents. We therefore employ an estimate of WTP to avoid a pollution-related case of CB, based on adjusting the Viscusi et al. (1991) estimate of the WTP to avoid a severe case. This is done to account for the likelihood that an average case of pollution-related CB is not as severe. The adjustment is made by applying the elasticity of WTP with respect to severity reported in the Krupnick and Cropper (1992) study. Details of this adjustment procedure are provided in the benefits TSD for the nonroad diesel rulemaking (Abt Associates, 2003).

We use the mean of a distribution of WTP estimates as the central tendency estimate of WTP to avoid a pollution-related case of CB in this analysis. The distribution incorporates uncertainty from three sources: the WTP to avoid a case of severe CB, as described by Viscusi et al.; the severity level of an average pollution-related case of CB (relative to that of the case described by Viscusi et al.); and the elasticity of WTP with respect to severity of the illness. Based on assumptions about the distributions of each of these three uncertain components, we derive a distribution of WTP to avoid a pollution-related case of CB by statistical uncertainty analysis techniques. The expected value (i.e., mean) of this distribution, which is about $331,000 (2000$), is taken as the central tendency estimate of WTP to avoid a PM-related case of CB.

4.1.5.5.3 Valuing Reductions in Non-Fatal Myocardial Infarctions (Heart Attacks). The Agency has recently incorporated into its analyses the impact of air pollution on the expected number of nonfatal heart attacks, although it has examined the impact of reductions in other related cardiovascular endpoints. We were not able to identify a suitable WTP value for reductions in the risk of nonfatal heart attacks. Instead, we propose a cCOI unit value with two components: the direct medical costs and the opportunity cost (lost earnings) associated with the illness event. Because the costs associated with an myocardial infarction extend beyond the initial event itself, we consider costs incurred over several years. Using age-specific annual lost earnings estimated by Cropper and Krupnick (1990) and a 3 percent discount rate, we estimated a present discounted value in lost earnings (in 2000$) over 5 years due to an myocardial infarction of $8,774 for someone between the ages of 25 and 44, $12,932 for someone between the ages of 45 and 54, and $74,746 for someone between the ages of 55 and 65. The corresponding age-specific estimates of lost earnings (in 2000$) using a 7 percent discount rate are $7,855, $11,578, and $66,920, respectively. Cropper and Krupnick (1990) do not provide lost earnings estimates for populations under 25 or over 65. As such, we do not include lost earnings in the cost estimates for these age groups.

We found three possible sources in the literature of estimates of the direct medical costs of myocardial infarction:

- Wittels et al. (1990) estimated expected total medical costs of myocardial infarction over 5 years to be $51,211 (in 1986$) for people who were admitted to the hospital and survived hospitalization. (There does not appear to be any discounting used.) Wittels et al. was used to value coronary heart disease in the 812 Retrospective Analysis of the Clean Air Act. Using the CPI-U for medical care, the Wittels estimate is $109,474 in year 2000$. This estimated cost is based on a medical cost model, which incorporated therapeutic options, projected

outcomes, and prices (using "knowledgeable cardiologists" as consultants). The model used medical data and medical decision algorithms to estimate the probabilities of certain events and/or medical procedures being used. The authors note that the average length of hospitalization for acute myocardial infarction has decreased over time (from an average of 12.9 days in 1980 to an average of 11 days in 1983). Wittels et al. used 10 days as the average in their study. It is unclear how much further the length of stay for myocardial infarction may have decreased from 1983 to the present. The average length of stay for ICD code 410 (myocardial infarction) in the year-2000 AHQR HCUP database is 5.5 days. However, this may include patients who died in the hospital (not included among our nonfatal myocardial infarction cases), whose length of stay was therefore substantially shorter than it would be if they had not died.

- Eisenstein et al. (2001) estimated 10-year costs of $44,663 in 1997$, or $49,651 in 2000$ for myocardial infarction patients, using statistical prediction (regression) models to estimate inpatient costs. Only inpatient costs (physician fees and hospital costs) were included.

- Russell et al. (1998) estimated first-year direct medical costs of treating nonfatal myocardial infarction of $15,540 (in 1995$) and $1,051 annually thereafter. Converting to year 2000$, that would be $23,353 for a 5-year period (without discounting) or $29,568 for a 10-year period.

In summary, the three different studies provided significantly different values (see Table 4-13).

As noted above, the estimates from these three studies are substantially different, and we have not adequately resolved the sources of differences in the estimates. Because the wage-related opportunity cost estimates from Cropper and Krupnick (1990) cover a 5-year period, we use estimates for medical costs that similarly cover a 5-year period (i.e., estimates from Wittels et al. (1990) and Russell et al. (1998). We use a simple average of the two 5-year estimates, or $65,902, and add it to the 5-year opportunity cost estimate. The resulting estimates are given in Table 4-14.

Table 4-13. Alternative Direct Medical Cost of Illness Estimates for Nonfatal Heart Attacks

Study	Direct Medical Costs (2000$)	Over an x-Year Period, for x =
Wittels et al. (1990)	$109,474[a]	5
Russell et al. (1998)	$22,331[b]	5
Eisenstein et al. (2001)	$49,651[b]	10
Russell et al. (1998)	$27,242[b]	10

[a] Wittels et al. did not appear to discount costs incurred in future years.

[b] Using a 3 percent discount rate.

Table 4-14. Estimated Costs Over a 5-Year Period (in 2000$) of a Nonfatal Myocardial Infarction

Age Group	Opportunity Cost	Medical Cost[a]	Total Cost
0 - 24	$0	$65,902	$65,902
25-44	$8,774[b]	$65,902	$74,676
45 - 54	$12,253[b]	$65,902	$78,834
55 - 65	$70,619[b]	$65,902	$140,649
> 65	$0	$65,902	$65,902

[a] An average of the 5-year costs estimated by Wittels et al., 1990, and Russell et al., 1998.

[b] From Cropper and Krupnick, 1990, using a 3 percent discount rate.

4.1.5.5.4 Valuing Reductions in School Absence Days. School absences associated with exposure to ozone are likely to be due to respiratory-related symptoms and illnesses. Because the respiratory symptom and illness endpoints we are including are all PM-related rather than ozone-related, we do not have to be concerned about double counting of benefits if we aggregate the benefits of avoiding ozone-related school absences with the benefits of avoiding PM-related respiratory symptoms and illnesses.

One possible approach to valuing a school absence is using a parental opportunity cost approach. This method requires two steps: estimate the probability that, if a school child stays home from school, a parent will have to stay home from work to care for the child, and value the lost productivity at the person's wage. Using this method, we would estimate the proportion of families with school-age children in which both parents work, and value a school loss day as the probability of a work loss day resulting from a school loss day (i.e., the proportion of households with school-age children in which both parents work) times some measure of lost wages (whatever measure we use to value work loss days). There are two significant problems with this method, however. First, it omits WTP to avoid the symptoms/illness that resulted in the school absence. Second, it effectively gives zero value to school absences which do not result in a work loss day (unless we derive an alternative estimate of the value of the parent's time for those cases in which the parent is not in the labor force). We are investigating approaches using WTP for avoid the symptoms/illnesses causing the absence. In the interim, we will use the parental opportunity cost approach.

For the parental opportunity cost approach, we make an explicit, conservative assumption that in married households with two working parents, the female parent will stay home with a sick child. From the U.S. Census Bureau, Statistical Abstract of the United States: 2001, we obtained (1) the numbers of single, married, and "other" (i.e., widowed, divorced, or separated) women with children in the workforce, and (2) the rates of participation in the workforce of single, married, and "other" women with children. From these two sets of statistics, we inferred the numbers of single, married, and "other" women with children, and the corresponding percentages. These percentages were used to calculate a weighted average participation rate, as shown in Table 4-15.

Our estimated daily lost wage (if a mother must stay at home with a sick child) is based on the median weekly wage among women age 25 and older in 2000 (U.S. Census Bureau, Statistical Abstract of the United States: 2001, Section 12: Labor Force, Employment, and Earnings, Table No. 621). This median wage is $551. Dividing by 5 gives an estimated median daily wage of $103.

Table 4-15. Women with Children: Number and Percent in the Labor Force, 2000, and Weighted Average Participation Rate[a]

	Number (in millions) in Labor Force (1)	Participation Rate (2)	Implied Total Number in Population (in millions) (3) = (1)/(2)	Implied Percent in Population (4)	Weighted Average Participation Rate [=sum (2)*(4) over rows]
Single	3.1	73.9%	4.19	11.84%	
Married	18.2	70.6%	25.78	72.79%	
Other[b]	4.5	82.7%	5.44	15.36%	
Total:			35.42		
					72.85%

[a] Data in columns (1) and (2) are from U.S. Census Bureau, Statistical Abstract of the United States: 2001, Section 12: Labor Force, Employment, and Earnings, Table No. 577.

[b] Widowed, divorced, or separated.

The expected loss in wages due to a day of school absence in which the mother would have to stay home with her child is estimated as the probability that the mother is in the workforce times the daily wage she would lose if she missed a day = 72.85% of $103, or $75.[25]

[25] In a very recent article, Hall, Brajer, and Lurmann (2003) use a similar methodology to derive a mid-estimate value per school absence day for California of between $70 and $81, depending on differences in incomes between three counties in California. Our national average estimate of $75 per absence is consistent with these published values.

4.1.5.6 Unquantified Health Effects

In addition to the health effects discussed above, there is emerging evidence that human exposure to ozone may be associated with premature mortality (Ito and Thurston, 1996; Samet, et al. 1997, Ito and Thurston, 2001), PM and ozone with increased emergency room visits for non-asthma respiratory causes (US EPA, 1996a; 1996b), ozone with impaired airway responsiveness (US EPA, 1996a), ozone with increased susceptibility to respiratory infection (US EPA, 1996a), ozone with acute inflammation and respiratory cell damage (US EPA, 1996a), ozone and PM with premature aging of the lungs and chronic respiratory damage (US EPA, 1996a; 1996b), ozone with onset of asthma in exercising children (McConnell et al. 2002), and PM with reduced heart rate variability and other changes in cardiac function. An improvement in ambient PM and ozone air quality may reduce the number of incidences within each effect category that the U.S. population would experience. Although these health effects are believed to be PM or ozone-induced, effect estimates are not available for quantifying the benefits associated with reducing these effects. The inability to quantify these effects lends a downward bias to the monetized benefits presented in this analysis.

4.1.6 Human Welfare Impact Assessment

PM and ozone have numerous documented effects on environmental quality that affect human welfare. These welfare effects include direct damages to property, either through impacts on material structures or by soiling of surfaces, direct economic damages in the form of lost productivity of crops and trees, indirect damages through alteration of ecosystem functions, and indirect economic damages through the loss in value of recreational experiences or the existence value of important resources. EPA's Criteria Documents for PM and ozone list numerous physical and ecological effects known to be linked to ambient concentrations of these pollutants (US EPA, 1996a; 1996b). This section describes individual effects and how we quantify and monetize them. These effects include changes in commercial crop and forest yields, visibility, and nitrogen deposition to estuaries.

4.1.6.1 Visibility Benefits

Changes in the level of ambient particulate matter caused by the reduction in emissions from the IAQR will change the level of visibility in much of the Eastern U.S. Visibility directly affects people's enjoyment of a variety of daily activities. Individuals value visibility both in the places they live and work, in the places they travel to for recreational purposes, and at sites of unique public value, such as the Great Smokey

Mountains National Park. This section discusses the measurement of the economic benefits of visibility.

It is difficult to quantitatively define a visibility endpoint that can be used for valuation. Increases in PM concentrations cause increases in light extinction. Light extinction is a measure of how much the components of the atmosphere absorb light. More light absorption means that the clarity of visual images and visual range is reduced, *ceteris paribus*. Light absorption is a variable that can be accurately measured. Sisler (1996) created a unitless measure of visibility based directly on the degree of measured light absorption called the *deciview*. Deciviews are standardized for a reference distance in such a way that one deciview corresponds to a change of about 10 percent in available light. Sisler characterized a change in light extinction of one deciview as "a small but perceptible scenic change under many circumstances." Air quality models were used to predict the change in visibility, measured in deciviews, of the areas affected by the preliminary control options.[26]

EPA considers benefits from two categories of visibility changes: residential visibility and recreational visibility. In both cases economic benefits are believed to consist of both use values and non-use values. Use values include the aesthetic benefits of better visibility, improved road and air safety, and enhanced recreation in activities like hunting and birdwatching. Non-use values are based on people's beliefs that the environment ought to exist free of human-induced haze. Non-use values may be a more important component of value for recreational areas, particularly national parks and monuments.

Residential visibility benefits are those that occur from visibility changes in urban, suburban, and rural areas, and also in recreational areas not listed as federal Class I areas.[27] For the purposes of this analysis, recreational visibility improvements are defined as those that occur specifically in federal Class I areas. A key distinction between recreational and residential benefits is that only those people living in residential areas are assumed to receive benefits from residential visibility, while all households in the U.S. are assumed to derive

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[26]A change of less than 10 percent in the light extinction budget represents a measurable improvement in visibility, but may not be perceptible to the eye in many cases. Some of the average regional changes in visibility are less than one deciview (i.e. less than 10 percent of the light extinction budget), and thus less than perceptible. However, this does not mean that these changes are not real or significant. Our assumption is then that individuals can place values on changes in visibility that may not be perceptible. This is quite plausible if individuals are aware that many regulations lead to small improvements in visibility which when considered together amount to perceptible changes in visibility.

[27]The Clean Air Act designates 156 national parks and wilderness areas as Class I areas for visibility protection.

some benefit from improvements in Class I areas. Values are assumed to be higher if the Class I area is located close to their home.[28]

Only two existing studies provide defensible monetary estimates of the value of visibility changes. One is a study on residential visibility conducted in 1990 (McClelland, et. al., 1993) and the other is a 1988 survey on recreational visibility value (Chestnut and Rowe, 1990a; 1990b). While there are a number of other studies in the literature, they were conducted in the early 1980s and did not use methods that are considered defensible by current standards. Both the Chestnut and Rowe and McClelland et al studies utilize the contingent valuation method. There has been a great deal of controversy and significant development of both theoretical and empirical knowledge about how to conduct CV surveys in the past decade. In EPA's judgment, the Chestnut and Rowe study contains many of the elements of a valid CV study and is sufficiently reliable to serve as the basis for monetary estimates of the benefits of visibility changes in recreational areas.[29] This study serves as an essential input to our estimates of the benefits of recreational visibility improvements in the primary benefits estimates. Consistent with SAB advice, EPA has designated the McClelland, et al. study as significantly less reliable for regulatory benefit-cost analysis, although it does provide useful estimates on the order of magnitude of residential visibility benefits (EPA-SAB-COUNCIL-ADV-00-002, 1999). Residential visibility benefits are therefore only included as a sensitivity estimate in Appendix 9-B (to be completed for the Supplemental Analysis).

The Chestnut and Rowe study measured the demand for visibility in Class I areas managed by the National Park Service (NPS) in three broad regions of the country: California, the Southwest, and the Southeast. Respondents in five states were asked about their willingness to pay to protect national parks or NPS-managed wilderness areas within a particular region. The survey used photographs reflecting different visibility levels in the specified recreational areas. The visibility levels in these photographs were later converted to deciviews for the current analysis. The survey data collected were used to estimate a WTP

[28]For details of the visibility estimates discussed in this chapter, please refer to the benefits technical support document for the Nonroad Diesel rulemaking (Abt Associates 2003).

[29] An SAB advisory letter indicates that"many members of the Council believe that the Chestnut and Rowe study is the best available." (EPA-SAB-COUNCIL-ADV-00-002, 1999) However, the committee did not formally approve use of these estimates because of concerns about the peer-reviewed status of the study. EPA believes the study has received adequate review and has been cited in numerous peer-reviewed publications (Chestnut and Dennis, 1997).

equation for improved visibility. In addition to the visibility change variable, the estimating equation also included household income as an explanatory variable.

The Chestnut and Rowe study did not measure values for visibility improvement in Class I areas outside the three regions. Their study covered 86 of the 156 Class I areas in the U.S. We can infer the value of visibility changes in the other Class I areas by transferring values of visibility changes at Class I areas in the study regions. However, these values are not as defensible and are thus presented only as a sensitivity analysis (to be completed for the Supplemental Analysis). A complete description of the benefits transfer method used to infer values for visibility changes in Class I areas outside the study regions is provided in the benefits TSD for the Nonroad Diesel rulemaking (Abt Associates, 2003).

The estimated relationship from the Chestnut and Rowe study is only directly applicable to the populations represented by survey respondents. EPA used benefits transfer methodology to extrapolate these results to the population affected by the proposed IAQR. A general willingness to pay equation for improved visibility (measured in deciviews) was developed as a function of the baseline level of visibility, the magnitude of the visibility improvement, and household income. The behavioral parameters of this equation were taken from analysis of the Chestnut and Rowe data. These parameters were used to calibrate WTP for the visibility changes resulting from the IAQR. The method for developing calibrated WTP functions is based on the approach developed by Smith, et al. (2002). Available evidence indicates that households are willing to pay more for a given visibility improvement as their income increases (Chestnut, 1997). The benefits estimates here incorporate Chestnut's estimate that a 1 percent increase in income is associated with a 0.9 percent increase in WTP for a given change in visibility.

Using the methodology outlined above, EPA estimates that the total WTP for the visibility improvements in Southeastern Class I areas brought about by the IAQR is $880 million in 2010 and $1,400 million in 2015. This value includes the value to households living in the same state as the Class I area as well as values for all households in the U.S. living outside the state containing the Class I area, and the value accounts for growth in real income. We examine the impact of expanding the visibility benefits analysis to other areas of the country in a sensitivity analysis to be completed for the Supplemental Analysis.

The benefits resulting from visibility improvements in Southeastern Class I areas under the Proposed IAQR are presented in Figure 4-2. This figure presents these benefits both in terms of the total benefits modeled for each of the Class I areas (i.e., the "Park Benefits" map) and the benefits realized by the populations in each of the 48 contiguous states (i.e., the "State Benefits" map). The latter results reflect the willingness to pay of state residents for visibility improvements occuring in Class I areas in the Southeastern United States.

One major source of uncertainty for the visibility benefit estimate is the benefits transfer process used. Judgments used to choose the functional form and key parameters of the estimating equation for willingness to pay for the affected population could have significant effects on the size of the estimates. Assumptions about how individuals respond to changes in visibility that are either very small, or outside the range covered in the Chestnut and Rowe study, could also affect the results.

4.1.6.2 Agricultural, Forestry and other Vegetation Related Benefits

The Ozone Criteria Document notes that "ozone affects vegetation throughout the United States, impairing crops, native vegetation, and ecosystems more than any other air pollutant" (US EPA, 1996). Changes in ground level ozone resulting from the preliminary control options are expected to impact crop and forest yields throughout the affected area.

Well-developed techniques exist to provide monetary estimates of these benefits to agricultural producers and to consumers. These techniques use models of planting decisions, yield response functions, and agricultural products supply and demand. The resulting welfare measures are based on predicted changes in market prices and production costs. Models also exist to measure benefits to silvicultural producers and consumers. However, these models have not been adapted for use in analyzing ozone related forest impacts. As such, our analysis (to be completed for the Supplemental Analysis) provides monetized estimates of agricultural benefits, and a discussion of the impact of ozone changes on forest productivity, but does not monetize commercial forest related benefits.

4.1.6.2.1 Agricultural Benefits. Laboratory and field experiments have shown reductions in yields for agronomic crops exposed to ozone, including vegetables (e.g., lettuce) and field crops (e.g., cotton and wheat). The most extensive field experiments, conducted under the National Crop Loss Assessment Network (NCLAN) examined 15 species and numerous cultivars. The

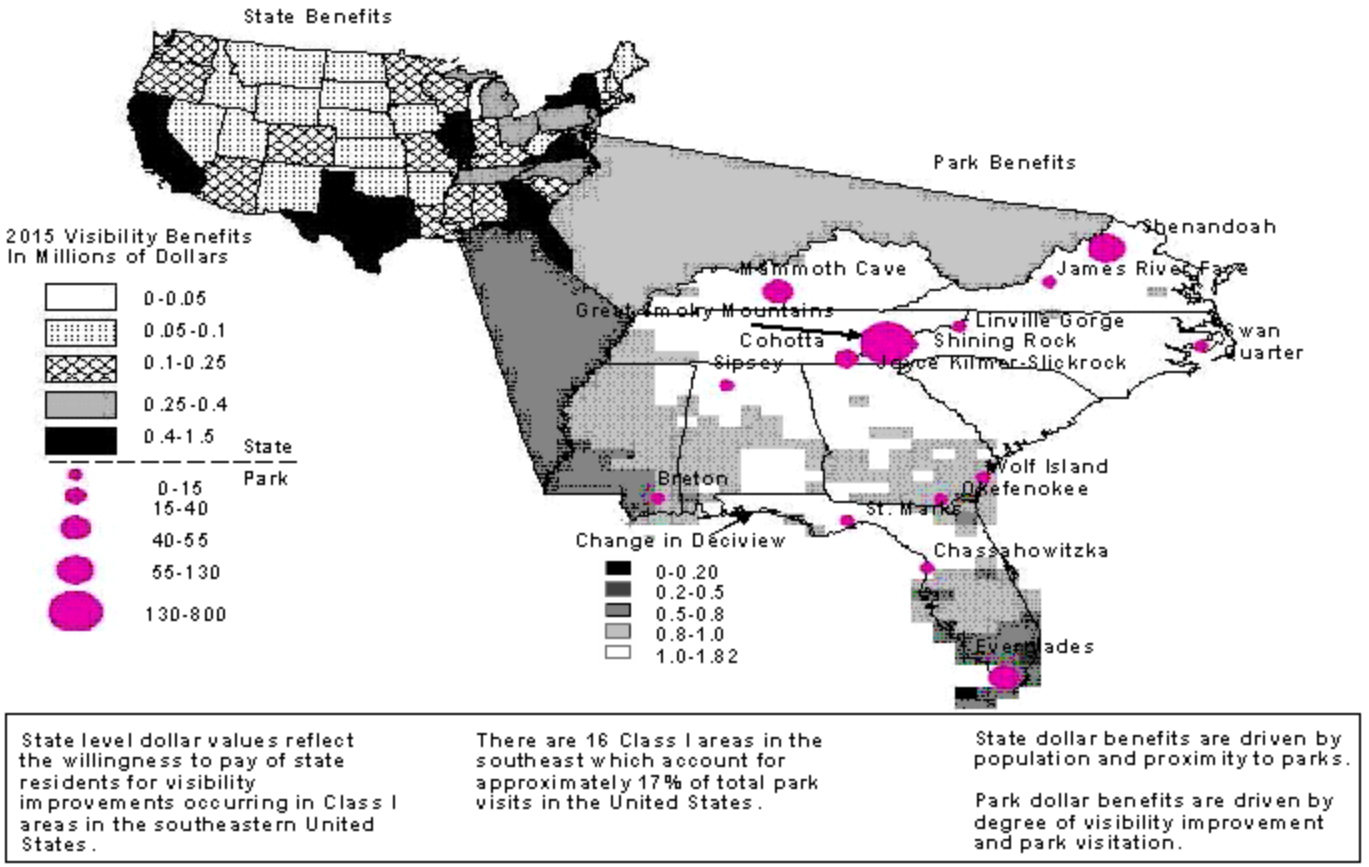


Figure 4.2. Visibility Improvements in Southeastern Class I Areas

NCLAN results show that "several economically important crop species are sensitive to ozone levels typical of those found in the U.S." (US EPA, 1996). In addition, economic studies have shown a relationship between observed ozone levels and crop yields (Garcia, et al., 1986). Due to data limitations, we were unable to assess ozone-related agricultural benefits associated with the proposed IAQR. However, we will be assessing these benefits for the Supplemental Analysis and for the analysis of the final IAQR.

4.1.6.2.2 Forestry Benefits. Ozone also has been shown conclusively to cause discernible injury to forest trees (US EPA, 1996; Fox and Mickler, 1996). In our previous analysis of the HD Engine/Diesel Fuel rule, we were able to quantify the effects of changes in ozone concentrations on tree growth for a limited set of species. Due to data limitations, we were not able to quantify such impacts for this analysis. We plan to assess both physical impacts on tree growth and the economic value of those physical impacts in our analysis of the final rule. We will use econometric models of forest product supply and demand to estimate changes in prices, producer profits and consumer surplus. These benefits will be estimated for the final IAQR.

4.1.6.2.3 Other Vegetation Effects. An additional welfare benefit expected to accrue as a result of reductions in ambient ozone concentrations in the U.S. is the economic value the public receives from reduced aesthetic injury to forests. There is sufficient scientific information available to reliably establish that ambient ozone levels cause visible injury to foliage and impair the growth of some sensitive plant species (US EPA, 1996c, p. 5-521). However, present analytic tools and resources preclude EPA from quantifying the benefits of improved forest aesthetics.

Urban ornamentals represent an additional vegetation category likely to experience some degree of negative effects associated with exposure to ambient ozone levels and likely to impact large economic sectors. In the absence of adequate exposure-response functions and economic damage functions for the potential range of effects relevant to these types of vegetation, no direct quantitative economic benefits analysis has been conducted. It is estimated that more than $20 billion (1990 dollars) are spent annually on landscaping using ornamentals (Abt Associates, 1995), both by private property owners/tenants and by governmental units responsible for public areas. This is therefore a potentially important welfare effects category. However, information and valuation methods are not available to allow for plausible estimates of the percentage of these expenditures that may be related to impacts associated with ozone exposure.

The EGU standards, by reducing NO_X emissions, will also reduce nitrogen deposition on agricultural land and forests. There is some evidence that nitrogen deposition may have positive effects on agricultural output through passive fertilization. Holding all other factors constant, farmers' use of purchased fertilizers or manure may increase as deposited nitrogen is reduced. Estimates of the potential value of this possible increase in the use of purchased fertilizers are not available, but it is likely that the overall value is very small relative to other health and welfare effects. The share of nitrogen requirements provided by this deposition is small, and the marginal cost of providing this nitrogen from alternative sources is quite low. In some areas, agricultural lands suffer from nitrogen over-saturation due to an abundance of on-farm nitrogen production, primarily from animal manure. In these areas, reductions in atmospheric deposition of nitrogen represent additional agricultural benefits.

Information on the effects of changes in passive nitrogen deposition on forests and other terrestrial ecosystems is very limited. The multiplicity of factors affecting forests, including other potential stressors such as ozone, and limiting factors such as moisture and other nutrients, confound assessments of marginal changes in any one stressor or nutrient in forest ecosystems. However, reductions in deposition of nitrogen could have negative effects on forest and vegetation growth in ecosystems where nitrogen is a limiting factor (US EPA, 1993).

On the other hand, there is evidence that forest ecosystems in some areas of the United States are nitrogen saturated (US EPA, 1993). Once saturation is reached, adverse effects of additional nitrogen begin to occur such as soil acidification which can lead to leaching of nutrients needed for plant growth and mobilization of harmful elements such as aluminum. Increased soil acidification is also linked to higher amounts of acidic runoff to streams and lakes and leaching of harmful elements into aquatic ecosystems.

4.1.6.3 Benefits from Reductions in Materials Damage

The preliminary control options that we modeled are expected to produce economic benefits in the form of reduced materials damage. There are two important categories of these benefits. Household soiling refers to the accumulation of dirt, dust, and ash on exposed surfaces. Criteria pollutants also have corrosive effects on commercial/industrial buildings and structures of cultural and historical significance. The effects on historic buildings and outdoor works of art are of particular concern because of the uniqueness and irreplaceability of many of these objects.

Previous EPA benefit analyses have been able to provide quantitative estimates of household soiling damage. Consistent with SAB advice, we determined that the existing data (based on consumer expenditures from the early 1970's) are too out of date to provide a reliable enough estimate of current household soiling damages (EPA-SAB-Council-ADV-003, 1998) to include in our base estimate. We calculate household soiling damages in a sensitivity estimate that will be completed as part of the Supplemental Analysis.

EPA is unable to estimate any benefits to commercial and industrial entities from reduced materials damage. Nor is EPA able to estimate the benefits of reductions in PM-related damage to historic buildings and outdoor works of art. Existing studies of damage to this latter category in Sweden (Grosclaude and Soguel, 1994) indicate that these benefits could be an order of magnitude larger than household soiling benefits.

4.1.6.4 Benefits from Reduced Ecosystem Damage

The effects of air pollution on the health and stability of ecosystems are potentially very important, but are at present poorly understood and difficult to measure. The reductions in NO_X caused by the final rule could produce significant benefits. Excess nutrient loads, especially of nitrogen, cause a variety of adverse consequences to the health of estuarine and coastal waters. These effects include toxic and/or noxious algal blooms such as brown and red tides, low (hypoxic) or zero (anoxic) concentrations of dissolved oxygen in bottom waters, the loss of submerged aquatic vegetation due to the light-filtering effect of thick algal mats, and fundamental shifts in phytoplankton community structure (Bricker et al., 1999).

Direct functions relating changes in nitrogen loadings to changes in estuarine benefits are not available. The preferred WTP based measure of benefits depends on the availability of these functions and on estimates of the value of environmental responses. Because neither appropriate functions nor sufficient information to estimate the marginal value of changes in water quality exist at present, calculation of a WTP measure is not possible.

If better models of ecological effects can be defined, EPA believes that progress can be made in estimating WTP measures for ecosystem functions. These estimates would be superior to avoided cost estimates in placing economic values on the welfare changes associated with air pollution damage to ecosystem health. For example, if nitrogen or sulfate loadings can be linked to measurable and definable changes in fish populations or definable indexes of biodiversity, then CV studies can be designed to elicit individuals' WTP for changes in these effects. This is an important area for further research and analysis, and will require close collaboration among air quality modelers, natural scientists, and economists.

4.2 Benefits Analysis—Results

Applying the impact and valuation functions described in Section C to the estimated changes in ozone and PM described in Section B yields estimates of the changes in physical damages (i.e. premature mortalities, cases, admissions, change in light extinction, etc.) and the associated monetary values for those changes. Estimates of physical health impacts are presented in Table 4-16. Monetized values for both health and welfare endpoints are presented in Table 4-17, along with total aggregate monetized benefits. All of the monetary benefits are in constant year 1999 dollars.

Not all known PM- and ozone-related health and welfare effects could be quantified or monetized. The monetized value of these unquantified effects is represented by adding an unknown "B" to the aggregate total. The estimate of total monetized health benefits is thus equal to the subset of monetized PM- and ozone-related health and welfare benefits plus B, the sum of the nonmonetized health and welfare benefits.

Total monetized benefits are dominated by benefits of mortality risk reductions. The primary analysis estimate projects that the proposed rule will result in 9,600 avoided premature deaths in 2010 and 13,000 avoided premature deaths in 2015. The increase in benefits from 2010 to 2015 reflects additional emission reductions from the standards, as well as increases in total population and the average age (and thus baseline mortality risk) of the population. Note that unaccounted for changes in baseline mortality rates over time may lead to reductions in the estimated number of avoided premature mortalities.

Table 4-16. Reductions in Incidence of Adverse Health Effects Associated with Reductions in Particulate Matter and Ozone Associated with the Proposed IAQR[a]

Endpoint	2010	2015
PM-related Endpoints		
Premature mortality[b]	9,600	13,000
Long-term exposure (adults, 30 and over) Long-term exposure (infant, <1 yr)	22	29
Chronic bronchitis (adults, 26 and over)	5,200	6,900
Non-fatal myocardial infarctions (adults, 18 and older)	13,000	18,000
Hospital admissions—Respiratory (all ages)[c]	4,200	5,800
Hospital admissions—Cardiovascular (adults, >18)[d]	3,700	5,000
Emergency Room Visits for Asthma (18 and younger)	7,000	9,200
Acute bronchitis (children, 8-12)	12,000	16,000
Lower respiratory symptoms (children, 7-14)	140,000	190,000
Upper respiratory symptoms (asthmatic children, 9-18)	490,000	620,000
Asthma Exacerbations (asthmatic children, 6-18)	190,000	240,000
Work loss days (adults, 18-65)	1,000,000	1,300,000
Minor restricted activity days (adults, age 18-65)	6,100,000	7,900,000
Ozone-related Endpoints		
Hospital Admissions—Respiratory Causes (adults, 65 and older)[e]	630	1,500
Hospital Admissions—Respiratory Causes (children, under 2 years)	380	840
Emergency Room Visits for Asthma (all ages)	120	250
Minor restricted activity days (adults, age 18-65)	280,000	610,000
School absence days (children, age 6-18)	180,000	390,000

[a] Incidences are rounded to two significant digits.

[b] Premature mortality associated with ozone is not separately included in this analysis. It is assumed that the Impact function for premature mortality captures both PM mortality benefits and any mortality benefits associated with other air pollutants.

[c] Respiratory hospital admissions for PM includes admissions for COPD, pneumonia, and asthma.

[d] Cardiovascular hospital admissions for PM includes total cardiovascular and subcategories for ischemic heart disease, dysrhythmias, and heart failure.

[e] Respiratory hospital admissions for ozone includes admissions for all respiratory causes and subcategories for COPD and pneumonia.

Table 4-17. Results of Human Health and Welfare Benefits Valuation for the Proposed IAQR (millions of 1999 dollars)[a,b]

Endpoint	Pollutant	2010	2015
Premature mortality[c]	PM		
Long-term exposure, (adults, >30yrs)			
3% discount rate		$53,000	$77,000
7% discount rate		$50,000	$72,000
Long-term exposure (child <1yr)		$130	$180
Chronic bronchitis (adults, 26 and over)	PM	$1,900	$2,700
Non-fatal myocardial infarctions	PM		
3% discount rate		$1,100	$1,500
7% discount rate		$1,000	$1,400
Hospital Admissions from Respiratory Causes	O_3 and PM	$85	$130
Hospital Admissions from Cardiovascular Causes	PM	$78	$110
Emergency Room Visits for Asthma	O_3 and PM	$2.0	$2.6
Acute bronchitis (children, 8-12)	PM	$4.3	$5.7
Lower respiratory symptoms (children, 7-14)	PM	$2.3	$3.0
Upper respiratory symptoms (asthmatic children, 9-11)	PM	$13	$17
Asthma exacerbations	PM	$8.0	$10
Work loss days (adults, 18-65)	PM	$140	$170
Minor restricted activity days (adults, age 18-65)	O_3 and PM	$320	$440
School absence days (children, age 6-11)	O_3	$13	$28
Worker productivity (outdoor workers, age 18-65)	O_3	$8.1	$17
Recreational visibility (Southeastern Class I areas)	PM	$880	$1,400
Monetized Total[d]	O_3 and PM		
Base estimate			
3% discount rate		$58,000+B	$84,000+B
7% discount rate		$54,000+B	$79,000+B

[a] Monetary benefits are rounded to two significant digits.

[b] Monetary benefits are adjusted to account for growth in real GDP per capita between 1990 and the analysis year (2010 or 2015).

[c] Valuation assumes the 5 year distributed lag structure described earlier. Results reflect the use of two different discount rates; a 3 percent rate which is recommended by EPA's Guidelines for Preparing Economic Analyses (US EPA, 2000c), and 7 percent which is recommended by OMB Circular A-94 (OMB, 1992).

[d] B represents the monetary value of the nonmonetized health and welfare benefits. A detailed listing of unquantified PM, ozone, and mercury related health effects is provided in Table XI-B.1.

Our estimate of total monetized benefits in 2010 for the proposed rule is $58 billion using a 3 percent discount rate and $54 billion using a 7 percent discount rate. In 2015, the monetized benefits are estimated at $84 billion using a 3 percent discount rate and $79 billion using a 7 percent discount rate. Health benefits account for 98 percent of total benefits, mainly because we are unable to quantify most of the non-health benefits. The monetized benefit associated with reductions in the risk of premature mortality, which accounts for $53 billion in 2010 and $77 billion in 2015, is over 90 percent of total monetized health benefits. The next largest benefit is for reductions in chronic illness (CB and non-fatal heart attacks), although this value is more than an order of magnitude lower than for premature mortality. Hospital admissions for respiratory and cardiovascular causes, visibility, minor restricted activity days, work loss days, school absence days, and worker productivity account for the majority of the remaining benefits. The remaining categories account for less than $10 million each, however, they represent a large number of avoided incidences affecting many individuals.

A comparison of the incidence table to the monetary benefits table reveals that there is not always a close correspondence between the number of incidences avoided for a given endpoint and the monetary value associated with that endpoint. For example, there are 100 times more work loss days than premature mortalities, yet work loss days account for only a very small fraction of total monetized benefits. This reflects the fact that many of the less severe health effects, while more common, are valued at a lower level than the more severe health effects. Also, some effects, such as hospital admissions, are valued using a proxy measure of WTP. As such the true value of these effects may be higher than that reported in Table 4-16.

Ozone benefits are in aggregate positive for the nation. However, due to ozone increases occurring during certain hours of the day in some urban areas, there is a dampening of overall ozone benefits in both 2010 and 2015, although the net incidence and benefits estimates for all health effects categories are net positive. Overall, ozone benefits are low relative to PM benefits for similar endpoint categories because of the increases in ozone concentrations during some hours of some days in certain urban areas.

4.3 Discussion

This analysis has estimated the health and welfare benefits of reductions in ambient concentrations of particulate matter and ozone resulting from reduced emissions of NOx and SO_2 from affected EGUs. The result suggests there will be significant health and welfare

benefits arising from the regulation of emissions from EGUs in the U.S. Our estimate that 13,000 premature mortalities would be avoided in 2015, when emission reductions from the regulation are fully realized, provides additional evidence of the important role that pollution from the EGU sector plays in the public health impacts of air pollution.

To examine the importance of specific assumptions and analytical choices we made for this analysis, we will be providing a number of sensitivity analyses in an appendix to be completed for the upcoming Supplemental Analysis of the proposed rule. In addition, there are other uncertainties that we could not quantify, such as the importance of unquantified effects and uncertainties in the modeling of ambient air quality. Inherent in any analysis of future regulatory programs are uncertainties in projecting atmospheric conditions, and source-level emissions, as well as population, health baselines, incomes, technology, and other factors. The assumptions used to capture these elements are reasonable based on the available evidence. However, data limitations prevent an overall quantitative estimate of the uncertainty associated with estimates of total economic benefits. If one is mindful of these limitations, the magnitude of the benefit estimates presented here can be useful information in expanding the understanding of the public health impacts of reducing air pollution from EGUs.

The U.S. EPA will continue to evaluate new methods and models and select those most appropriate for the estimation the health benefits of reductions in air pollution. It is important to continue improving benefits transfer methods in terms of transferring economic values and transferring estimated Impact functions. The development of both better models of current health outcomes and new models for additional health effects such as asthma and high blood pressure will be essential to future improvements in the accuracy and reliability of benefits analyses (Guo et al., 1999; Ibald-Mulli et al., 2001). Enhanced collaboration between air quality modelers, epidemiologists, and economists should result in a more tightly integrated analytical framework for measuring health benefits of air pollution policies. The Agency welcomes comments on how we can improve the quantification and monetization of health and welfare effects and on methods for characterizing uncertainty in our estimates.

SECTION 5

QUALITATIVE ASSESSMENT OF NONMONETIZED BENEFITS

5.1 Introduction

This proposal will result in benefits in addition to the enumerated human health and welfare benefits resulting from reductions in ambient levels of PM and ozone. This rule will also result in benefits that we were unable to monetize. This chapter discusses welfare benefits associated with reduced acid deposition, reduced eutrophication in water bodies, and the reduced health and welfare effects due to the deposition of mercury. Welfare benefits including visibility benefits, agricultural, forestry and other benefits due to reductions in ozone levels, and benefits from reductions in materials damage are discussed in chapter 4 of this report. In contrast to the benefits discussed, it is also possible that this proposal will lessen the benefits of passive fertilization for forest and terrestrial ecosystems where nutrients are a limiting factor and for some croplands.

5.2 Atmospheric Deposition of Sulfur and Nitrogen—Impacts on Aquatic, Forest, and Coastal Ecosystems

Atmospheric deposition of sulfur and nitrogen, more commonly known as acid rain, occurs when emissions of SO_2 and NO_x react in the atmosphere (with water, oxygen, and oxidants) to form various acidic compounds. These acidic compounds fall to earth in either a wet form (rain, snow, and fog) or a dry form (gases and particles). Prevailing winds transport the acidic compounds hundreds of miles, often across state and national borders. Acidic compounds (including small particles such as sulfates and nitrates) cause many negative environmental effects. These pollutants

- acidify lakes and streams,
- harm sensitive forests, and
- harm sensitive coastal ecosystems.

The effect of atmospheric deposition of acids on freshwater and forest ecosystems depends largely on the ecosystem's ability to neutralize the acid (Driscoll et al., 2001). This is

referred to as an ecosystem's acid neutralizing capacity (ANC). Acid neutralization occurs when positively charged ions such as calcium, potassium, sodium, and magnesium, collectively known as base cations, are released. As water moves through a watershed, two important chemical processes act to neutralize acids. The first involves cation exchange in soils, a process by which hydrogen ions from the acid deposition displace other cations from the surface of soil particles, releasing these cations to soil and surface water. The second process is mineral weathering, where base cations bound in the mineral structure of rocks are released as the minerals gradually break down over long time periods. As the base cations are released by weathering, they neutralize acidity and increase the pH level in soil water and surface waters. Acid deposition, because it consists of acid anions (e.g., sulfate, nitrate), leaches some of the accumulated base cation reserves from the soils into drainage waters. The leaching rate of these base cations may accelerate to the point where it significantly exceeds the resupply via weathering (Driscoll et al., 2001).

Soils, forests, surface waters and aquatic biota (fish, algae, and the rest), and coastal ecosystems share water, nutrients, and other essential ecosystem components and are inextricably linked by the chemical processes described above. For example, the same base cations that help to neutralize acidity in lakes and streams are also essential nutrients in forest soils, meaning that cation depletion both increases freshwater acidification and decreases forest productivity. Similarly, the same nitrogen atom that contributes to stream acidification can ultimately contribute to coastal eutrophication as it travels downstream to an estuarine environment. Therefore, to understand the full effects of atmospheric deposition, it is necessary to recognize the interactions between all of these systems.

5.2.1 Freshwater Acidification

Acid deposition causes acidification of surface waters. In the 1980s, acid rain was found to be the dominant cause of acidification in 75 percent of acidic lakes and 50 percent of acidic streams. Areas especially sensitive to acidification include portions of the Northeast (particularly the Adirondack and Catskill Mountains, portions of New England, and streams in the mid-Appalachian highlands) and Southeastern streams. Some high elevation Western lakes, particularly in the Rocky Mountains, have become acidic, especially during snowmelt. However, although many Western lakes and streams are sensitive to acidification, they are not subject to continuously high levels of acid deposition and so have not become chronically acidified (NAPAP, 1990).

ANC, a key indicator of the ability of the water and watershed soil to neutralize the acid deposition it receives, depends largely on the watershed's physical characteristics: geology, soils, and size. Waters that are sensitive to acidification tend to be located in small watersheds that have few alkaline minerals and shallow soils. Conversely, watersheds that contain alkaline minerals, such as limestone, tend to have waters with a high ANC.

As acidity increases, aluminum leached from the soil flows into lakes and streams and can be toxic to aquatic species. The lower pH levels and higher aluminum levels that result from acidification make it difficult for some fish and other aquatic species to survive, grow, and reproduce. In some waters, the number of species of fish able to survive has been directly correlated to water acidity. Acidification can also decrease fish population density and individual fish size (U.S. Department of the Interior 2003).

Recent watershed mass balance studies in the Northeast reveal that loss of sulfate from the watershed exceeds atmospheric sulfur deposition (Driscoll et al., 2001). This suggests that these soils have become saturated with sulfur, meaning that the supply of sulfur from deposition exceeds the sulfur demands of the ecosystem. As a result, sulfur is gradually being released or leached from the watershed into the surface waters as sulfate. Scientists now expect that the release of sulfate that previously accumulated in watersheds will delay the recovery of surface waters in the Northeast that is anticipated in response to the recent SO_2 emission controls (Driscoll et al., 2001).

A recent study at a stream in the Catskill Mountains found that stream nitrate concentrations were positively correlated to mean annual air temperature but not to annual nitrogen deposition (Murdoch et al., 1998). This research suggests that, in nitrogen-saturated soils, microbial processes (nitrogen mineralization and nitrification), which are sensitive to changes in temperature and moisture, are the primary factors controlling nitrate leaching, rather than atmospheric deposition or vegetation uptake of nitrogen. Therefore, declines in nitrogen deposition in nitrogen-saturated soils may not immediately lead to improvements in stream water chemistry (Murdoch et al., 1998).

A major study of the ecological response to acidification is taking place in the Bear Brook Watershed in Maine. Established in 1986 as part of the EPA's Watershed Manipulation Project, the project has found that experimental additions of sulfur and nitrogen to the watershed increased the concentrations of both sulfate and nitrate in the West Bear Brook stream. Stream water concentrations of several other ions, including base cations, aluminum, and ANC, changed substantially as well (Norton et al., 1999). During the first

year of treatment, 94 percent of the nitrogen added experimentally to the Bear Brook watershed was retained, while the remainder leached into streams as nitrate. Nitrogen retention decreased to about 82 percent in subsequent years (Kahl et al., 1993, 1999). Although the forest ecosystem continued to accumulate nitrogen, nitrate leaching into the stream continued at elevated levels throughout the length of the experiment. This nitrate contributed to both episodic and chronic acidification of the stream. This and other similar studies have allowed scientists to quantify acidification and recovery relationships in eastern watersheds in much more detail than was possible in 1990.

The Appalachian Mountain region receives some of the highest rates of acid deposition in the United States (Herlihy et al., 1993). The acid-base status of stream waters in forested upland watersheds in the Appalachian Mountains was extensively investigated in the early 1990s (e.g., Church et al. [1992], Herlihy et al. [1993], Webb et al. [1994], van Sickle and Church [1995]). A more recent assessment of the southern Appalachian region from West Virginia to Alabama identified watersheds that are sensitive to acid deposition using geologic bedrock and the associated buffering capacity of soils to neutralize acid. The assessment found that approximately 59 percent of all trout stream length in the region is in areas that are highly vulnerable to acidification, and that 27 percent is in areas that are moderately vulnerable (SAMAB, 1996). Another study estimated that 18 percent of potential brook trout streams in the mid-Appalachian Mountains are too acidic for brook trout survival (Herlihy et al., 1996). Perhaps the most important study of acid-base chemistry of streams in the Appalachian region in recent years has been the Virginia Trout Stream Sensitivity Study (Webb et al., 1994). Trend analyses of these streams indicate that few long-term sampling sites are recovering from acidification, most are continuing to acidify, and the continuing acidification is at levels that are biologically significant for brook trout populations (Webb et al., 2000).

5.2.1.1 Water/Watershed Modeling

Researchers have used models to help them understand and predict atmospheric, environmental, and human health responses to acid deposition for well over 20 years. Since 1990, watershed modeling capabilities have also improved as researchers are continuing to refine and expand models that project acidification of waterbodies. Unlike the response of air quality and deposition to changes in emissions, lakes and streams take years to decades to fully reflect reductions in acid deposition. In some cases, soil chemistry has been significantly altered and ions must either build up or be leached out before the chemistry can

return to its pre-acidification status. Therefore, lake and stream conditions are presented for 2030.

5.2.1.2 Description of the MAGIC Model and Methods

A number of mathematical models of soil and surface water acidification in response to atmospheric deposition were developed in the early 1980s (e.g., Christopherson and Wright [1981]; Christopherson et al. [1982]; Schnoor et al. [1984]; Booty and Kramer [1984]; Goldstein et al. [1984]; Cosby et al. [1985a,b,c]). These models were based on process-level information about the acidification process and were built for a variety of purposes ranging from estimating transient water quality responses for individual storm events to estimating chronic acidification of soils and base flow surface water. One of these models (MAGIC—the Model of Acidification of Groundwater In Catchments; Cosby et al. [1985a,b,c]) has been in use now for more than 15 years. MAGIC has been applied extensively in North America and Europe to both individual sites and regional networks of sites and has also been used in Asia, Africa, and South America. The utility of MAGIC for simulating a variety of water and soil acidification responses at the laboratory, plot, hillslope, and catchment scales has been tested using long-term monitoring data and experimental manipulation data. MAGIC has been widely used in policy and assessment activities in the United States and in several countries in Europe.

5.2.1.3 Model Structure

MAGIC is a lumped-parameter model of intermediate complexity, developed to predict the long-term effects of acidic deposition on surface water chemistry. The model simulates soil solution chemistry and surface water chemistry to predict the monthly and annual average concentrations of the major ions in these waters. MAGIC consists of the following: 1) a section in which the concentrations of major ions are assumed to be governed by simultaneous reactions involving sulfate adsorption, cation exchange, dissolution-precipitation-speciation of aluminum, and dissolution-speciation of inorganic carbon; and 2) a mass balance section in which the flux of major ions to and from the soil is assumed to be controlled by atmospheric inputs, chemical weathering, net uptake, and loss in biomass and losses to runoff. At the heart of MAGIC is the size of the pool of exchangeable base cations in the soil. As the fluxes to and from this pool change over time owing to changes in atmospheric deposition, the chemical equilibria between soil and soil solution shift to give changes in surface water chemistry. The degree and rate of change of surface water acidity thus depend both on flux factors and the inherent characteristics of the affected soils.

Cation exchange is modeled using equilibrium (Gaines-Thomas) equations with selectivity coefficients for each base cation and aluminum. Sulfate adsorption is represented by a Langmuir isotherm. Aluminum dissolution and precipitation are assumed to be controlled by equilibrium with a solid phase of aluminum trihydroxide. Aluminum speciation is calculated by considering hydrolysis reactions as well as complexation with sulfate, fluoride, and dissolved organic compounds. Effects of carbon dioxide on pH and on the speciation of inorganic carbon are computed from equilibrium equations. Organic acids are represented in the model as tri-protic analogues. Weathering rates are assumed to be constant. Two alternate mechanisms are offered for simulation of nitrate and ammonium in soils: either 1) first order equations representing net uptake and retention or 2) a set of equations and compartments describing process-based nitrogen dynamics in soils controlled by soil nitrogen pools. Input-output mass balance equations are provided for base cations and strong acid anions, and charge balance is required for all ions in each compartment. Given a description of the historical, current, and expected future deposition at a site, the model equations are solved numerically to give long-term reconstructions of surface water chemistry (for complete details of the model see Cosby et al. [1985 a,b,c], [2001]).

MAGIC has been used to reconstruct the history of acidification, to examine current patterns of recovery, and to simulate the future trends in stream water acidity in both individual catchment and regional applications at a large number of sites across North America and Europe (e.g., Beier et al. [1995]; Cosby et al. [1985b,1990, 1995, 1996, 1998]; Ferrier, et al. [2001]; Hornberger et al. [1989]; Jenkins et al. [1990]; Moldan et al. [1998]; Norton et al. [1992]; Whitehead et al. [1988, 1997]; Wright et al. [1990, 1994, 1998]).

5.2.1.4 Model Implementation

Atmospheric deposition and net uptake-release fluxes for the base cations and strong acid anions are required as inputs to the model. These inputs are generally assumed to be uniform over the catchment. Atmospheric fluxes are calculated from concentrations of the ions in precipitation and the rainfall volume into the catchment. The atmospheric fluxes of the ions must be corrected for dry deposition of gas, particulates, and aerosols and for inputs in cloud/fog water. The volume discharge for the catchment must also be provided to the model. In general, the model is implemented using average hydrologic conditions and meteorological conditions in annual or seasonal simulations (i.e., mean annual or mean monthly deposition); precipitation and lake discharge are used to drive the model. Values for soil and surface water temperature, partial pressure of carbon dioxide, and organic acid concentrations must also be provided at the appropriate temporal resolution.

As implemented in this project, the model is a two-compartment representation of a catchment. Atmospheric deposition enters the soil compartment, and the equilibrium equations are used to calculate soil water chemistry. The water is then routed to the stream compartment, and the appropriate equilibrium equations are reapplied to calculate runoff chemistry.

Once initial conditions (initial values of variables in the equilibrium equations) have been established, the equilibrium equations are solved for soil water and surface water concentrations of the remaining variables. These concentrations are used to calculate the lake discharge output fluxes of the model for the first time step. The mass balance equations are (numerically) integrated over the time step, providing new values for the total amounts of base cations and strong acid anions in the system. These in turn are used to calculate new values of the remaining variables, new lake discharge fluxes, and so forth. The output from MAGIC is thus a time trace for all major chemical constituents for the period of time chosen for the integration.

5.2.1.5 Calibration Procedure

The aggregated nature of the model requires that it be calibrated to observed data from a system before it can be used to examine potential system response. Calibration is achieved by setting the values of certain parameters within the model that can be directly measured or observed in the system of interest (called "fixed" parameters). The model is then run (using observed atmospheric and hydrologic inputs) and the simulated values of surface water and soil chemical variables (called "criterion" variables) are compared to observed values of these variables. If the observed and simulated values differ, the values of another set of parameters in the model (called "optimized" parameters) are adjusted to improve the fit. After a number of iterations, the simulated-minus-observed values of the criterion variables usually converge to zero (within some specified tolerance). The model is then considered calibrated. If new assumptions (or values) for any of the fixed variables or inputs to the model are subsequently adopted, the model must be recalibrated by readjusting the optimized parameters until the simulated-minus-observed values of the criterion variables again fall within the specified tolerance.

Calibrations are based on volume weighted mean annual or seasonal fluxes for a given period of observation. The length of the period of observation used for calibration is not arbitrary. Model output will be more reliable if the annual flux estimates used in calibration are based on a number of years rather than just 1 year. There is a lot of year-to-

year variability in atmospheric deposition and catchment runoff. Averaging over a number of years reduces the likelihood that an "outlier" year (very dry, etc.) is the primary data on which model forecasts are based. On the other hand, averaging over too long a period may remove important trends in the data that the model needs to simulate.

The calibration procedure requires that stream water quality, soil chemical and physical characteristics, and atmospheric deposition data be available for each catchment. The water quality data needed for calibration are the concentrations of the individual base cations (Ca, Mg, Na, and K) and acid anions (Cl, SO_4, and NO_3) and the pH. The soil data used in the model include soil depth and bulk density, soil pH, soil cation-exchange capacity, and exchangeable bases on the soil (Ca, Mg, Na, and K). The atmospheric deposition inputs to the model must be estimates of total deposition, not just wet deposition. In some instances, direct measurements of either atmospheric deposition or soil properties may not be available for a given site with stream water data. In these cases, the required data can often be estimated by assigning soil properties based on some landscape classification of the catchment and assigning deposition using model extrapolations from some national or regional atmospheric deposition monitoring network.

Soil Physical and Chemical Properties. Soil data for model calibration are usually derived as a really averaged values of soil parameters within a catchment. If soils data for a given location are vertically stratified, the soils data for the individual soil horizons at that sampling site can be aggregated based on horizon, depth, and bulk density to obtain single vertically aggregated values for the site, or the stratified data can be used directly in the model.

Total Atmospheric Deposition. Total atmospheric deposition consists of three components: wet deposition, the flux of ions occurring in precipitation; dry deposition, resulting from gaseous and particulate fluxes; and cloud/fog deposition (which can be particularly important in mountainous inland areas or moderate highlands in areas adjacent to oceans or seas). Estimates of precipitation volume and ionic concentrations in precipitation can be used to calculate wet deposition for a site. Observations of dry deposition or cloud/fog deposition are very infrequent. The approach usually used to quantify these components relies on some estimate of the ratio of estimated total deposition to the observed wet deposition for important ions (e.g., sulphate, nitrate, and ammonium ions). These ratios (called dry deposition factors) are then used to calculate total deposition from the observed wet deposition data.

Historical Loading. Calibration of the model (and estimation of the historical changes at the sites) requires a temporal sequence of historical anthropogenic deposition. Our current understanding of ecosystem responses to acidic deposition suggests that future ecosystem responses can be strongly conditioned by historical acidic loadings. Thus, as part of the model calibration process, the model should be constrained by some measure of historical deposition to the site. However, such long-term, continuous historical deposition data do not exist. The usual approach is to use historical emissions data as a surrogate for deposition. The emissions for each year in the historical period can be normalized to emissions in a reference year (a year for which observed deposition data are available). Using this scaled sequence of emissions, historical deposition can be estimated by multiplying the total deposition estimated for each site in reference year by the emissions scale factor for any year in the past to obtain deposition for that year.

5.2.1.6 MAGIC Modeling Results

Watershed modeling undertaken for IAQR projects that, under IAQR, 1 percent of northeastern lakes would be chronically acidic in 2030. In contrast, the same model used to analyze existing control programs projects 6 percent of northeastern lakes would be chronically acidic in 2030. The modeling projects that, under IAQR, 28 percent of northeastern lakes would be episodically acidic in 2030, compared to 25 percent in 2030 under existing control programs. For Adirondack lakes, a subset of northeastern lakes, the signals of surface water chemical recovery are much stronger. Under IAR, no Adirondack lakes would be chronically acidic, and 64 percent would be episodically acidic in 2030, as opposed to 12 percent chronically acidic and 52 percent episodically acidic in 2030 under current control programs.

Because of the age and types of soils in many high elevation areas of the southeast, streams in that region are more frequently characterized by a delayed response to changes in deposition. For the ecosystems modeled in this region, 17 percent of streams are currently chronically acidic, and this level stays the same under IAQR 2030; the proportion of episodically acidic streams increases from 19 percent under current conditions to 23 percent under IQAR, which reflects a decrease in the proportion of nonacidic streams from 64 percent under current conditions to 60 percent under IQAR in 2030. It is important to note that, under the Base Case, the proportion of nonacidic streams decreases even further, dropping from 64 percent under current conditions to 58 percent in 2030. Thus, in the southeast, IQAR would slow the deterioration of stream health (episodically acidic) expected

under the Base Case and would prevent additional streams from becoming chronically acidic. Results of the MAGIC modeling are summarized in Table 5-1.

5.2.2 Forest Ecosystems

Our current understanding of the effects of acid deposition on forest ecosystems has come to focus increasingly on the effects of biogeochemical processes that affect plant uptake, retention, and cycling of nutrients within forested ecosystems. Research results from the 1990s indicate that documented decreases in base cations (calcium, magnesium, potassium, and others) from soils in the northeastern and southeastern United States are at least partially attributable to acid deposition (Lawrence et al., 1997; Huntington et al., 2000). Base cation depletion is a cause for concern because of the role these ions play in acid neutralization and, in the case of calcium, magnesium, and potassium, their importance as essential nutrients for tree growth. It has been known for some time that depletion of base cations from the soil interferes with the uptake of calcium by roots in forest soils (Shortle and Smith 1988). Recent research indicates it also leads to aluminum mobilization (Lawrence et al., 1995), which can have harmful effects on fish (US Dept. of Interior 2003).

The plant physiological processes affected by reduced calcium availability include cell wall structure and growth, carbohydrate metabolism, stomatal regulation, resistance to plant pathogens, and tolerance of low temperatures (DeHayes et al., 1999). Soil structure, macro and micro fauna, decomposition rates, and nitrogen metabolism are also important processes that are significantly influenced by calcium levels in soils. The importance of calcium as an indicator of forest ecosystem function is due to its diverse physiological roles, coupled with the fact that calcium mobility in plants is very limited and can be further reduced by tree age, competition, and reduced soil water supply (McLaughlin and Wimmer 1999).

A clear link has now been established in red spruce stands between acid deposition, calcium supply, and sensitivity to abiotic stress. Red spruce uptake and retention of calcium is affected by acid deposition in two main ways: leaching of important stores of calcium from needles (DeHayes et al., 1999) and decreased root uptake of calcium due to calcium depletion from the soil and aluminum mobilization (Smith and Shortle, 2001; Shortle et al., 1997; Lawrence et al., 1997). Acid deposition leaches calcium from mesophyll cells of

1-year old red spruce needles (Schaberg et al., 2000), which in turn reduces freezing tolerance (DeHayes et al., 1999). These changes increase the sensitivity of red spruce to winter injuries under normal winter conditions in the Northeast, result in the loss of needles, and impair the overall health of forest ecosystems (DeHayes et al., 1999). Red spruce must also expend more metabolic energy to acquire calcium from soils in areas with low calcium/aluminum ratios, resulting in slower tree growth (Smith and Shortle, 2001).

Losses of calcium from forest soils and forested watersheds have now been documented as a sensitive early indicator of the soil response to acid deposition for a wide range of forest soils in the United States (Lawrence et al., 1999; Huntington et al., 2000). There is a strong relationship between acid deposition and leaching of base cations from hardwood forest (e.g., maple, oak) soils, as indicated by long-term data on watershed mass balances (Likens et al., 1996; Mitchell et al., 1996), plot- and watershed-scale acidification experiments in the Adirondacks (Mitchell et al., 1994) and in Maine (Norton et al., 1994; Rustad et al., 1996), and studies of soil solution chemistry along an acid deposition gradient from Minnesota to Ohio (MacDonald et al., 1992).

Although sulfate is the primary cause of base cation leaching, nitrate is a significant contributor in watersheds that are nearly nitrogen saturated (Adams et al., 1997). Recent studies of the decline of sugar maples in the Northeast demonstrate a link between low base cation availability, high levels of aluminum and manganese in the soil, and increased levels of tree mortality due to native defoliating insects (Horsley et al., 2000). The chemical composition of leaves and needles may also be altered by acid deposition, resulting in changes in organic matter turnover and nutrient cycling.

5.2.3 Coastal Ecosystems

Since 1990, a large amount of research has been conducted on the impact of nitrogen deposition to coastal waters. It is now known that nitrogen deposition is a significant source of nitrogen to many estuaries (Valigura et al., 2001; Howarth 1998). The amount of nitrogen entering estuaries due to atmospheric deposition varies widely, depending on the size and location of the estuarine watershed and other sources of nitrogen in the watershed. For a handful of estuaries, atmospheric deposition of nitrogen contributes well over 40 percent of the total nitrogen load; however, in most estuaries for which estimates exist, the contribution from atmospheric deposition ranges from 15 to 30 percent. The area with the highest deposition rates stretches from Massachusetts to the Chesapeake Bay and along the central Gulf of Mexico coast.

Nitrogen is often the limiting nutrient in coastal ecosystems. Increasing the levels of nitrogen in coastal waters can cause significant changes to those ecosystems. Approximately 60 percent of estuaries in the United States (65 percent of the estuarine surface area) suffer from overenrichment of nitrogen, a condition known as eutrophication (Bricker et al., 1999). Symptoms of eutrophication include changes in the dominant species of plankton (the primary food source for many kinds of marine life) that can cause algal blooms, low levels of oxygen in the water column, fish and shellfish kills, and cascading population changes up the food chain. Many of the most highly eutrophic estuaries are along the Gulf and mid-Atlantic coasts, overlapping many of the areas with the highest nitrogen deposition, but there are eutrophic estuaries in every region of the coterminous U.S. coastline.

5.3 Benefits of Reducing Mercury Emissions

According to baseline emission estimates, the sources affected by this proposal would emit approximately 45.1 tons of mercury per year in 2010. This estimate is specific to fossil-fired electric generating units in excess of 25 megawatt capacity. The proposed regulation would reduce approximately 10.6 tons of mercury (or 23.5 percent) from the 2010 baseline, 11.8 tons of mercury (or 26.3 percent) from the 2015 baseline, and 14.3 tons (or 32 percent) from the 2020 baseline at affected electric generating units.

Mercury emitted from utilities and other natural and man-made sources is carried by winds through the air and eventually is deposited to water and land. Recent estimates (which are highly uncertain) of annual total global mercury emissions from all sources (natural and anthropogenic) are about 5,000 to 5,500 tons per year (tpy). Of this total, about 1,000 tpy are estimated to be natural emissions and about 2,000 tpy are estimated to be contributions through the natural global cycle of re-emissions of mercury associated with past anthropogenic activity. Current anthropogenic emissions account for the remaining 2,000 tpy. Point sources such as fuel combustion; waste incineration; industrial processes; and metal ore roasting, refining, and processing are the largest point source categories on a world-wide basis. Given the global estimates noted above, U.S. anthropogenic mercury emissions are estimated to account for roughly 3 percent of the global total, and U.S. utilities are estimated to account for about 1 percent of total global emissions. Mercury exists in three forms: elemental mercury, inorganic mercury compounds (primarily mercuric chloride), and organic mercury compounds (primarily methylmercury). Mercury is usually released in an elemental form and later converted into methylmercury by bacteria. Methylmercury is more toxic to humans than other forms of mercury, in part because it is more easily absorbed in the body (EPA, 1996).

If the deposition is directly to a water body, then the processes of aqueous fate, transport, and transformation begin. If deposition is to land, then terrestrial fate and transport processes occur first and then aqueous fate and transport processes occur once the mercury has cycled into a water body. In both cases, mercury may be returned to the atmosphere through resuspension. In water, mercury is transformed to methylmercury through biological processes and for exposures affected by this rulemaking, methylmercury is considered to be the form of greatest concern. Once mercury has been transformed into methylmercury, it can be ingested by the lower trophic level organisms where it can bioaccumulate in fish tissue (i.e., concentrations of mercury remain in the fish's system for a long period of time and accumulates in the fish tissue as predatory fish consume other species in the food chain). Fish and wildlife at the top of the food chain can, therefore, have mercury concentrations that are higher than the lower species, and they can have concentrations of mercury that are higher than the concentration found in the water body itself. In addition, when humans consume fish contaminated with methylmercury, the ingested methymercury is almost completely absorbed into the blood and distributed to all tissues (including the brain); it also readily passes through the placenta to the fetus and fetal brain (EPA, 2001a).

Based on the findings of the National Research Council, EPA has concluded that benefits of Hg reductions would be most apparent at the human consumption stage, as consumption of fish is the major source of exposure to methylmercury. At lower levels, documented Hg exposure effects may include more subtle, yet potentially important, neurodevelopmental effects. Figure 5-1 shows how emissions of mercury can transport from the air to water and impact human health and ecosystems.

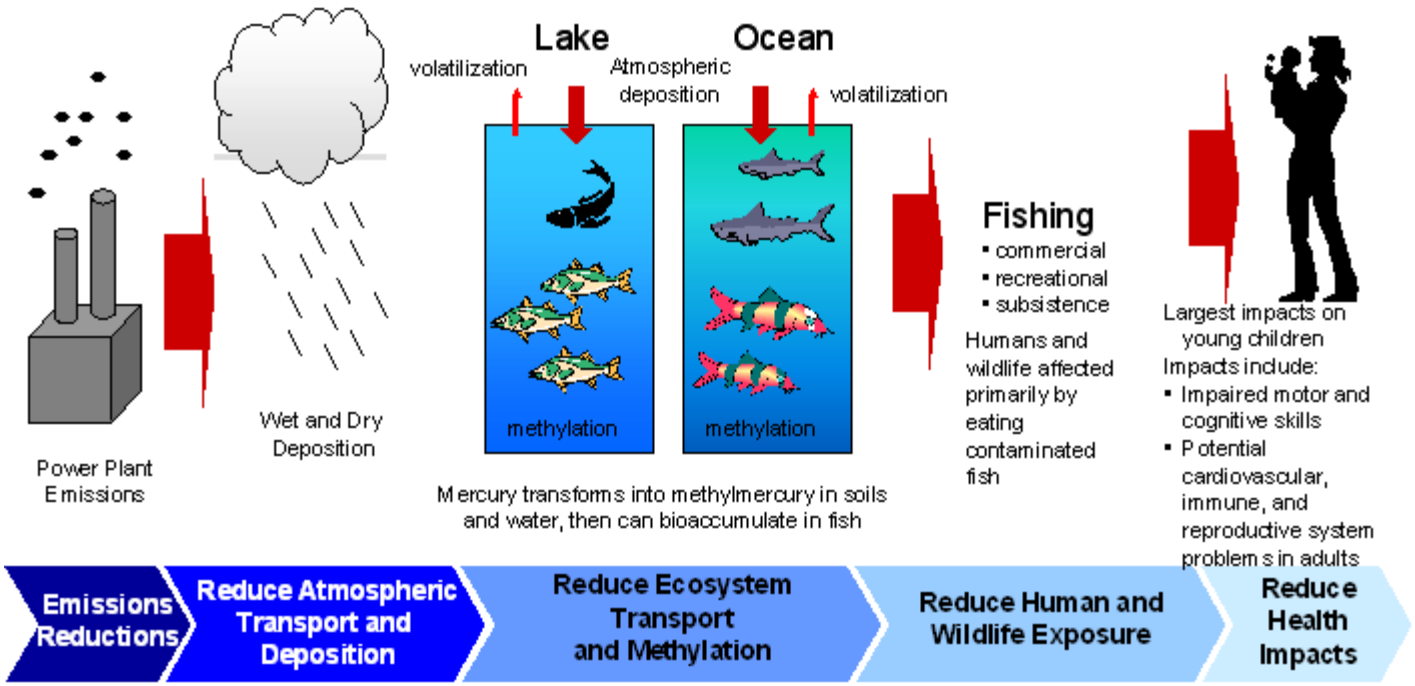


Figure 5-1. How Emissions of Mercury Can Impact Human Health and Ecosystems[30]

[30] Cardiovascular, immune, and reproductive system problems in adults are potential effects as the literature is either contradictory or incomplete.

Some subpopulations in the U.S., such as: Native Americans, Southeast Asian Americans, and lower income subsistence fishers, may rely on fish as a primary source of nutrition and/or for cultural practices. Therefore, they consume larger amounts of fish than the general population and may be at a greater risk to the adverse health effects from Hg due to increased exposure. In pregnant women, methylmercury can be passed on to the developing fetus, and at sufficient exposure may lead to a number of neurological disorders in children. Thus, children who are exposed to low concentrations of methylmercury prenatally may be at increased risk of poor performance on neurobehavioral tests, such as those measuring attention, fine motor function, language skills, visual-spatial abilities (like drawing), and verbal memory. The effects from prenatal exposure can occur even at doses that do not result in effects in the mother. Mercury may also affect young children who consume fish contaminated with Hg. Consumption by children may lead to neurological disorders and developmental problems, which may lead to later economic consequences.

Monitoring the concentrations of mercury in the blood of women of child-bearing age can help identify the proportion of children who may be at risk. EPA's reference dose (RfD) for methylmercury is 0.1 micrograms per kilogram body weight per day, which is approximately equivalent to a concentration of 5.8 parts per billion mercury in blood. Although the prenatal period is the most sensitive period of exposure, exposure to mercury during childhood also could pose a potential health risk (NAS, 2000).

Figure 5-2 shows reported concentrations of mercury in blood of women of childbearing age from the National Health and Nutrition Examination Survey (NHANES) (EPA, 2003b). The data presented are for total mercury, which includes methylmercury and other forms of mercury. Total blood mercury is a reasonable indicator of methylmercury exposure in people who consume fish and have no significant exposure to inorganic or elemental mercury (JAMA, April 2003). Thus the measured concentrations are a good indication of methylmercury concentrations. From this survey, about 8 percent of women of child-bearing age had at least 5.8 parts per billion of mercury in their blood in 1999-2000.

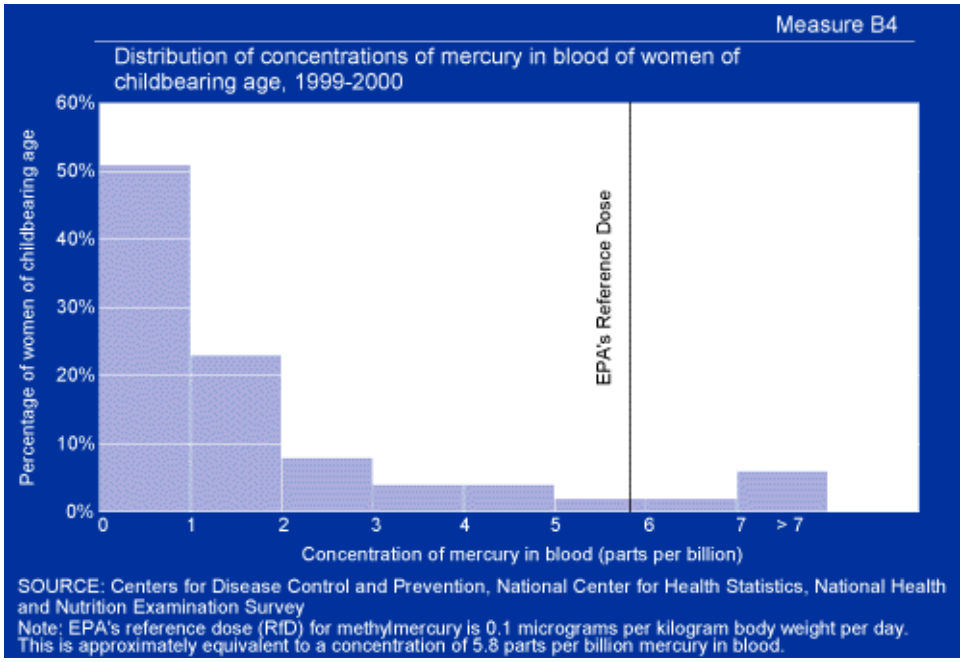


Figure 5-2. Concentrations of Mercury in Blood of Women of Childbearing Age

Figure 5-3 shows relative values of the BMD, BMDL and the RfD. The data show a Benchmark Dose (BMD) BMD at 85 ppb. The BMD is the dose or concentration that produced a doubling of the number of children with a response at the 5th percentile of the population. In this case, the changes evaluated were changes on neuropsychological testing batteries (i.e. the Boston Naming Test). In determining the RfD, EPA started with the BMD (85 ppb) and then used the 95% lower confidence limit to arrive at the 58 ppb BMDL. EPA then applied a composite uncertainty factor of 10 to calculate a final RfD of 5.8 ppb. The uncertainty factor adjustment was used to account for pharmacokinetic and pharmacodynamic uncertainty and variability.

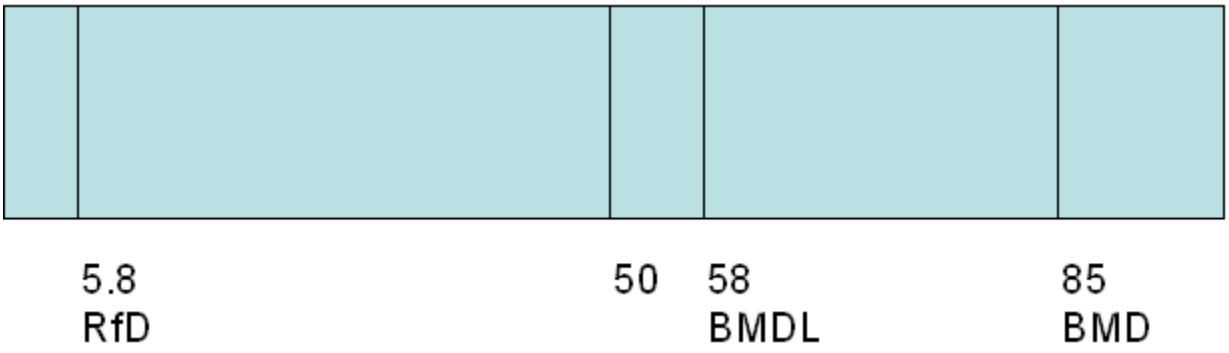


Figure 5-3. Relative Values of BMD, BMDL, and the RfD

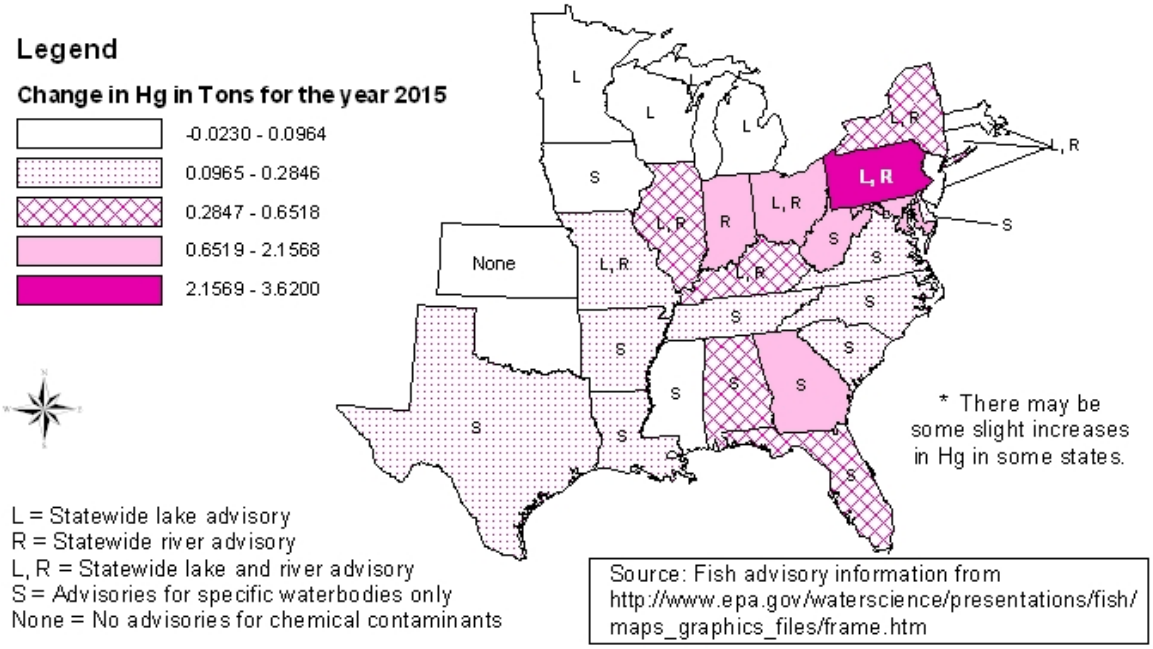
(Values in ppb)

In response to potential risks of mercury-contaminated fish consumption, EPA and FDA have issued fish consumption advisories which provide recommended limits on consumption of certain fish species for different populations. EPA and FDA are currently developing a joint advisory that has been released in draft form. This newest draft FDA-EPA fish advisory recommends that women and young children reduce the risks of Hg consumption in their diet by moderating their fish consumption, diversifying the types of fish they consume, and by checking any local advisories that may exist for local rivers and streams. This collaborative FDA-EPA effort will greatly assist in educating the most susceptible populations. Additionally, the reductions of Hg from this regulation may potentially lead to fewer fish consumption advisories (both from federal or state agencies), which will benefit the fishing community. As Figure 5-4 shows, currently 44 states have issued fish consumption advisories for non-commercial fish for some or all of their waters due to contamination of mercury. The scope of FCA issued by states varies considerably, with some warnings applying to all water bodies in a state and others applying only to

individual lakes and streams. Note that the absence of a state advisory does not necessarily indicate that there is no risk of exposure to unsafe levels of mercury in recreationally caught fish. Likewise, the presence of a state advisory does not indicate that there is a risk of exposure to unsafe levels of mercury in recreationally caught fish, unless people consume these fish at levels greater than those recommended by the fish advisory.

Reductions in methylmercury concentrations in fish should reduce exposure, subsequently reducing the risks of mercury-related health effects in the general population, to children, and to certain subpopulations. Fish consumption advisories (FCA) issued by the States may also help to reduce exposures to potential harmful levels of methylmercury in fish (although some studies have shown limited knowledge of and compliance with advisories by at risk populations (May and Burger, 1996; Burger, 2000)). To the extent that reductions in mercury emissions reduces the probability that a water body will have a FCA issued, there are a number of benefits that will result from fewer advisories, including increased fish consumption, increased fishing choices for recreational fishers, increased producer and consumer surplus for the commercial fish market, and increased welfare for subsistence fishing populations.

Figure 5-4. Mercury Reductions By State In 2015



There is a great deal of variability among individuals in fish consumption rates; however, critical elements in estimating methylmercury exposure and risk from fish consumption include the species of fish consumed, the concentrations of methylmercury in the fish, the quantity of fish consumed, and how frequently the fish is consumed. The typical U.S. consumer eating a wide variety of fish from restaurants and grocery stores is not in danger of consuming harmful levels of methylmercury from fish and is not advised to limit fish consumption. Those who regularly and frequently consume large amounts of fish, either marine or freshwater, are more exposed. Because the developing fetus may be the most sensitive to the effects from methylmercury, women of child-bearing age are regarded as the population of greatest interest. The EPA, Food and Drug Administration, and many States have issued fish consumption advisories to inform this population of protective consumption levels.

The EPA's 1997 Mercury Study RTC supports a plausible link between anthropogenic releases of Hg from industrial and combustion sources in the U.S. and methylmercury in fish. However, these fish methylmercury concentrations also result from existing background concentrations of Hg (which may consist of Hg from natural sources, as well as Hg which has been re-emitted from the oceans or soils) and deposition from the

global reservoir (which includes Hg emitted by other countries). Given the current scientific understanding of the environmental fate and transport of this element, it is not possible to quantify how much of the methylmercury in locally-caught fish consumed by the U.S. population is contributed by U.S. emissions relative to other sources of Hg (such as natural sources and re-emissions from the global pool). As a result, the relationship between Hg emission reductions from Utility Units and methylmercury concentrations in fish cannot be calculated in a quantitative manner with confidence. In addition, there is uncertainty regarding over what time period these changes would occur. This is an area of ongoing study.

Given the present understanding of the Hg cycle, the flux of Hg from the atmosphere to land or water at one location is comprised of contributions from: the natural global cycle; the cycle perturbed by human activities; regional sources; and local sources. Recent advances allow for a general understanding of the global Hg cycle and the impact of the anthropogenic sources. It is more difficult to make accurate generalizations of the fluxes on a regional or local scale due to the site-specific nature of emission and deposition processes. Similarly, it is difficult to quantify how the water deposition of Hg leads to an increase in fish tissue levels. This will vary

based on the specific characteristics of the individual lake, stream, or ocean.

SECTION 6

COMPARISON OF BENEFITS AND COSTS

The estimated social costs to implement the proposed IAQR, as described in the cost analysis document, are approximately $2.9 billion annually and $3.7 billion annually for 2010 and 2015, respectively (1999$). Thus, the net benefits (social benefits minus social costs) of the program in 2010 are approximately $55 + B billion annually in 2010 and $80 + B billion annually in 2015 (1999$). (B represents the sum of all unquantified benefits and disbenefits.) Therefore, implementation of the proposed rule is expected, based purely on economic efficiency criteria, to provide society with a significant net gain in social welfare, even given the limited set of health and environmental effects we were able to quantify. Addition of ozone-, directly emitted $PM_{2.5}$-, mercury-, acidification-, and eutrophication-related impacts would increase the net benefits of the proposed rule. As discussed in section IX of the notice for this rulemaking, we did not complete air quality modeling that precisely matches the IAQR region. We anticipate that any differences in estimates presented due to the modeling region analyzed will be small. Table 6-1 presents a summary of the benefits, costs, and net benefits of the proposed rule.

Table 6-1. Summary of Annual Benefits, Costs, and Net Benefits of the Inter-State Air Quality Rule

Description	2010 (billions of 1999 dollars)	2015 (billions of 1999 dollars)
Social costs[a]	$2.9	$3.7
Social benefits[b,c]		
Ozone-related benefits	$0.1	$0.1
PM-related health benefits	$56.8 + B	$82.3 + B
Visibility benefits	$0.9	$1.4
Net benefits (benefits-costs)[b,c,d]	$55 + B	$80 + B

[a] Note that costs are the annual total costs of reducing pollutants including NO_x and SO_2.

[b] As the table indicates, total benefits are driven primarily by PM-related health benefits. The reduction in premature fatalities each year accounts for over 90 percent of total benefits. Benefits in this table are associated with NO_x and SO_2 reductions.

[c] Not all possible benefits or disbenefits are quantified and monetized in this analysis. B is the sum of all unquantified benefits and disbenefits. Potential benefit categories that have not been quantified and monetized are listed in Table 1-4.

[d] Net benefits are rounded to the nearest billion. Columnar totals may not sum due to rounding.

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