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May 9, 2014

VIA E-FILING
Dr. Burl W. Haar Executive Secretary
Minnesota Public Utilities Commission
121 7th Place East, Suite 350
St. Paul, MN 55101-2147

Re: In the Matter of the Investigation into
Environmental and Socioeconomic Costs
under Minn. Stat. § 216B.2422, Subd.3
Docket No. E999/CI-00-1636
Minnesota Power's Written Comments

Dear Dr. Haar:

Minnesota Power hereby electronically submits the attached Written Comments in response to the Minnesota Department of Commerce – Division of Energy Resources' and Minnesota Pollution Control Agency's Notice dated April 1, 2014, in the above-referenced Docket. The written comments supplement and reinforce oral comments provided by Minnesota Power staff at the April 24, 2014, stakeholder group public meeting.

Please contact me at (218) 355-3601 with any questions related to this matter.

Yours truly,

Lori Hoyum
Policy Manager

c: Service List

STATE OF MINNESOTA
BEFORE THE
MINNESOTA PUBLIC UTILITIES COMMISSION

In the Matter of the Investigation into Environmental and Socioeconomic Costs under Minn. Stat. § 216B.2422, Subd.3	Docket No. E999/CI-00-1636 **MINNESOTA POWER'S WRITTEN COMMENTS**

I. Overview

Minnesota Power (or "Company") files these Written Comments in response to the Minnesota Department of Commerce – Division of Energy Resources' ("Department") and Minnesota Pollution Control Agency's ("MPCA") Notice dated April 1, 2014, in the above-referenced Docket. The Notice stated that the Department and MPCA are hosting a public stakeholder meeting on April 24, 2014, at the Public Utilities Commission Large Hearing Room to "address the scope of the investigation, whether to retain an expert under Minn. Stat. § 216B.62, Subd.8, and the possible role of an expert should one be retained." In a subsequent Discussion Document issued on April 17, 2014, by the Department and MPCA for the April 24, 2014, public stakeholder meeting, stakeholders were notified that in addition to the oral comments provided at the meeting, "written comments are welcome and must be provided by May 9, 2014, to ensure consideration by the Agencies." The Department and MPCA are required to provide a recommendation to the Minnesota Public Utilities Commission regarding the scope of the investigation by June 10, 2014.

Representatives from Minnesota Power attended the April 24th public stakeholder meeting and provided oral comments. These Written Comments supplement and reinforce the oral comments made by Minnesota Power during the meeting. Minnesota Power appreciates the opportunity to provide input into the scope of the investigation.

II. Minnesota Power's Comments

Minnesota Power provides Comments for the following areas of inquiry identified in the April 17, 2014, Discussion Document: 1) proposed scenarios and criteria used in their evaluation; 2) should additional greenhouse gases besides carbon dioxide ("CO_2") be included in the investigation; and 3) whether an outside expert should be retained to do this work and, if so, what is their role, and critical competencies needed to evaluate the scenarios.

A. Proposed Scenarios and Evaluation Criteria

During the April 24th public stakeholder meeting attendees' comments covered a wide array of opinions regarding criteria for consideration and potential process scenarios. Some stakeholders suggested that the questions being raised might more appropriately be resolved in a contested case hearing setting before an Administrative Law Judge. Minnesota Power agrees and, consequently, the Company has mostly refrained from commenting on these points.

Three criteria integral to determining environmental externality valuation were cited and discussed at the meeting. These criteria included:

1. Minnesota Region. The MPCA and the Minnesota Center for Environmental Advocacy ("MCEA") asserted that the determination of Minnesota environmental externality valuation would consider prospective global environmental damages rather than attempting to limit the relationship between Minnesota emissions to Minnesota regional environmental damages.
2. Air quality. The MPCA asserted that Minnesota or regional attainment with protective National Ambient Air Quality Standards ("NAAQS") established by the United States Environmental Protection Agency ("EPA") under the Clean Air Act does not matter when assessing environmental externality damage valuation.
3. Existence of or pending regulations that constrain emissions to meet national targets. The MPCA and MCEA asserted that the existence of regulations that constrain the emissions of environmental externality parameters doesn't matter when assessing environmental damage valuation.

Minnesota Power believes that calculating environmental externalities under the construct described above will result in externality valuations that are inaccurate and overstated. The Company believes that local and regional environmental impacts, local and regional air quality attainment, and existing and pending regulations are integral factors that must be considered and used in any methodology used for environmental externality valuation.

Similarly, if accuracy in environmental externality valuation is a goal, calculations done on a prospective basis should also adhere to the above framework.

$PM_{2.5}$ Zero Out Analysis

Minnesota Power notes that the "zero out" study[1] (see Attachment A) that had been suggested for Minnesota was performed by Abt Associates[2] about fourteen years ago, when emissions loading and concerns over air quality degradation from power plants was considerably higher. In October 2000, Abt Associates was retained to do air quality modeling for the Clean Air Task Force that included a "zero out" and 75 percent emission reduction scenario of all electric utility emissions nation-wide that contribute to fine particulate matter ("$PM_{2.5}$") formation, i.e. sulfur dioxide ("SO_2") and oxides of nitrogen ("NO_x").[3] The study used EPA reviewed 1990 meteorological conditions and assigned 1997 emissions conditions. Power plant emissions in 2014, specifically SO_2 and NO_x which are precursors to fine particulate matter formation, have been reduced significantly since 1997 and will be reduced even further once EPA's Mercury and Air Toxics Standards are fully implemented. Overall electric utility industry emissions of SO_2 and NO_x are down industry-wide 79 percent and 76 percent, respectively, since 1990.[4] The significant emission reductions achieved since the Abt study suggests that that the study's estimated health benefits should have been updated to reflect these changes. The environmental externalities valuation investigation should take into account the improved $PM_{2.5}$, SO_2 and

[1] "The Particulate-related Health Benefits of Reducing Power Plant Emissions", October 2000, Project Manager, Clean Air Task Force Conrad Schneider, Abt Associates et. al.
[2] Abt Associates is a mission-driven global leader in research and program implementation in the fields of health, social and environmental policy, and international development.
[3] Pages 3-3 and 3-4 of the Study illustrate the $PM_{2.5}$ concentration reductions resulting from 100 percent and 75 percent emission reductions.
[4] Source: U.S. Department of Energy, Energy Information Administration, U.S. Environmental Protection Agency, and U.S. Bureau of Economic Analysis. Edison Electric Institute, May 2013.

NO_x related air quality benefits resulting from large scale reductions in these power plant emissions. This point is emphasized through consideration of $PM_{2.5}$ NAAQS attainment in the region affected by Minnesota power plant emissions.

Similarly, the environmental externalities valuation investigation should take into account recent analysis that shows $PM_{2.5}$ also carries the influence of emissions from other sources such as carbonaceous particles and unburned carbon from motor vehicles. Carbonaceous particles have been identified as significant health benefit contributors and have often been present concurrently with other particulates such as compounds formed from SO_2 and NO_x.

$PM_{2.5}$ NAAQS

EPA has used a 95 percent confidence interval when establishing the $PM_{2.5}$ threshold for ambient air quality standards. The purpose of the confidence interval is to assure that there is 95 percent certainty that no adverse health impact is occurring due to the exposure to the pollutant in question.

Under the requirements of the Clean Air Act, EPA must ensure that the air quality standard thresholds will "protect the public health" with an "adequate margin of safety." While the current primary $PM_{2.5}$ level is 12 ug/m3, Minnesota Power notes that the study performed in the 2002 time frame using a $PM_{2.5}$ level set at 15 ug/m3 and a 95 percent confidence level, crosses over the 0 percent premature death calculation.

The point of the above example is two-fold. First, EPA's application of NAAQS standards is rigorous and, as required by law, is protective of public health while providing an adequate margin of safety. Second, applying environmental externality factors to emissions in areas where the NAAQS is being met is not justified, would produce pointless results, and is not supported by the rigorous science used by EPA in setting the NAAQS. Figure 1 illustrates the 95 percent confidence interval that had been determined for $PM_{2.5}$ with a compendium of health impact studies referred to as the "Six Cities Study."

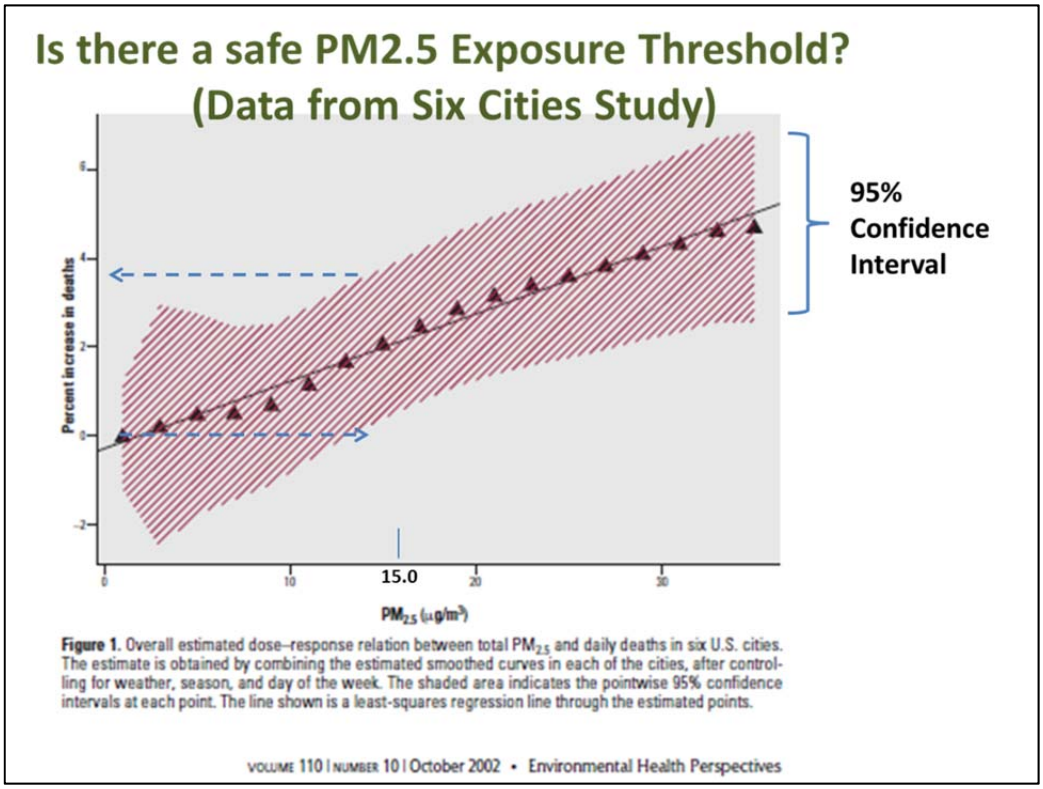

Figure 1 - 95% Confidence Interval for $PM_{2.5}$

Recent Regulatory Mechanisms

A recent development that may impact the scope of the Commission's investigation is that the United States Supreme Court overturned interpretations that the lower DC Circuit Court of Appeals applied to justify vacatur of the Cross State Air Pollution Rule ("CSAPR"). The DC Circuit Court and the EPA have been instructed to determine their basis for reinstatement of the CSAPR regulation of emissions associated with transport across state lines. Minnesota Power notes that since the CSAPR was structured by the EPA to identify significant contributors to downwind nonattainment areas and subject such sources in a State to emission budget constraints, there remains no reasonable basis by which an environmental externality should be assigned. Areas in attainment would appropriately not exhibit environmental externality damages. Nonattainment areas will have had measures as determined by the EPA, from both in-state and out-of-state sources, implemented through the State Implementation Plan process. Attached is a briefing of the CSAPR decision by Latham and Watkins (see Attachment B).

B. Inclusion of other Greenhouse Gases

Minnesota Power notes that over 99 percent of the greenhouse gas emissions associated with electric generation occur at the power plant and are emitted as CO_2. Minnesota should limit focus of electric utility environmental externality valuation investigation to the greenhouse gas emissions associated with electricity production. When consideration is given to measures such as regulation of utility CO_2 emissions under the Clean Air Act Section 111, and the EPA has set emissions guidelines appropriate for compliance at the regulated facility, i.e. "within the fence line", there should be flexibility for substituting other, more cost effective means for reducing CO_2 equivalent emissions, including reductions of other greenhouse gas emissions that occur outside of a regulated facility. Additionally, once the EPA acts to internalize CO_2 emission regulations for the electric utility industry by implementing their authority under the Clean Air Act, there remains no basis for assignment of a Minnesota CO_2 externality valuation.

C. Retaining an Expert – Competencies and Role if Retained

Minnesota Power recognizes that the Department, MPCA and Commission have many demands on their resources which may make it difficult to assign this work internally. Additionally, expertise of the current staff would need to be evaluated to determine if anyone internally has the required knowledge and skill to conduct the investigation. Minnesota Power appreciates the opportunity to provide input into the decision of whether an expert should be retained to perform this work, but respectfully declines to comment at this time. Minnesota Power will retain its own expert should the Company determine one is needed.

III. Conclusion

Minnesota Power supports the Commission's decision to reopen the investigation on externality values and appreciates the ability to submit written comments. In order to ensure the best outcome for all stakeholders, Minnesota Power respectfully requests that that environmental externality valuation investigation give consideration to the 1) area or conditions under which environmental emissions may be able to exert damages; 2) affected region's air quality standard attainment status; and 3) the existence of regulations designed to curtail emissions and related environmental damages for a pollutant that is also under consideration for assignment of environmental externality valuation. The Company thanks the Department and MPCA for their work on this docket and looks forward to working with the agencies and stakeholders in the future.

Dated May 9, 2014

Respectfully submitted,

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218-355-3601

The Particulate-Related Health Benefits of Reducing Power Plant Emissions

October 2000

Prepared for
Clean Air Task Force
Boston, MA

Project Manager:
Conrad Schneider

Prepared by
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with

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Abt Associates' Environmental Research Area provides multi-disciplinary scientific research and environmental policy analysis for the U.S. Environmental Protection Agency , the U.S. Agency for International Development, the Inter-American Development Bank, the World Bank, and directly to foreign, state and local governments. Abt Associates has extensive experience in estimating the potential public health improvements and economic costs and benefits from improving ambient air quality. The Environmental Research Area conducted extensive health analysis for the U.S. EPA in support of the 1997 revisions to both the ozone and the particulate matter National Ambient Air Quality Standards. They also prepared the health and economic analyses for EPA's 1997 Report to Congress *The Benefits and Costs of the Clean Air Act: 1970 to 1990*, and the 1999 Report *The Benefits and Costs of the Clean Air Act: 1990 to 2010*. Abt Associates conducts similar policy, health and economic analyses for EPA of regulations on the electric generating industry, automobile exhaust, diesel vehicles, regional haze, and potential policies for climate change mitigation strategies. Abt Associate's Environmental Research Area conducts public health analysis projects worldwide, including air pollution health assessment projects with the environmental and health ministries in Argentina, Brazil, Canada, Chile, Korea, Russia, Thailand, the Ukraine and for the World Health Organization.

Mr. Kenneth Davidson specializes in the analysis of air quality policy. He has a master's degree in resource economics and policy from Duke University's Nicholas School of the Environment, and worked with the Innovative Strategies and Economics Group at the U.S. EPA's Office of Air Quality Planning and Standards.

Dr. Leland Deck specializes in economic and risk analysis of environmental policies. His research projects include estimating the risks and economic value of health and welfare benefits from reducing air pollution, the costs of alternative pollution prevention technologies, and designing effective and enforceable economic incentive programs as a part of an overall strategy for controlling pollution from stationary and mobile sources. In addition to his own research projects, Dr. Deck manages Abt Associates' Environmental Economics Practice, and is a Vice President of Abt Associates.

Ms. Emily King graduated from Washington University in St. Louis with a B.A. in Environmental Science. Her undergraduate research focused on the analysis of satellite imagery to document changes in the Missouri River floodplain. She has extensive experience using GIS software to analyze environmental problems. Currently, she participates in the analysis of air quality policy and uses ArcView to map air quality results from various policy scenarios.

Mr. Mark Landry specializes in spatial and economic analysis of environmental policy. He graduated with a B.S. and M.S. in Natural Resource Management from Texas A&M University and is finishing a Master's degree in Applied Economics from Virginia Tech.

Dr. Don McCubbin has twelve years of experience analyzing air pollution and other environmental issues, covering air pollution, hazardous waste management, and growth and development. At Abt Associates, he conducts air quality, health and economic analyses of proposed air pollution regulations, and regulations on pesticides. Prior to joining Abt Associates, he conducted research on the social costs of air pollution, such as adverse health effects, crop losses, and decreased visibility. He also conducted research on the linkage between growth and development, and the management of small quantity generators of hazardous waste.

Dr. Ellen Post has fourteen years of experience in the scientific, economic, and policy analysis of environmental issues, with particular emphasis on (1) criteria air pollution risk assessment and economic benefit analysis, and (2) methods of assessing uncertainty surrounding individual estimates. She is one of the primary analysts conducting a particulate matter air pollution risk assessment for EPA's Office of Air Quality Planning and Standards, and has been a key economist in ongoing work analyzing the economic benefits associated with risk reductions from a number of air quality regulations, including the implementation of proposed particulate matter and ozone standards in the United States.

Systems Applications International, Inc. (SAI) is a wholly owned subsidiary of **ICF Consulting**. Throughout its nearly 30-year history, SAI has been a leader in the development of innovative air quality analysis and modeling techniques for primary and secondary pollutants. From the original development of the Urban Airshed Model (UAM) modeling system in the early 1970s, its update in 1992 resulting in the UAM-V version, to the recent development of the Regulatory Modeling System for Aerosols and Deposition (REMSAD – now at version 5.0), ICF/SAI has provided state-of-the-science tools with which to conduct a multitude of analyses related to air quality assessment and planning. ICF/SAI staff have extensive experience in meteorological and air quality data analysis (including the development of a novel and objective technique for modeling-related episode selection); emission inventory preparation and quality assurance; meteorological modeling (and, in particular, the use of dynamic meteorological models to prepare inputs for air quality modeling); development and application of photochemical and particulate matter (PM) models (for both regulatory and research purposes and both regional- and urban-scale analysis); evaluation of model performance; and preparation of EPA-approved technical support documents (that have been submitted by states as part of their attainment and maintenance plans). Air quality modeling systems developed by ICF/SAI are being applied around the world by a variety of business, public, and educational institutions. Modeling procedures and techniques originally developed by ICF/SAI scientists have become standard practice for the application of air quality modeling systems.

Dr. Mita Das specializes in the analysis of air quality data and modeling results. She has more than four years of experience in the application of the REMSAD model and the analysis of results. She is also experienced in the preparation of emissions (specifically biogenic emissions) for air quality modeling.

Ms. Sharon G. Douglas has more than 13 years of experience in meteorological and air quality data analysis and modeling. At ICF/SAI, she has been principally involved in the development and application urban- and regional-scale air quality models for regulatory assessment and planning purposes. Areas of specialization with respect to air quality modeling include meteorological input preparation, model performance evaluation, and interpretation of modeling results.

Dr. Kamala Jayaraman is a senior economist with over 14 years of experience, comprising economic and policy analyses of domestic and international environmental issues, electric sector modeling, econometric and statistical applications, teaching, and financial analysis and operation. Since joining ICF in 1995, Dr. Jayaraman has analyzed various issues related to two principal areas: Climate Change, and Electric Power Market Modeling. Dr. Jayaraman's other work experience includes analysis of issues related to international trade in hazardous wastes, Superfund, agricultural policy, education, and flood impact assessment. Dr. Jayaraman has a Ph.D. in Economics from University of Maryland, College Park, USA; and a M.A. in Economics from Bharathidasan University, and a B.A. in Economics from University of Madras, India.

Mr. Thomas Myers specializes in the development and application of air quality modeling systems. He has more than 20 years of experience in air quality modeling and is the principal developer of the UAM-V modeling system. He is currently directing a national-scale application of REMSAD for the analysis of mercury deposition.

Dr. Boddu N. Venkatesh applies systems and operations research tools to complex problems. Energy and environmental planning have been his area of focus. At ICF Consulting, Dr. Venkatesh has been primarily involved with supporting U.S. EPA with IPM™ based analytical work in regards to electric utility environmental compliance planning for NO_x, SO_2, Mercury, and Global Climate Change. In addition, he has managed the Environmental Assessment for the FERC Order 2000 and was the lead analyst involved in developing the ICF Consulting's Bulk Power Outlook 1999.

Ms. Yi-Hua Wei specializes in the preparation and quality assurance of detailed emission inventories for regional- and urban-scale air quality modeling. She has more than 15 year of experience in emission inventory preparation, Gaussian modeling, and meteorological, air quality, and emissions data analysis.

E.H. Pechan & Associates, Inc. is a technology-oriented consulting firm specializing in a full range of air pollution consulting services, including economic, energy, risk/benefit, and financial analyses. The firm has a staff of over 40 professionals, including environmental scientists, chemical engineers, air quality specialists, transportation and policy analysts, economists, operations and communications specialists, and support staff. Managers at Pechan have extensive experience in many technical areas and have developed successful working relationships with government, industry, and business. Pechan's analytical and policy-oriented services are backed by proven project management experience and a national reputation for state-of-the-art computer analysis. The firm has designed, developed, and applied analysis techniques to provide government and private industry with customized tools to gain valuable insight into a wide range of air and water quality issues. Pechan applies its capabilities to a variety of economic activities, ranging from resource extraction and transportation to manufacturing and consumption. The firm is recognized for its in-depth knowledge of Federal and State air and water programs and for its experience in developing and improving: emission inventories, complex economic and policy models, air toxic programs, databases,
pollution control technology assessments, and environmental and human health benefits analysis.

Mr. Michael Cohen is an environmental engineer in Pechan's Virginia office. Most of his work in the past year has been with utility data bases; this includes comparison and aggregation of data, development of user-friendly interfaces for utility data, and web-based utility data reports. He also has been active in ozone nonattainment projects relating to emission inventories and control technology assessment. Other present work in the utility area relates to developing web pages for both Emissions Tracking System/Continuous Emissions Monitoring NOx-related data and for steam utility data at the plant, boiler, and fuel levels.

Dr. Frank Divita is a Program Manager and Senior Scientist at Pechan's Springfield, Virginia office and has 10 years of experience in performing and managing technical studies of air pollution issues. His experience relates to the collection, control, chemical analysis, transport, and source apportionment of atmospheric pollutants from point and area sources. He also has experience in receptor and dispersion modeling, statistical data analysis, and interpretation of ambient and meteorological data. Most of his research in the past 4 years has been in ozone and PM nonattainment issues, including regulatory and planning analyses, emission inventory development, and control strategy analysis.

Ms. Patricia Horch is a chemical engineer at Pechan's Springfield, Virginia office. Her experience includes using Pechan's S-R matrix model to predict the air quality changes associated with alternative pollution control scenarios. In addition, Ms. Horch has extensive experience performing complex analyses on large computer databases and developing technical Internet sites.

Dr. Susy Rothschild has spent more than 17 years at Pechan designing, developing, maintaining, and conducting extensive quality assurance and quality control (QA/QC) reviews of utility data bases - merging, updating, analyzing, and writing technical support documents for large-scale national air quality and emissions data bases. She is the principal developer of EPA's electric utility data bases and technical support documents, including the Emission & Generation Resource Integrated Database (E-GRID), the Acid Rain Data Bases, the three National Allowance Data Bases, and the fossil-fuel steam utility components of the National Emissions Trends (NET) data bases. Dr. Rothschild's experience also includes a long history of involvement in air pollution-related health studies.

TABLE OF CONTENTS

List of Exhibits

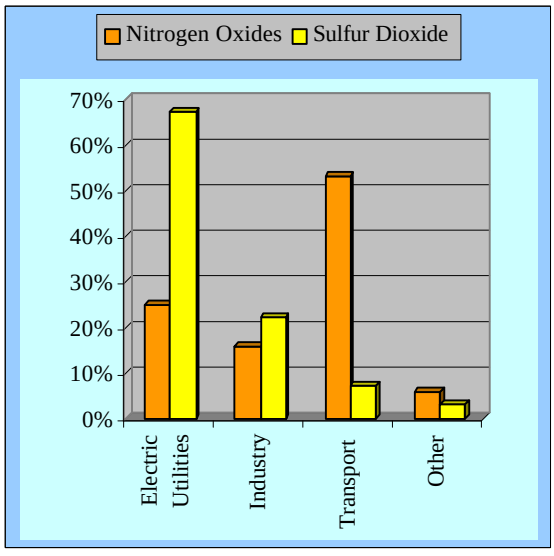
1. INTRODUCTION

This report estimates the adverse human health effects due to exposure to particulate matter from power plants. Power plants are significant emitters of sulfur dioxide (SO_2) and nitrogen oxides (NO_x). In many parts of the country, especially the Midwest, power plants are the largest contributors. These gases are harmful themselves, and they contribute to the formation of acid rain and particulate matter. Particulate matter reduces visibility, often producing a milky haze that blankets wide regions, and it is a serious public health problem. Over the past decade and more, numerous studies have linked particulate matter to a wide range of adverse health effects in people of all ages.

Epidemiologists have consistently linked particulate matter with effects ranging from premature death, hospital admissions and asthma attacks to chronic bronchitis. This study documents the health impacts from power plant air pollution emissions. Using the best available emissions and air quality modeling programs, we forecast ambient air quality for a business-as-usual "baseline" scenario for 2007, assuming full implementation of the Acid Rain program and the U.S. Environmental Protection Agency's Summer Smog rule (the 1999 NO_x SIP Call). We then estimate the attributable health impacts from all power plant emissions (the "All Power Plant Scenario"). Finally, we estimate air quality for a specific policy alternative: reducing total power plant emissions of SO_2 and NO_x 75 percent from the levels emitted in 1997. The difference between this "75 Percent Reduction Scenario" and the baseline provides an estimate of the health effects that would be avoided by this reduction in power plant emissions.

In addition to this policy scenario, we perform sensitivity analyses to examine alternative emission reductions and forecast ambient air quality using a second air quality model. The U.S. Environmental Protection Agency (EPA) uses both air quality models extensively, and both suggest that power plants make a large contribution to ambient particulate matter levels in the Eastern U.S. To put the power plant results in context, we also examine air pollution from all on-road and off-road diesel engine emissions. The results suggest that both power plants and diesel engines make a large contribution to ambient particulate matter levels and the associated health effects.

Exhibit 1-1 National Emissions 1997



Chapter 2 describes the development of the emissions inventory. Chapter 3 describes the methods we used to estimate changes in particulate matter concentrations. Chapter 4 describes general issues arising in estimating and valuing changes in adverse health effects associated with changes in particulate matter. Chapter 5 describes in some detail the methods used for estimating and valuing adverse health effects, and in Chapter 6 we present the results of these analyses.

This study has six appendices. Appendix A provides results of this analysis for all metropolitan areas

in the U.S. and a list of the counties in each metropolitan area. Appendices B, C and D present a detailed examination of how we derived our pollution emission estimates and translated emissions into forecasts of ambient particulate matter levels. Appendix E presents the results of an alternative air quality model. Appendix F presents a derivation of the particulate matter concentration-response functions used in all the analyses.

2. EMISSIONS INVENTORY

This chapter documents the development of the emission inventories and modeling input files used in this analysis. E. H. Pechan and Associates developed the emissions inventories for the business-as-usual (baseline) scenario and for three scenarios: a "75 Percent Reduction" scenario, an "All Power Plant" scenario, and a "Diesel Vehicle scenario".

To estimate emissions for each scenario, Pechan (2000) summed the emissions of five major emission sectors: power plant, non-power plant point, stationary area, non-road, and on-road mobile source sectors. To estimate power plant emissions, Pechan used the results of the Integrated Planning Model™ (IPM™), which we discuss in detail in Appendix B. Except for the power plants, Pechan previously developed the emissions inventory used in this analysis for EPA in support of EPA's Tier 2 rulemaking analysis (Pechan 1999). These non-power plant emission inventories contain 2007 emission estimates for on-road mobile, non-power plant point, stationary area, and non-road sources. In general, Pechan (1999) developed the non-power plant emission inventories by projecting 1996 National Emission Trends (NET) emission estimates to 2007.

In order to quantify the total contribution from all power plants and all diesel engines, we eliminate in turn the emissions from these two emission source categories and calculate the resulting air quality. This identifies the total air quality "footprint" of power plants and diesels on fine particulate matter concentrations.

Appendix C provides further detail on Pechan's emission inventory work.

2.1 POWER PLANT EMISSIONS

ICF Consulting (2000) used the IPM™ to forecast SO_2 and NO_x emissions at power plants. For the baseline, ICF assumed a continuation of current EPA policies until the year 2007: full implementation of the NO_x State Implementation Plan (SIP) Call by 2003, full implementation of Phase II of Title IV of the Clean Air Act (CAA) Amendments of 1990, and no explicit adoption of a global warming climate treaty. Using these results and data on plant and fuel types, Pechan (2000) complemented the estimates of SO_2 and NO_x by estimating emissions of carbon monoxide (CO), volatile organic carbon (VOC), ammonia (NH_3), secondary organic aerosols (SOA) and direct particulates for 2007 baseline and control scenario inventories. We discuss this further below and in Appendix A.

2.1.1 Integrated Planning Model™

IPM™ is an industry-leading energy modeling system that simulates the deregulated wholesale market for electricity. The EPA has used IPM™ a number of times to evaluate the economic, operational and emission impacts of policies and rulemakings affecting the power sector.[1] The Federal Energy Regulatory Commission (FERC) has also used the model to assess the potential emission impact of open access

[1]Recent analyses performed for EPA using the IPM™ model include: (i) EPA (1998b); (ii) EPA (1998a); and (iii) supporting analyses for EPA's Section 126 Ozone Transport Rulemaking, December 1999.

transmission policies and to develop an Environmental Assessment of the Regional Transmission Organization (RTO) Proposed Rulemaking.

IPM™ is a multi-region linear programming model that determines the least-cost capacity expansion and dispatch strategy for operating the power system over specified future periods, under specified operational, market, and regulatory constraints. Constraints include emissions caps, transmission constraints, regional reserve margins, and meeting regional electric demand. Given a specified set of parameters and constraints, IPM™ develops an optimal capacity expansion plan, dispatch order, and air emissions compliance plan for the power generation system based on factors such as fuel prices, capital costs and operation and maintenance (O&M) costs of power generation, etc. EPA (1998b) provides additional details about the IPM™ model.

The model is dynamic: it makes decisions based on expectations of future conditions, such as fuel prices, and technology costs. Decisions are made on the basis of minimizing the net present value of capital plus operating costs over the full planning horizon. The model draws on a database containing information on the characteristics of each power plant (such as unit ID, unit type, unit location, fuel used, heat rate, emission rate, existing emission control technology, etc.) in the U.S.

2.2 NON-POWER PLANT EMISSIONS

Pechan (2000) extrapolated the 2007 non-power plant point source inventory from the 1996 national emission inventory using Bureau of Economic Analysis (BEA) Gross State Product (GSP) growth factors at the State level by 2-digit Standard Industrial Classification (SIC) Code. power plant

The emissions inventory for point sources other than power plants incorporated control measures reflecting CAA requirements in addition to the NO_x SIP Call control requirements (22 States plus the District of Columbia). The NO_x SIP Call controls applied annual NO_x emission reductions for point sources for controls expected to operate for 12 months/year. Five month reductions were applied to source types with controls expected to operate only during the ozone season. This was necessary to estimate accurate annual emissions since controls such as low NO_x burners cannot be turned off in the winter.

3. AIR QUALITY MODELING

The analysis used results from the Regulatory Modeling System for Aerosols and Acid Deposition (REMSAD) to forecast changes in the ambient concentration of both PM_{10} and $PM_{2.5}$ at the REMSAD grid cell level. Because it accounts for spatial and temporal variations as well as differences in the reactivity of emissions, REMSAD is ideal for evaluating the air-quality effects of emission control scenarios.

To provide additional scenarios and a point of comparison with previous analyses (e.g., EPA 1998a), we also used the Source Receptor (S-R) matrix to forecast PM formation. The S-R matrix is based on the Climatological Regional Dispersion Model (CRDM), and uses a less sophisticated approach than the resource-intensive three-dimensional REMSAD approach. The S-R Matrix consists of fixed coefficients that reflect the relationship between annual average PM concentration values at a single receptor in the center of each county and the contribution by PM species to this concentration from each emission source in all counties in the 48 contiguous states (Pechan 2000).

Modeling future air quality anticipated to result from policy-driven emissions changes is extremely difficult and inherently uncertain. Alternative air quality models inevitably produce differing results. Scientific understanding of the complex atmospheric processes involved with PM formation and transport is increasing rapidly. The new $PM_{2.5}$ monitoring data now being collected nationwide, and improvements in the estimates of emissions from all sources, will help calibrate and verify the performance of air quality models. Existing air quality models are being improved constantly, and the next generation of PM air quality models are under development. By including health effects estimates based on two different air quality models used by EPA, this analysis can present both a better picture of the potential range of estimates and information about the sensitivity of the health effect estimates to the selection of air quality models. As will be seen below, REMSAD estimates a larger change in $PM_{2.5}$ levels in much of the country than does the S-R matrix approach, resulting in larger estimates of avoidable health effects.

3.1 PARTICULATE MATTER FORMATION

Ambient concentrations of PM are composed of directly emitted particles and of secondary aerosols of sulfate, nitrate, and organics. Particulate matter is the generic term for the mixture of microscopic solid particles and liquid droplets found in the air. The particles are either emitted directly from these combustion sources or are formed in the atmosphere through reactions involving gases, such as SO_2 and NO_x.

3.2 REMSAD AIR QUALITY MODEL

REMSAD was used to simulate estimates of particulate matter concentration for three future-year scenarios. ICF Consulting/Systems Applications International, Inc. (ICF/SAI) performed the REMSAD modeling. Subsequently we used the modeling results to estimate the health-related costs for each of the scenarios in the primary analysis.

The REMSAD model is designed to simulate the effects of changes in emissions on PM concentrations and deposition. REMSAD calculates concentrations of pollutants by simulating the physical and chemical processes in the atmosphere. The basis for REMSAD is the atmospheric diffusion or species continuity

equation. This equation represents a mass balance that includes all of the relevant emissions, transport, diffusion, chemical reactions, and removal processes in mathematical terms.

Because it accounts for spatial and temporal variations as well as differences in the reactivity of emissions, REMSAD can evaluate the air-quality effects of specific emission control scenarios. This is achieved by first replicating a historical ozone episode to establish a base-case simulation. ICF/SAI prepared model inputs from observed meteorological, emissions, and air quality data for selected episode days using various input preparation techniques. They apply the REMSAD model with these inputs, and the results are evaluated to determine model performance. Once the model results have been evaluated and determined to perform within prescribed levels, they combine the same base-case meteorological inputs with *modified* or *projected* emission inventories to simulate possible alternative/future emission scenarios.

The meteorological fields for this application of the REMSAD modeling system represent a base year of 1990. EPA (1999b) tested and evaluated these inputs, and thus no additional modeling of the 1990 base year was needed for this study. The modeling domain encompasses the contiguous 48 state, as well as portions of Canada and Mexico. ICF/SAI applied REMSAD using a horizontal grid resolution of approximately 56 km. The model was run for an entire year to enable the calculation of annual average values of particulate concentrations.

Exhibit 3-1 presents the power plant contribution to annual average $PM_{2.5}$ levels. We mapped this for each REMSAD grid-cell, but taking the difference of the annual average $PM_{2.5}$ levels in the baseline and the "All Power Plant" scenario. Exhibit 3-2 presents the power plant contribution that remains after implementing the "75 Percent Reduction" scenario. We estimated this by taking the difference of the annual average $PM_{2.5}$ levels in the 2007 baseline power plant scenario and the "75 Percent Reduction" scenario.

3.3 FORECASTING AIR QUALITY AT CAPMS GRID-CELLS

The Criteria Pollutant Air Modeling System (CAPMS), developed by Abt Associates, is a population-based computer program that models human exposure to changes in air pollution concentrations and estimates the associated health benefits. CAPMS divides the United States into eight kilometer by eight kilometer grid cells, and estimates the changes in incidence of adverse health effects associated with given changes in air quality in each CAPMS grid cell. We assigned each CAPMS grid cell to the nearest REMSAD grid cell, by calculating the shortest distance between the center of the CAPMS grid cell to the center of a REMSAD grid cell. Given the air quality change and the population, we estimated the change in adverse health effects in each CAPMS grid cell (described in Chapters 4 and 5 and in Appendix F). To get the national incidence change (or the changes within individual states or counties) we summed the CAPMS grid-cell-specific changes.

Exhibit 3-1 The Power Plant $PM_{2.5}$ "Footprint":
Change in Annual Mean $PM_{2.5}$ Levels From Eliminating All Power Plant Emissions

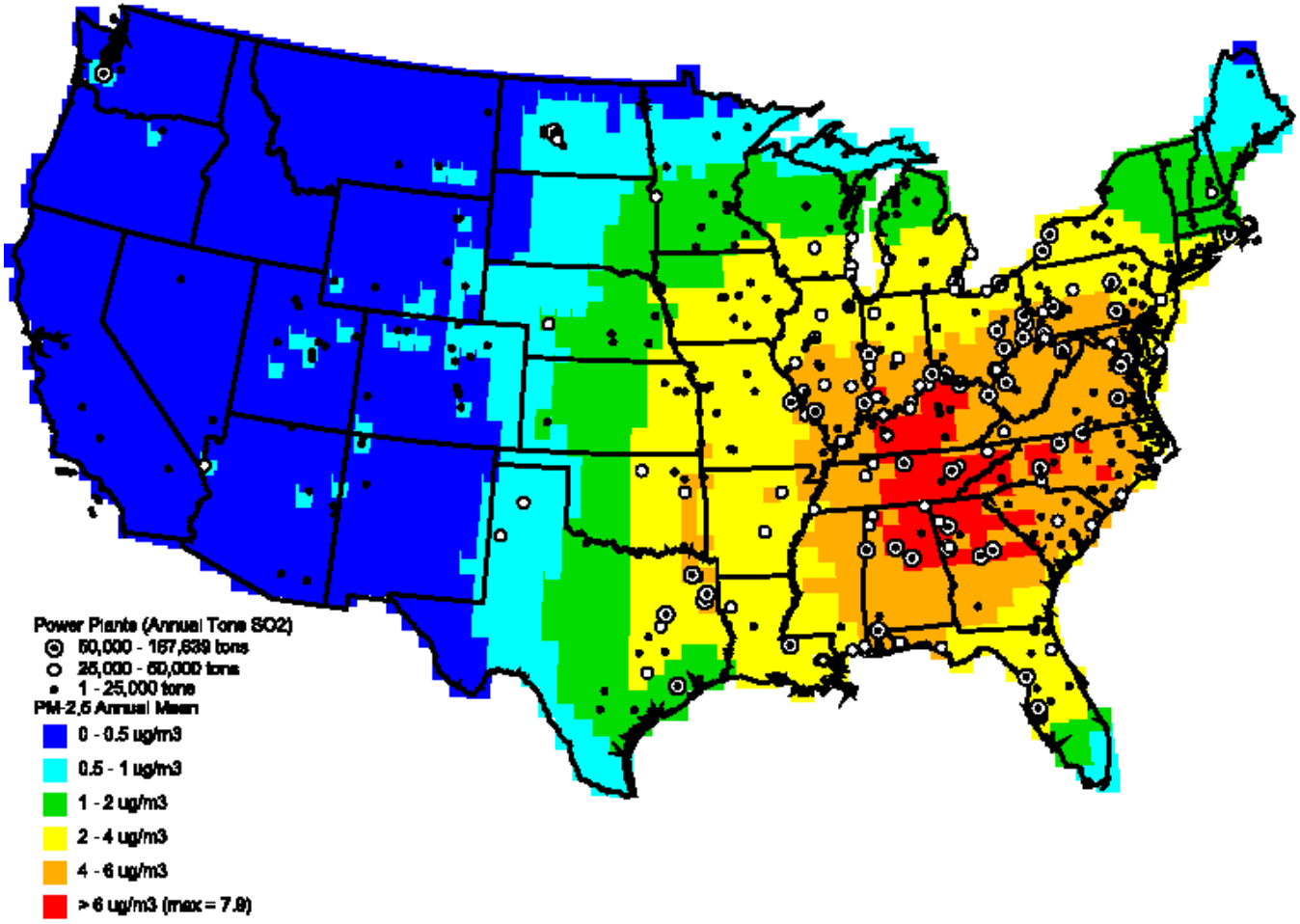
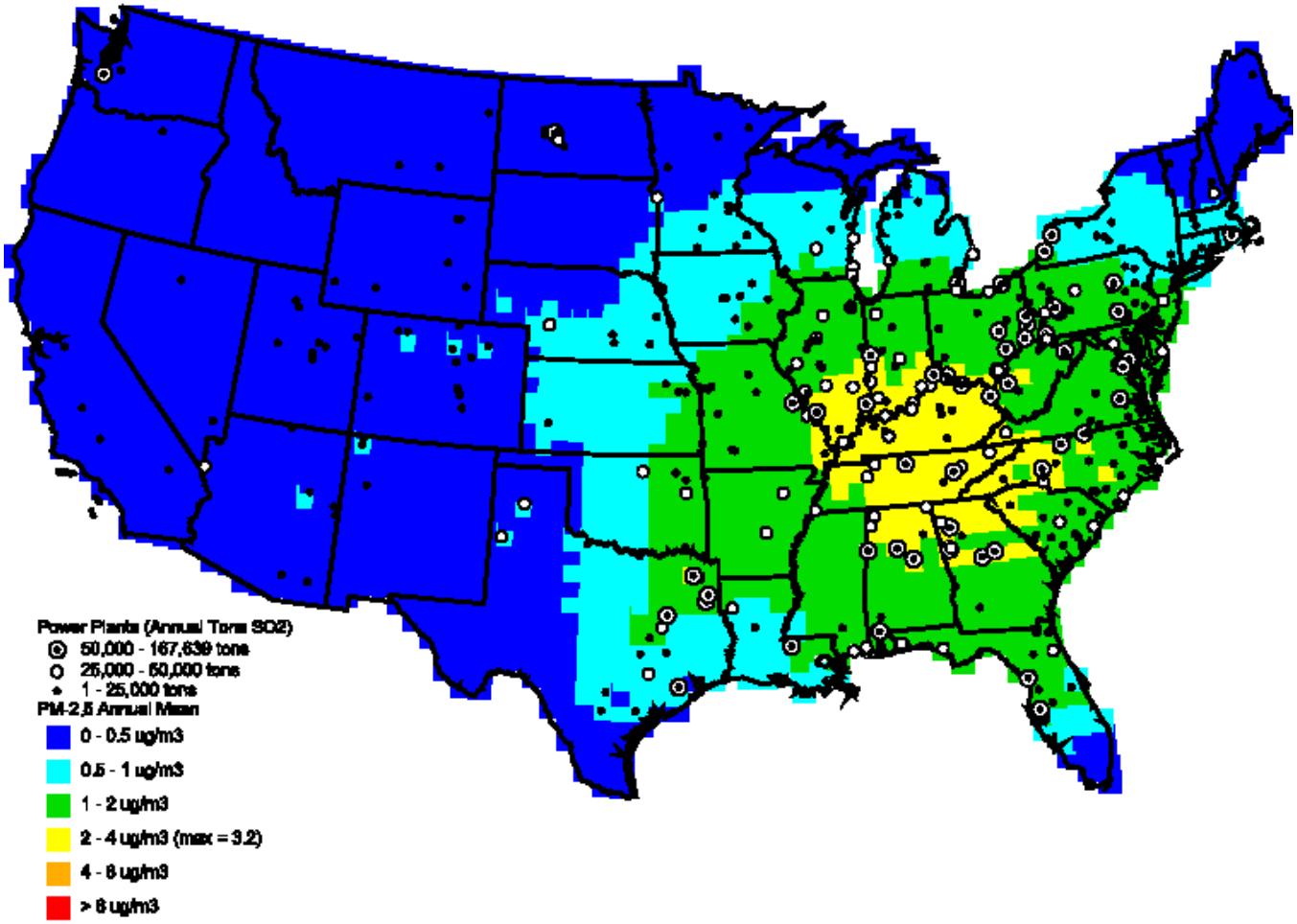


Exhibit 3-2 The Power Plant "Footprint" After 75 Percent Emission Reduction: Change in Annual Mean $PM_{2.5}$ From All Power Plants After 75 Percent Emission Reduction



4. ISSUES IN ESTIMATING HEALTH BENEFITS

Changes in PM levels result in changes in a number of health effects, or "endpoints," that society values. This chapter discusses key issues in the estimation of adverse health effects and in the valuation of health benefits. Section 1 describes general issues that particularly affect the estimation of changes in health effects. Section 2 describes general issues in valuing health changes. Finally, Section 3 discusses how uncertainty is characterized in this analysis.

4.1 ESTIMATING ADVERSE HEALTH EFFECTS

This section reviews issues that arise in the estimation of adverse health effects. It reviews the derivation of C-R functions, and it reviews how CAPMS combines air quality data and C-R functions. In addition, we discuss how we handle overlapping health effects, thresholds, estimating the baseline incidence rates for the C-R functions, and other issues.

4.1.1 Basic Concentration-Response Model

While several health endpoints have been associated with exposure to ambient PM, the discussion below refers only to a generic "health endpoint," denoted as y. The discussion refers to estimation of changes in the incidence of the health endpoint at a single location (the population cell, which is equivalent to the CAPMS gridcell). Region-wide changes are estimated by summing the estimated changes over all population cells in the region.

Different epidemiological studies may have estimated the relationship between PM and a particular health endpoint in different locations. The C-R functions estimated by these different studies may differ from each other in several ways. They may have different functional forms; they may have measured PM concentrations in different ways; they may have characterized the health endpoint, y, in slightly different ways; or they may have considered different types of populations. For example, some studies of the relationship between ambient PM concentrations and mortality have excluded accidental deaths from their mortality counts; others have included all deaths. One study may have measured daily (24-hour) average PM concentrations while another study may have used two-day averages. Some studies have assumed that the relationship between y and PM is best described by a linear form (i.e., the relationship between y and PM is estimated by a linear regression in which y is the dependent variable and PM is one of several independent variables). Other studies have assumed that the relationship is best described by a log-linear form (i.e., the relationship between the natural logarithm of y and PM is estimated by a linear regression).[2] Finally, one study may have considered changes in the health endpoint only among members of a particular subgroup of the population (e.g., individuals 65 and older), while other studies may have considered the entire population in the study location.

The estimated relationship between PM and a health endpoint in a study location is specific to the type of population studied, the measure of PM used, and the characterization of the health endpoint considered. For

[2]The log-linear form used in the epidemiological literature on PM-related health effects is often referred to as "Poisson regression" because the underlying dependent variable is a count (e.g., number of deaths), assumed to be Poisson distributed. The model may be estimated by regression techniques but is often estimated by maximum likelihood techniques. The form of the model, however, is still log-linear.

example, a study may have estimated the relationship between daily average PM concentrations and daily hospital admissions for "respiratory illness," among individuals age 65 and older, where "respiratory illness" includes International Classification of Disease (ICD) codes A, B, and C.[3] If any of the inputs had been different (for example, if the entire population had been considered, or if "respiratory illness" had consisted of a different set of ICD codes), the estimated C-R function would have been different. When using a C-R function estimated in an epidemiological study to estimate changes in the incidence of a health endpoint corresponding to a particular change in PM in a population cell, then, it is important that the inputs be appropriate for the C-R function being used -- i.e., that the measure of PM, the type of population, and the characterization of the health endpoint be the same as (or as close as possible to) those used in the study that estimated the C-R function.

Estimating the relationship between PM and a health endpoint, y, consists of (1) choosing a functional form of the relationship and (2) estimating the values of the parameters in the function assumed. The two most common functional forms in the epidemiological literature on PM and health effects are the log-linear and the linear relationship. The log-linear relationship is of the form:

$$y = Be^{b \cdot PM} \ ,$$

or, equivalently,

$$\ln(y) = a + b \cdot PM \ ,$$

where the parameter B is the incidence of y when the concentration of PM is zero, the parameter β is the coefficient of PM, ln(y) is the natural logarithm of y, and $\alpha = \ln(B)$.[4] If the functional form of the C-R relationship is log-linear, the relationship between ΔPM and Δy is:

$$\Delta y = y \cdot \left(e^{b \cdot \Delta PM} - 1\right) \ ,$$

where y is the baseline incidence of the health effect (i.e., the incidence before the change in PM). For a log-linear C-R function, the relative risk (RR) associated with the change ΔPM is:

$$RR_{\Delta PM} = e^{b \cdot \Delta PM} \ .$$

Epidemiological studies often report a relative risk for a given ΔPM, rather than the coefficient, β, in the C-R function. The coefficient can be derived from the reported relative risk and ΔPM, however, by solving for β:

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[3] The International Classification Codes are described at the website of the Medical Center Information Systems: Duke University Health Systems (1999).

[4] Other covariates besides pollution clearly affect mortality. The parameter B might be thought of as containing these other covariates, for example, evaluated at their means. That is, $B = B_o \exp\{\beta_1 x_1 + ... + \beta_n x_n\}$, where B_o is the incidence of y when all covariates in the model are zero, and $x_1, ... , x_n$ are the other covariates evaluated at their mean values. The parameter B drops out of the model, however, when changes in incidences are calculated, and is therefore not important.

$$b = \frac{\ln(RR)}{\Delta PM} \ .$$

The linear relationship is of the form:

$$y = a + b \cdot PM \ ,$$

where α incorporates all the other independent variables in the regression (evaluated at their mean values, for example) times their respective coefficients. When the C-R function is linear, the relationship between a relative risk and the coefficient, β, is not quite as straightforward as it is when the function is log-linear. Studies using linear functions usually report the coefficient directly.

If the functional form of the C-R relationship is linear, the relationship between ΔPM and Δy is simply:

$$\Delta y = b \cdot \Delta PM \ .$$

A few epidemiological studies, estimating the relationship between certain morbidity endpoints and PM, have used functional forms other than linear or log-linear forms. Of these, logistic regressions are the most common. Abt Associates (1999, Appendix A) provides further details on the derivation of dose-response functions.

4.1.2 Calculation of Adverse Health Effects with CAPMS

CAPMS is a population-based system for modeling exposure to ambient levels of criteria air pollutants and estimating the adverse health effects associated with this exposure. CAPMS divides the United States into multiple grid cells, and estimates the changes in incidence of adverse health effects associated with given changes in air quality in each grid cell. The national incidence change (or the changes within individual states or counties) is then calculated as the sum of grid-cell-specific changes.

To reflect the uncertainty surrounding predicted incidence changes resulting from the uncertainty surrounding the pollutant coefficients in the C-R functions used, CAPMS produces a *distribution* of possible incidence changes for each adverse health, rather than a single point estimate. To do this, it uses both the point estimate of the pollutant coefficient (β in the above equation) and the standard error of the estimate to produce a normal distribution with mean equal to the estimate of β and standard deviation equal to the standard error of the estimate. Using a Latin Hypercube method,[5] we take the n^{th} percentile value of β from this normal distribution, for n = 0.5, 1.5, ..., 99.5, and follow the procedure outlined in the section above to produce an estimate of the incidence change, given the β selected. Repeating the procedure for each value of β selected results in a distribution of incidence changes in the CAPMS grid cell. This distribution is stored, and CAPMS

[5]The Latin Hypercube method is used to enhance computer processing efficiency. It is a sampling method that divides a probability distribution into intervals of equal probability, with an assumption value for each interval assigned according to the interval's probability distribution. Compared with conventional Monte Carlo sampling, the Latin Hypercube approach is more precise over a fewer number of trials because the distribution is sampled in a more even, consistent manner (Decisioneering, 1996, pp. 104-105).

proceeds to the next grid cell, where the process is repeated. We calculate the distribution of the national change (or change in a designated geographical area) by summing the n^{th} percentile grid cell-specific changes, for n = 0.5, 1.5, ..., 99.5.

4.1.3 Overlapping Health Effects

Several endpoints reported in the health effects literature overlap with each other. For example, hospital admissions for single respiratory ailments (e.g. pneumonia) overlap with estimates of hospital admissions for "all respiratory" ailments.[6] Similarly, several studies quantify the occurrence of respiratory symptoms where the definitions of symptoms are not unique (e.g., shortness of breath or upper respiratory symptoms). In choosing studies to include in the aggregated benefits estimate (discussed below), this analysis carefully considers the issue of double-counting benefits that might arise from overlapping health effects.

4.1.4 Baseline Incidences

As noted above, most of the relevant C-R functions are log-linear, and the estimation of incidence changes based on a log-linear C-R function requires a baseline incidence. The baseline incidence for a given CAPMS population cell is the baseline incidence rate in that location multiplied by the relevant population. County mortality rates are used in the estimation of air pollution-related mortality, and all CAPMS population cells in the county are assumed to have the same mortality rate. Hospital admissions are only available at the national level, so all areas are assumed to have the same incidence rate for a given population age group. For some endpoints, such as respiratory symptoms and illnesses and restricted activity days, baseline incidence rates are not available even at the national level. The only sources of estimates of baseline incidence rates in such cases are the studies reporting the C-R functions for those health endpoints. The baseline incidence rate and its source are given for each C-R function in Appendix F.

4.1.5 Thresholds

A very important issue in applied modeling of changes in PM is whether to apply the C-R functions to all predicted changes in ambient concentrations, even small changes occurring at levels approaching the concentration in which they exist in the natural environment (without interference from humans), referred to as "anthropogenic background." Different assumptions about whether to model thresholds, and if so, at what levels, can have a major effect on the resulting benefits estimates.

None of the epidemiological functions relating PM to various health endpoints incorporate thresholds. Instead, all of these functions are continuous and differentiable down to zero pollutant levels. A threshold may be imposed on these models, however, in several ways, and there are various points at which the threshold could be set. (A threshold can be set at any point. There are some points, however, that may be considered more obvious candidates than others.) One possible threshold might be the background level of the pollutant. Another might be a relevant standard for the pollutant. Whatever the threshold, the implication is that there are no effects below the threshold.

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[6]Pneumonia is often classified with the International Classification of Diseases (ICD) codes of 480-486, while all respiratory admissions are classified with ICD codes 460-519.

A threshold model can be constructed in more than one way. One method is to simply truncate the C-R function at the threshold (i.e., to not include any physical effect changes associated with PM concentrations below the designated threshold). This method uses the original C-R function, but calculates the change in PM as [max(T,baseline PM) - max(T, regulatory alternative PM)], where T denotes the designated threshold. This threshold model will predict a smaller incidence of the health effect than the original model without a threshold. Clearly, as T increases, the predicted incidence of the health effect will decrease.

An alternative method is to replace the original C-R function with a "hockey stick" model that best approximates the original function that was estimated using actual data. The hockey stick model is horizontal up to a designated threshold PM level, T, and is linear with a positive slope for PM concentrations greater than T. Recall the log-linear C-R function:

$$y = a + b \cdot PM \ .$$

Assuming that the value of the coefficient, β, depends on the level of PM, we get:

$$\ln(y) = a' \ , \quad for\ PM \le T \ , and$$
$$\ln(y) = a' + b' \cdot PM \ \ , \quad for\ PM > T \ .$$

Ideally, the coefficients would be estimated based on the data in the original study – that is, a hockey stick model would be fit to the original data, so that the threshold model that is most consistent with the available information would be chosen. If a threshold model could be estimated from the original data, it is unlikely that α' would equal α or that β' would equal β, because such a hockey stick model would be consistently below the original model (equation (6)), except at PM=0 (where the two models would coincide). If that were the hockey stick model that best fit the data, then it is unlikely that the best fitting linear model would be consistently above it. Instead, the hockey stick model that best fits the same data would most likely have $\alpha' > \alpha$ and $\beta' > \beta$. A graph of this model would therefore cross the graph of the linear model at two points. Whether such a hockey stick threshold model predicted a greater or smaller incidence of the health effect than the linear model would depend on the distribution of PM levels. It is worth noting that the graph of the first type of threshold model, in which the C-R function is simply truncated at the threshold, would be discontinuous at the threshold. This is highly unlikely to be a good model of the actual relationship between PM and any health endpoint.

There is some evidence that, at least for particulate matter, not only is there no threshold, but the PM coefficient may actually be larger at lower levels of PM and smaller at higher levels. Examining the relationship between particulate matter (measured as TSP) and mortality in Milan, Italy during the ten year period 1980-1989, Rossi et al. (1999) fitted a model with one slope across the entire range of TSP and an additional slope for TSP greater than 200 $\mu g/m^3$. The second slope was statistically significant ($p<0.0001$) and negative, indicating a lower slope at higher TSP levels.

4.1.6 Application of a Single C-R Function Everywhere

Whether the C-R relationship between a pollutant and a given health endpoint is estimated by a single function from a single study or by a pooled function of C-R functions from several studies, that same C-R relationship is applied everywhere in the benefits analysis. Although the C-R relationship may in fact vary somewhat from one location to another (for example, due to differences in population susceptibilities or

differences in the composition of PM), location-specific C-R functions are available only for those locations in which studies were conducted. While a single function applied everywhere may result in overestimates of incidence changes in some locations and underestimates of incidence changes in other locations, these location-specific biases will to some extent cancel each other out when the total incidence change is calculated. It is not possible to know the extent or direction of the bias in the total incidence change based on application of a single C-R function everywhere.

4.1.7 Estimating Pollutant-Specific Benefits Using Single Pollutant vs. Multi-Pollutant Models

Many studies include multiple pollutants, like ozone and particulate matter, in their final models. For this analysis, however, we are estimating benefits for only particulate matter. This presents a challenge because it is often difficult to separate out the effect of a single pollutant from the effects of other pollutants in the mix. Multi-pollutant models have the advantage that the coefficient for a single pollutant in such a model will be unbiased (so that the effects of other pollutants will not be attributed falsely to the single pollutant). However, the variance of the estimator of the coefficient of the pollutant of interest will increase as the correlations between the other pollutants in the model and that pollutant increase. If the other pollutants in the model are highly correlated with the pollutant of interest, we would have an unbiased but unstable (high variance) estimator. However, while single pollutant models have the advantage of more stable estimators, the coefficient estimate in a single pollutant model could be biased in such a model. We could consider the single pollutant as an "indicator pollutant" – i.e., an indicator of a pollution mix – if we use single pollutant models. However, there is no guarantee that the composition of the pollution mix will remain the same under a control scenario that targets only a single pollutant.

This analysis uses both single pollutant and multi-pollutant models to derive PM-specific benefit estimates. When more than one study has estimated the relationship between a given endpoint and a given pollutant, information from both single-pollutant and multi-pollutant models may be pooled to derive pollutant-specific benefits estimates. For example, the benefits predicted by a model with only PM may be pooled with the benefits predicted by a model with both PM and ozone to derive an estimate of the PM-related benefits associated with a given endpoint.

Though this analysis estimates the benefits associated with reductions in PM alone, it is worth mentioning that there is the possibility of mis-characterizing benefits if some of the studies used are single pollutant models. Suppose, for example, that only ozone is actually associated with a given endpoint, but PM appears to be associated only because it is correlated with ozone. The benefits predicted by a single pollutant PM model would, in that case, actually reflect the benefits of reducing ozone, to the extent that PM and ozone are correlated. If only one pollutant is being associated with the endpoint in this analysis (e.g., chronic bronchitis is associated only with PM in this analysis), this is not a problem.

4.1.8 Pooling Study Results

When only a single study estimated the C-R relationship between a pollutant and a given health endpoint, the estimation of a population cell-specific incidence change, Δy, is straightforward, as noted above. When several studies have estimated C-R relationships between a pollutant and a given health endpoint, the results of the studies can be pooled to derive a single estimate of the function. If the functional forms, pollutant averaging times, and study populations are all the same (or very similar), a pooled, "central tendency" C-R function can be derived from multiple study-specific C-R functions. Even if there are differences among the studies, however, that make a pooled C-R function infeasible, a pooled estimate of the incidence change, Δy,

and/or the monetary benefit of the incidence change can be obtained by incorporating the appropriate air quality data into the study-specific C-R functions and pooling the resulting study-specific predictions of incidence change. Similarly, study-specific predictions of incidence change can be combined with unit dollar values to produce study-specific predictions of benefits.

Whether the pooling is done in "coefficient space," "incidence change space," or "dollar space," the question of the relative weights assigned to the estimates (of coefficients, incidence changes, or dollar benefits) from each input study must be addressed. One possibility is simply averaging the estimates from all the studies. This has the advantage of simplicity, but the disadvantage of not taking into account the measured uncertainty of each of the estimates. Estimates with great uncertainty surrounding them are given the same weight as estimates with very little uncertainty.

An alternative approach to pooling incidence estimates from different studies is to give more weight to studies with little estimated variance than to studies with a great deal of estimated variance. The exact way in which weights are assigned to estimates from different studies in a pooled analysis depends on the underlying assumption about how the different estimates are related to each other. Under the assumption that there is actually a distribution of true effect coefficients, or β's, that differ by location and/or study (referred to as the random effects model), the different coefficients reported by different studies may be estimates of *different* underlying coefficients, rather than just different estimates of the same coefficient. In contrast to the "fixed-effects" model (which assumes that there is only one β everywhere), the random-effects model allows the possibility that different studies are estimating different parameters.[7]

A third approach to pooling studies is to apply subjective weights to the studies, rather than conducting a random effects pooling analysis. If the analyst is aware of specific strengths and weaknesses of the studies involved, this prior information may be used as input to the calculation of weights which reflect the relative reliability of the estimates from the studies.

In those cases in which pooling of information from multiple studies was an option in this analysis, pooling was done in both "incidence change space" and "dollar benefit space." The hypothesis of fixed effects was tested. If this hypothesis was rejected, an underlying random effects model was used as the basis for weighting of studies. A more detailed description of the pooling procedure used is given below in the section on hospital admissions.

4.2 VALUING CHANGES IN HEALTH EFFECTS

This section discusses a number of issues that arise in valuing changes in health effects. The first section provides some background on willingness to pay (WTP). The second section discusses the possibility that as income changes then WTP would also change. The third section describes how WTP estimates, that

[7] In studies of the effects of PM_{10} on mortality, for example, if the composition of PM_{10} varies among study locations the underlying relationship between mortality and PM_{10} may be different from one study location to another. For example, fine particles make up a greater fraction of PM_{10} in Philadelphia County than in Southeast Los Angeles County. If fine particles are disproportionately responsible for mortality relative to coarse particles, then one would expect the true value of β for PM_{10} in Philadelphia County to be greater than the true value of β for PM_{10} in Southeast Los Angeles County. This would violate the assumption of the "fixed effects" model. However, applying a random effects model assumes that the observed set of coefficients is representative of coefficients in the policy region.

were originally calculated in 1990 dollars, are corrected for inflation to get estimates in 1997 dollars. In the last section, we briefly review how we aggregate benefits estimates.

4.2.1 Willingness To Pay Estimation

WTP is a measure of value an individual places on gaining an outcome viewed as desirable, be it something that can be purchased in a market or not. The WTP measure, therefore, is the amount of money such that the individual would be indifferent between having the good (or service) and having the money. An alternative measure of economic value is willingness to accept (WTA) a monetary compensation to offset a deterioration in welfare, such that the individual would be indifferent between having the money and not having the deterioration. Whether WTP or WTA is the appropriate measure depends on how property rights are assigned. Consider an increase in air pollution. If society has assigned property rights so that people have a right to clean air, then they must be compensated for an increase in the level of air pollution. The appropriate measure of the value of avoiding an increase in air pollution, in this case, would be the amount people would be willing to accept in compensation for the more polluted air. If, on the other hand, society has not assigned people the right to clean air, then the appropriate measure of the value of avoiding an increase in air pollution would be what people are willing to pay to avoid it. The assignment of property rights in our society is unclear. WTP is by far the more common measure used in benefits analyses, however, reflecting the fact that this is a much more common measure in the empirical valuation literature. In this analysis, wherever possible, the valuation measures are in terms of WTP. Where such estimates are not available, alternative measures are used, such as cost-of-illness and wage-risk studies. These are discussed for each endpoint where applicable.

For both market and non-market goods, WTP reflects individuals' preferences. Because preferences are likely to vary from one individual to another, WTP for both market (e.g., the purchase of a new automobile) and non-market goods (e.g., health-related improvements in environmental quality) is likely to vary from one individual to another. In contrast to market goods, non-market goods such as environmental quality improvements, are public goods, whose benefits are shared by many individuals. The individuals who benefit from the environmental quality improvement may have different WTPs for this non-market good. The total social value of the good is the sum of the WTPs of all individuals who "consume" (i.e., benefit from) the good.

In the case of health improvements related to pollution reduction, it is not certain specifically who will receive particular benefits of reduced pollution. For example, the analysis may predict 100 hospital admissions for respiratory illnesses avoided, but the analysis does not estimate which individuals will be spared those cases of respiratory illness that would have required hospitalization. The health benefits conferred on individuals by a reduction in pollution concentrations are, then, actually *reductions in the risk* of having to endure certain health problems. These benefits (reductions in risk) may not be the same for all individuals (and could be zero for some individuals). Likewise, the WTP for a given benefit is likely to vary from one individual to another. In theory, the total social value associated with the decrease in risk of a given health problem resulting from a given reduction in pollution concentrations is:

$$\sum_{i=1}^{N} WTP_i\left(B_i\right) ,$$

where B_i is the benefit (i.e., the reduction in risk of having to endure the health problem) conferred on the i^{th} individual (out of a total of N) by the reduction in pollution concentrations, and $WTP_i(B_i)$ is the i^{th} individual's WTP for that benefit.

If a reduction in pollution concentrations affects the risks of several health endpoints, the total health-related social value of the reduction in pollution concentrations is:

$$\sum_{i=1}^{N}\sum_{j=1}^{J} WTP_i\left(B_{i,j}\right),$$

where B_{ij} is the benefit related to the j^{th} health endpoint (i.e., the reduction in risk of having to endure the j^{th} health problem) conferred on the i^{th} individual by the reduction in pollution concentrations, and $WTP_i(B_{ij})$ is the i^{th} individual's WTP for that benefit.

The reduction in risk of each health problem for each individual is not known, nor is each individual's WTP for each possible benefit he or she might receive known. Therefore, in practice, benefits analysis estimates the value of a *statistical* health problem avoided. For example, although a reduction in pollutant concentrations may save actual lives (i.e., avoid premature mortality), whose lives will be saved cannot be known *ex ante*. What is known is that the reduction in air pollutant concentrations results in a reduction in mortality risk. It is this reduction in mortality risk that is valued in a monetized benefit analysis. Individual WTPs for small reductions in mortality risk are summed over enough individuals to infer the value of a *statistical* life saved. This is different from the value of a particular, identified life saved. Rather than "WTP to avoid a death," then, it is more accurate to use the term "the value of a statistical life."

Suppose, for example, that a given reduction in PM concentrations results in a decrease in mortality risk of 1/10,000. Then for every 10,000 individuals, one individual would be expected to die in the absence of the reduction in PM concentrations (who would not die in the presence of the reduction in PM concentrations). If WTP for this 1/10,000 decrease in mortality risk is $500 (assuming, for now, that all individuals' WTPs are the same), then the value of a statistical life is 10,000 x $500, or $5 million.

A given reduction in PM concentrations is unlikely, however, to confer the same risk reduction (e.g., mortality risk reduction) on all exposed individuals in the population. (In terms of the expressions above, B_i is not necessarily equal to B_j , for $i \neq j$). In addition, different individuals may not be willing to pay the same amount for the same risk reduction. The above expression for the total social value associated with the decrease in risk of a given health problem resulting from a given reduction in pollution concentrations may be rewritten to more accurately convey this. Using mortality risk as an example, for a given unit risk reduction (e.g., 1/1,000,000), the total mortality-related benefit of a given pollution reduction can be written as:

$$\sum_{i=1}^{N}\int_{0}^{n_i} marginal\ WTP_i(x)dx\ ,$$

where marginal $WTP_i(x)$ is the i^{th} individual's marginal willingness to pay curve, n_i is the number of units of risk reduction conferred on the i^{th} exposed individual as a result of the pollution reduction, and N is the total number of exposed individuals.

The values of a statistical life implied by the value-of-life studies were derived from specific risk reductions. Implicit in applying these values to a situation involving possibly different risk reductions is the assumption that the marginal willingness to pay curve is horizontal – that is, that WTP for n units of risk reduction is n times WTP for one unit of risk reduction. If the marginal willingness to pay curve is horizontal, the integral in the above expression becomes a simple product of the number of units of risk reduction times the WTP per unit. The total mortality-related benefit (the expression above) then becomes:

$$\sum_{i=1}^{N} \left(number\ of\ units\ of\ risk\ reduction\right)_i \cdot \left(\frac{WTP_i}{unit\ of\ risk\ reduction} \right).$$

If different subgroups of the population have substantially different WTPs for a unit risk reduction and substantially different numbers of units of risk reduction conferred on them, then estimating the total social benefit by multiplying the population mean WTP (MWTP) to save a statistical life times the predicted number of statistical lives saved could yield a biased result. Suppose, for example, that older individuals' WTP per unit risk reduction is less than that of younger individuals (e.g., because they have fewer years of expected life to lose). Then the total benefit will be less than it would be if everyone's WTP were the same. In addition, if each older individual has a larger number of units of risk reduction conferred on him (because a given pollution reduction results in a greater absolute reduction in risk for older individuals than for younger individuals), this, in combination with smaller WTPs of older individuals, would further reduce the total benefit.

While the estimation of WTP for a market good (i.e., the estimation of a demand schedule) is not a simple matter, the estimation of WTP for a non-market good, such as a decrease in the risk of having a particular health problem, is substantially more difficult. Estimation of WTP for decreases in very specific health risks (e.g., WTP to decrease the risk of a day of coughing or WTP to decrease the risk of admission to the hospital for respiratory illness) is further limited by a paucity of information.[8] Derivation of the dollar value estimates discussed below was often limited by available information.

4.2.2 Change Over Time in WTP in Real Dollars

The WTP for health-related environmental improvements (in real dollars) could change between now and the year 2007. If real income increases between now and the year 2007, for example, it is reasonable to expect that WTP, in real dollars, would also increase. Below we summarize the evidence regarding this effect, however we do not adjust our results in this analysis, because of the uncertainty regarding the size of the effect.

Based on historical trends, the U.S. Bureau of Economic Analysis projects that, for the United States as a whole as well as for regions and states within the U.S., mean per capita real income will increase. For the U.S. as a whole, for example, mean per capita personal income is projected to increase by about 16 percent from 1993 to 2005 (U.S. Bureau of Economic Analysis, 1995).

The mean WTP in the population is the correct measure of the value of a health problem avoided, and that WTP is a function of income. If the mean per capita real income rises by the year 2007, the mean WTP would probably rise as well. While this is most likely true, the degree to which mean WTP rises with a rise

[8] Some health effects, such as technical measures of pulmonary functioning (e.g., forced expiratory volume in one second) are frequently studied by epidemiologists, but there has been very little work by economists on valuing these changes (e.g., Ostro et al., 1989).

in mean per capita income is unclear unless the elasticity of WTP with respect to changes over time in real income is 1.0.

There is some evidence (Loehman and De, 1982; Mitchell and Carson, 1986; Alberini et al., 1997) that the elasticity of WTP for health-related environmental improvements with respect to real income is less than 1.0, possibly substantially so. If this is the case, then changes in mean income cannot be readily translated into corresponding changes in mean WTP. Although an increase in mean income is likely to imply an increase in mean WTP, the degree of the increase cannot be ascertained from information only about the means.

Several factors, in addition to real income, that could affect the estimated benefit associated with reductions in air pollution concentrations could also change in the future Demographic characteristics of exposed populations could change. Technological advances could change both the nature of precursor emissions to the ambient air and the susceptibility of individuals to air pollution. Any such changes would be reflected in C-R functions that differ from those that describe current relationships between ambient concentrations and the various health endpoints. While adjustments of WTP to reflect changes in real income are of interest, such adjustments would by no means necessarily reflect all possible changes that could affect the future benefits of reduced air pollution.

4.2.3 Adjusting Benefits Estimates from 1990 Dollars to 1999 Dollars

This section describes the methods used to convert benefits estimates to constant 1999 dollars. This is necessary because some of the estimates that we use are in 1990 dollars. The method that we use depends on the basis of the benefits estimates. Exhibit 4-1 delineates these bases.[9]

Exhibit 4-1 Bases of Benefits Estimation

Basis of Benefit Estimation	Benefit Endpoints
Cost of illness	Hospital admissions avoided
Direct estimates of WTP	Statistical lives saved Chronic bronchitis Morbidity endpoints using WTP
Earnings	Work loss days (WLDs) avoided

Benefits estimates based on cost-of-illness have been adjusted by using the consumer price indexes (CPI-Us) for medical care. Because increases in medical costs have been significantly greater than the general rate of inflation, using a general inflator (the CPI-U for "all items" or some other general inflator) to adjust from 1990 to 1999 dollars would downward bias cost-of-illness estimates in 1999 dollars.

Benefits estimates based directly on estimates of WTP have been adjusted using the CPI-U for "all items." The CPI-Us, published by the U.S. Dept. of Labor, Bureau of Labor Statistics, can be found in

[9]Agricultural benefits are discussed in Chapter 3.

Council of Economic Advisers (2000, Table B-58). An overview of the adjustments from 1990 to 1999 dollars for WTP-based and cost-of-illness based valuations is given in Exhibit 4-2.

Exhibit 4-2 Consumer Price Indexes Used to Adjust WTP-Based and Cost-of-Illness-Based Benefits Estimates from 1990 Dollars to 1999 Dollars

	1990 (1)	1999 (2)	Adjustment Factor [a] (2)/(1)	Relevant Endpoints
CPI-U for "All Items" [b]	130.7	166.6	1.275	WTP-based valuation: 1. Statistical lives saved [c] 2. Chronic bronchitis 3. Morbidity endpoints using WTP [d]
CPI-U for Medical Care [b]	162.8	250.6	1.539	Cost-of-illness based valuation: Hospital admissions avoided [e]

[a] Benefits estimates in 1990 dollars are multiplied by the adjustment factor to derive benefits estimates in 1999 dollars.

[b] Source: Dept. of Labor, Bureau of Labor Statistics; reported in Council of Economic Advisers (2000, Table B-58)

[c] Adjustments to 1990 $ were originally made by Industrial Economics Inc. using the CPI-U for "all items" (IEc1992).

[d] Adjustments of WTP-based benefits for morbidity endpoints to 1990 $ were originally made by Industrial Economics Inc. (1993) using the CPI-U for "all items."

[e] Adjustments of cost-of-illness based estimates of all hospital admissions avoided to 1990 $ were made by Abt Associates Inc. in previous analyses, such as the NAAQS RIA (U.S. EPA, 1997).

Benefit estimates for work loss days (WLDs) avoided have in past analyses been based on either the mean or median daily wage. For this analysis, the valuation of the benefit of avoiding a work loss day used the median daily income rather than the mean, consistent with economic welfare theory. The income distribution in the United States is highly skewed, so that the mean income is substantially larger than the median income. However, the incomes of those individuals who lose work days due to pollution are not likely to be a random sample from this income distribution. In particular, the probability of being drawn from the upper tail of the distribution is likely to be substantially less than the probability mass in that tail. To reflect this likelihood, we used the median income rather than the mean income as the value of a work loss day. This is explained more fully below in the section on valuing work loss days.

The benefits estimates for WLDs avoided can be put into 1999 dollars in several ways. One approach is to obtain the 1998 median weekly earnings (the most up-to-date measure of earnings available), divide by five to derive the median daily earnings, and adjust the median earnings from 1998 to 1999 dollars. This is an alternative to relying on adjustments from 1990 to 1999 dollars. The median weekly earnings of full-time wage and salary workers in 1998 was $523 (U.S. Bureau of the Census 1998, Table 696). This implies a median daily earnings of $104.6, or rounded to the nearest dollar, $105. Alternatively, we can adjust the median daily wage for 1990 to 1999 dollars, using the CPI-U for "all items." The result turns out to be the same. The adjustment factor (the ratio of the 1999 CPI-U to the 1990 CPI-U) is 1.275. Applied to the median daily earnings of $82.4 in 1990, the median daily earnings in 1997 would be $105.1, or rounded to the nearest dollar, $105.

4.2.4 Aggregation of Monetized Benefits

The total monetized benefit associated with attaining a given set of pollution changes in a given location is just the sum of the non-overlapping benefits associated with these changes. In theory, the total health-related social value of the reduction in pollution concentrations is:

$$\sum_{i=1}^{N}\sum_{j=1}^{J} WTP_i\left(B_{i,j}\right),$$

where B_{ij} is the benefit related to the j^{th} health endpoint (i.e., the reduction in probability of having to endure the j^{th} health problem) conferred on the i^{th} individual by the reduction in pollution concentrations, and $WTP_i(B_{ij})$ is the i^{th} individual's WTP for that benefit.

As stated earlier, the reduction in probability of each health problem for each individual is not known, nor do we know each individual's WTP for each possible benefit he or she might receive. Therefore, in practice, benefits analysis estimates the value of a *statistical* health problem avoided. The benefit in the k^{th} location associated with the j^{th} health endpoint is just the change in incidence of the j^{th} health endpoint in the k^{th} location, Δy_{jk}, times the value of an avoided occurrence of the j^{th} health endpoint.

Assuming that WTP to avoid the risk of a health effect varies from one individual to another, there is a *distribution* of WTPs to avoid the risk of that health effect. This population distribution has a mean. It is this population mean of WTPs to avoid or reduce the risk of the j^{th} health effect, $MWTP_j$, that is the appropriate value in the benefit analysis.[10] The monetized benefit associated with the j^{th} health endpoint resulting from attainment of standard(s) in the k^{th} location, then, is:

$$benefit_{jk} = \Delta y_{jk} \cdot MWTP_j$$

and total monetized benefit in the k^{th} location (TMB_k) may be written as the sum of the monetized benefits associated with all non-overlapping endpoints:

$$TMB_k = \sum_{j=1}^{N} \Delta y_{jk} \cdot MWTP_j \ .$$

The location- and health endpoint-specific incidence change, Δy_{jk}, is modeled as the population response to the change in pollutant concentrations in the k^{th} location. Assuming a log-linear C-R function, the change in incidence of the j^{th} health endpoint in the k^{th} location corresponding to a change in PM, ΔPM_k, in the k^{th} location is:

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[10]The population of interest has not been defined. In a location-specific analysis, the population of interest is the population in that location. The MWTP is ideally the mean of the WTPs of all individuals in the location. There is insufficient information, however, to estimate the MWTP for any risk reduction in any particular location. Instead, estimates of MWTP for each type of risk reduction will be taken to be estimates of the MWTP in the United States as a whole, and it will be assumed that $MWTP_i$, i=1, ..., N in each location is approximately the same as in the United States as a whole.

$$\Delta y_{jk} = y_{jk} \cdot \left(e^{b_{jk} \cdot \Delta PM_k} - 1 \right) ,$$

where y_{jk} is the baseline incidence of the j^{th} health endpoint in the k^{th} location and β_{jk} is the value of β_j, the coefficient of PM in the C-R relationship between PM and the j^{th} health endpoint, in the k^{th} location.

This approach assumes that there is a *distribution* of β_j's across the United States, that is, that the value of β_j in one location may not be the same as the value of β_j in another location. The value of β_j in the k^{th} location is denoted as β_{jk}.

The total PM-related monetized benefit for the k^{th} location can now be rewritten as:

$$TMB_k = \sum_{j=1}^{N} y_{jk} \cdot \left(e^{b_{jk} \cdot \Delta PM_k} - 1 \right) \cdot MWTP_j ,$$

The total monetized PM-related benefit to be estimated for a location is thus a function of 2N parameters: the coefficient of PM, β_{jk}, in the C-R function for the j^{th} health endpoint, for j=1, ..., N, specific to the k^{th} location, and the population mean WTP to reduce the risk of the j^{th} health endpoint, $MWTP_j$, j=1, ..., N.

The above model assumes that total monetized benefit is the sum of the monetized benefits from all non-overlapping endpoints. If two or more endpoints were overlapping, or if one was contained within the other (as, for example, hospital admissions for Chronic Obstructive Pulmonary Disease (COPD) is contained within hospital admissions for "all respiratory illnesses"), then adding the monetized benefits associated with those endpoints would result in double (or multiple) counting of monetized benefits. If some endpoints that are not contained within endpoints included in the analysis are omitted, then the aggregated monetized benefits will be less than the total monetized benefits.

The total monetized benefit (TMB) is the sum of the total monetized benefits achieved in each location:

$$TMB = \sum_{k=1}^{K} TMB_k$$

where TMB_k denotes the total monetized benefit achieved in the k^{th} location, and K is the number of locations.

Theoretically, the nation-wide analysis could use location-specific C-R functions to estimate location-specific benefits. Total monetized benefits (TMB), then, would just be the sum of these location-specific benefits:

$$TMB = \sum_{k=1}^{K} TMB_k = \sum_{k=1}^{K} \sum_{j=1}^{N} y_{jk\cdot} \left(e^{b_{jk} \cdot \Delta PM_k} - 1 \right) \cdot MWTP_j ,$$

There are many locations in the United States, however, and the individual location-specific values of β_j (the β_{jk}'s) are not known.[11] Since the national incidence of the j^{th} health endpoint attributed to PM, I_j , is a continuous function of the set of β_{jk}'s, that is, since:

$$I_j = \sum_{k=1}^{K} \Delta y_{jk} = \sum_{k=1}^{K} y_{jk} \cdot \left(e^{b_{jk} \cdot \Delta PM_k} - 1 \right) ,$$

is a continuous function of the set of β_{jk}'s, there is some value of β_j , which can be denoted β_j*, that, if applied in *all* locations, would yield the same result as the proper set of location-specific β_{jk}'s. This follows from the Intermediate Value Theorem. While β_j* will result in overestimates of incidence in some locations, it will result in underestimates in others. If β_j* is applied in all locations, however, the *total regional* change in incidence will be correct. That is,

$$I_j = \sum_{k=1}^{K} \Delta y_{jk} = \sum_{k=1}^{K} y_{jk} \cdot \left(e^{b_j^* \cdot \Delta PM_k} - 1 \right) ,$$

$$= \sum_{k=1}^{K} y_{jk} \cdot \left(e^{b_{jk} \cdot \Delta PM_k} - 1 \right) .$$

The total regional monetized PM-related benefit can now be rewritten as:

$$TMB_k = \sum_{j=1}^{N} \sum_{k=1}^{K} y_{jk} \cdot \left(e^{b_j^* \cdot \Delta PM_k} - 1 \right) \cdot MWTP_j .$$

The total regional monetized (PM-related) benefit is thus a function of 2N population means: the β* for the j^{th} health endpoint (β_j* , for j=1, ..., N) and the population mean WTP to reduce the risk of the j^{th} health endpoint ($MWTP_j$, j=1, ..., N).

Both the endpoint-specific coefficients (the ÿ$_j$'s) and the endpoint-specific mean WTPs (the $MWTP_j$'s) are uncertain. One approach to estimating the total monetized benefit is to simply use the mean values of the endpoint-specific coefficients and mean WTPs in the above formula. We term this approach the "simple mean." Alternatively, we can characterize not only the mean total monetized benefit but the distribution of possible values of total monetized benefit, using a Monte Carlo approach. The Monte Carlo approach has three steps. First, in each of 5000 iterations, we randomly select a value from the distribution of (national) incidence change of the health effect. Second, we randomly select a value from the distribution of unit dollar values for that health effect. And third, we multiply the two values. The result is a distribution of (5000) monetized

[11]This may also be true of the y_{ij}'s. It may be desirable to apply the uncertainty analysis used for the β's to these population parameters as well. In the current discussion, however, it is assumed that the location-specific incidences are known and therefore have no uncertainty associated with them. It is also assumed that $MWTP_i$ is the same in all locations.

benefits associated with the given health effect. From this distribution, we present the mean as well as the 5th and 95th percentiles. We discuss the background of the Monte Carlo in the following sub-section.

4.3 CHARACTERIZATION OF UNCERTAINTY

In any complex analysis using estimated parameters and inputs from numerous different models, there are likely to be many sources of uncertainty. This analysis is no exception. There are many inputs that are used to derive the final estimate of benefits, including emission inventories, air quality models (with their associated parameters and inputs), epidemiological estimates of C-R functions, estimates of values (both from WTP and cost-of-illness 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 emissions can lead to much larger impacts on total benefits.

Exhibit 4-3 summarizes the wide variety of sources for uncertainty in this analysis. Some key sources of uncertainty in each stage of the benefits analysis are:

- gaps in scientific data and inquiry
- variability in estimated relationships, such as C-R functions, 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
- biases due to omissions or other research limitations.

Our approach to characterizing model uncertainty in the estimate of benefits is to present a primary estimate, based on the best available scientific literature and methods, with associated statistical uncertainty bounds. We used the REMSAD-based air quality data to calculate primary benefits in this analysis. For the sake of comparison, however, alternative estimates of benefits based upon an alternative air quality model (the S-R Matrix) are presented in Appendix E.

In some cases, it was not possible to quantify uncertainty. For example, many benefits categories, while known to exist, do not have enough information available to provide a quantified or monetized estimate. The uncertainty regarding these endpoints is such that we could determine neither a primary estimate nor a plausible range of values. Of the primary endpoints we do quantify, a number of alternative measures of mortality incidence can be calculated. We present the full suite of alternative mortality calculations as a way to address the range of plausible mortality incidence estimates. This is discussed in greater detail in Chapter 5.

A final approach to measuring uncertainty is through probabilistic assessments where statistical uncertainty bounds are calculated for each endpoint. We discuss statistical uncertainty bounds in the following section.

Exhibit 4-3 Key Sources of Uncertainty in the Benefit Analysis

1. Uncertainties Associated With Concentration-Response Functions
-The value of the PM-coefficient in each C-R function. -Application of a single C-R function to pollutant changes and populations in all locations. -Similarity of future year C-R relationships to current C-R relationships. -Correct functional form of each C-R relationship. -Extrapolation of C-R relationships beyond the range of PM concentrations observed in the study.
2. Uncertainties Associated With PM Concentrations
-Estimating future-year baseline daily PM concentrations. -Estimating the change in PM resulting from the control policy.
3. Uncertainties Associated with PM Mortality Risk
-No scientific literature supporting a direct biological mechanism for observed epidemiological evidence. -Direct causal agents within the complex mixture of PM responsible for reported health effects 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. -Possible confounding in the epidemiological studies of $PM_{2.5}$, effects with other factors (e.g., other air pollutants, weather, indoor/outdoor air, etc.). -The extent to which effects reported in the long-term 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
-What portion of the PM-related long-term exposure mortality effects associated with changes in annual PM levels would occur in a single year, and what portion 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 well approximate what baseline incidence rates will be in the year 2030. -Projected population and demographics -- used to derive incidences – may not well approximate future-year population and demographics.
6. Uncertainties Associated With Economic Valuation
-Unit dollar values associated with health 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 benefits estimates are limited to the available C-R functions. Thus, unquantified benefit categories will cause total benefits to be underestimated.

4.3.1 Statistical Uncertainty Bounds

Although there are several sources of uncertainty affecting estimates of endpoint-specific benefits, the sources of uncertainty that are most readily quantifiable in this analysis are the incidence changes (deriving from uncertainty about the C-R relationships) and uncertainty about unit dollar values. The total dollar benefit associated with a given endpoint depends on how much the endpoint will change due to the final standard (e.g., how many premature deaths will be avoided) and how much each unit of change is worth (e.g., how much a

premature death avoided is worth).[12] Based on these distributions, we provide estimates of the 5th and 95th percentile values of the distribution of estimated benefits. However, we hasten to add that this omits important sources of uncertainty, such as the contribution of air quality changes, baseline population incidences, projected populations exposed, transferability of the C-R function to diverse locations, and uncertainty about premature mortality. Thus, a confidence interval based on the standard error would provide a misleading picture about the overall uncertainty in the estimates. The empirical evidence about uncertainty is presented where it is available.

Both the uncertainty about the incidence changes and uncertainty about unit dollar values can be characterized by *distributions*. Each "uncertainty distribution" characterizes our beliefs about what the true value of an unknown (e.g., the true change in incidence of a given health effect) is likely to be, based on the available information from relevant studies.[13] Unlike a sampling distribution (which describes the possible values that an *estimator* of an unknown value might take on), this uncertainty distribution describes our beliefs about what values the unknown value itself might be. Such uncertainty distributions can be constructed for each underlying unknown (such as a particular pollutant coefficient for a particular location) or for a function of several underlying unknowns (such as the total dollar benefit of a regulation). In either case, an uncertainty distribution is a characterization of our beliefs about what the unknown (or the function of unknowns) is likely to be, based on all the available relevant information. Uncertainty statements based on such distributions are typically expressed as 90 percent credible intervals. This is the interval from the fifth percentile point of the uncertainty distribution to the ninety-fifth percentile point. The 90 percent credible interval is a "credible range" within which, according to the available information (embodied in the uncertainty distribution of possible values), we believe the true value to lie with 90 percent probability.

The uncertainty about the total dollar benefit associated with any single endpoint combines the uncertainties from these two sources, and is estimated with a Monte Carlo method. In each iteration of the Monte Carlo procedure, a value is randomly drawn from the incidence distribution and a value is randomly drawn from the unit dollar value distribution, and the total dollar benefit for that iteration is the product of the two.[14] If this is repeated for many (e.g., thousands of) iterations, the distribution of total dollar benefits associated with the endpoint is generated.

Using this Monte Carlo procedure, a distribution of dollar benefits may be generated for each endpoint. The mean and median of this Monte Carlo-generated distribution are good candidates for a point estimate of total monetary benefits for the endpoint. As the number of Monte Carlo draws gets larger and larger, the Monte Carlo-generated distribution becomes a better and better approximation to the underlying uncertainty distribution of total monetary benefits for the endpoint. In the limit, it is identical to the underlying distribution.

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[12] Because this is a regional analysis in which, for each endpoint, a single C-R function is applied everywhere, there are two sources of uncertainty about incidence: (1) statistical uncertainty (due to sampling error) about the true value of the pollutant coefficient in the location where the C-R function was estimated, and (2) uncertainty about how well any given pollutant coefficient approximates β^*.

[13] Although such an "uncertainty distribution" is not formally a Bayesian posterior distribution, it is very similar in concept and function (see, for example, the discussion of the Bayesian approach in Kennedy1990, pp. 168-172).

[14] This method assumes that the incidence change and the unit dollar value for an endpoint are stochastically independent.

4.3.2 Unquantified Benefits

In considering the monetized benefits estimates, the reader should remain aware of the limitations. One significant limitation of benefits analyses is the inability to quantify many of the PM adverse effects. For many effects, reliable C-R functions and/or valuation functions are not currently available such as infant mortality. In general, if it were possible to monetize these benefits categories, the benefits estimates presented here would increase.

5. HEALTH BENEFITS

The most significant monetized benefits of reducing ambient concentrations of PM are attributable to reductions in health risks associated with air pollution. This Chapter describes individual effects and the methods used to quantify and monetize changes in the expected number of incidences of various health effects.

We estimate the incidence of adverse health effects using PM-based C-R functions. The changes in incidence of PM-related adverse health effects and corresponding monetized benefits associated with these changes are estimated separately. Exhibit 5-1 presents the PM-related health endpoints included in this analysis, and Exhibit 5-2 presents the unit monetary values for each of these endpoints and associated uncertainty distributions. Appendix F presents the functional forms for each C-R function and their derivation.

Exhibit 5-1 PM-Related Health Endpoints

Endpoint	Population	PM Measure	Study
Mortality			
Associated with long-term exposure	Ages 30+	$PM_{2.5}$	(Krewski et al., 2000), reanalysis of Pope et al., 1995, using the annual mean and all-cause mortality, 63 city Dichotomous samplers.
Chronic Illness			
Chronic Bronchitis	varies by study	varies by study	Two studies [a]: Schwartz (1993)and Abbey et al. (1995b)
Hospital Admissions			
COPD (ICD-9 codes 4490-492, 494-496)	age 65+	PM_{10}	(Samet et al., 2000)[b]
Pneumonia (ICD-9 codes 480-487)	age 65+	PM_{10}	(Samet et al., 2000)[b]
Cardiovascular (ICD-9 codes 390-429)	age 65+	PM_{10}	(Samet et al., 2000)[b]
Asthma (ICD code 493)	< 65	$PM_{2.5}$	(Sheppard et al., 1999)
Asthma-related ER visits	< 65	PM_{10}	Schwartz et al. (1993)
Respiratory Symptoms/Illnesses Not Requiring Hospitalization			
Acute bronchitis	Ages 8-12	$PM_{2.5}$	Dockery et al. (1989)
Lower respiratory symptoms (LRS)	Ages 7-14	$PM_{2.5}$	Schwartz et al. (1994)
Upper respiratory symptoms (URS)	Asthmatics, ages 9-11	PM_{10}	Pope et al. (1991)
Minor restricted activity day (MRAD) (adjusted for asthma attacks)	Ages 18-65	$PM_{2.5}$ (estimated)	Ostro and Rothschild (1989),
Work loss days (WLDs)	Ages 18-65	$PM_{2.5}$	Ostro (1987)
Asthma Attacks	asthmatics, all ages	PM_{10}	Whittemore and Korn (1980)

[a] The incidence changes, and the associated monetized benefits, predicted by two studies are pooled. The separate studies and the method of pooling are described below.

[b] The pooled estimate, based on distributed lag models in each of 14 cities, is used because the estimated coefficients based on pooling are substantially more stable than the individual city-specific estimates.

Exhibit 5-2 Unit Values for Economic Valuation of Health Endpoints (1999 $)

Health Endpoint	Mean Estimate [a]	Uncertainty Distribution [a]
Mortality		
Value of a statistical life	$6.12 million per statistical life [b]	Weibull distribution, mean = $6.12 million; std. dev. = $4.13 million.
Chronic Bronchitis		
WTP approach	$331,000 per case	A Monte Carlo-generated distribution, based on three underlying distributions.
Hospital Admissions		
Pneumonia (ICD codes 480-487)	— [d]	None available [c]
COPD (ICD codes 490-492, 494-496)	— [d]	None available [c]
Respiratory	— [d]	None available [c]
Cardiovascular	— [d]	None available [c]
Asthma-related ER visits	$298.62 per visit	Triangular distribution centered at $280 over the interval [$221.65, $414.07].
Respiratory Ailments Not Requiring Hospitalization		
Acute bronchitis	$57.34 per case	Continuous uniform distribution over [$16.57, $98.15].
Lower resp. Symptoms	$15.30 per symptom-day	Continuous uniform distribution over [$6.37, $24.22].
Upper resp. Symptoms	$24.22 per symptom-day	Continuous uniform distribution over [$8.93,$42.06].
Minor respiratory activity day (MRAD)	$48.43 per day	Triangular distribution centered at $48.43 over [$20.34, $77.76].
Work loss days	$105 per day	None available
Asthma attacks	$40.79 per symptom-day	Continuous uniform distribution over [$15.30, $68.83]

[a] The derivation of each of the estimates is discussed in the text. All WTP-based dollar values were obtained by multiplying rounded 1990 $ values used in the §812 Prospective Analysis by 1.275 to adjust to 1999 $.

[b] An adjustment for lagged mortality, discussed in section 5.1.3, is used in this analysis. The lag-adjusted value of a statistical life is approximately 92% of the full value presented here.

[c] Standard errors were not available. However, the sample sizes on which these estimates (from the Agency for Healthcare Research and Policy's Healthcare Cost and Utilization Project) are very large and the standard errors are therefore negligible.

[d] Cost of illness unit dollar values were derived for each separate set of ICD codes for which a C-R model was estimated. These are given below.

5.1 PREMATURE MORTALITY

Changes in PM concentrations on mortality are estimated as a count of the expected number of deaths avoided due to a given reduction in PM concentrations. Mortality is a very important health endpoint in this economic analysis due to the high monetary value associated with risks to life.

There are two types of exposure to elevated levels of air pollution that may result in premature mortality. Acute (short-term) exposure (e.g., exposure on a given day) to peak pollutant concentrations may result in excess mortality on the same day or within a few days of the elevated exposure. Chronic (long-term) exposure (e.g., exposure over a period of a year or more) to levels of pollution that are generally higher may

result in mortality in excess of what it would be if pollution levels were generally lower. The excess mortality that occurs will not necessarily be associated with any particular episode of elevated air pollution levels.

Exhibit 5-3 Alternative Mortality Concentration-Response Functions

Endpoint	Population	PM Indicator	Study
Associated with long-term exposure	Ages 30+	$PM_{2.5}$	(Krewski et al., 2000), reanalysis of Pope et al., 1995, using the annual mean and all-cause mortality, 63 city Dichotomous sampler
Associated with long-term exposure	Ages 30+	$PM_{2.5}$	(Krewski et al., 2000), reanalysis of Pope et al., 1995, using the annual median, 50 city
Associated with long-term exposure	Ages 30+	$PM_{2.5}$	Pope et al. (1995)
Associated with long-term exposure	All ages	$PM_{2.5}$	Dockery et al. (1993)
Associated with short-term exposure	All ages	$PM_{2.5}$	(Schwartz et al., 1996)

5.1.1 Short-Term Versus Long-Term Studies

There are two types of epidemiological studies that examine the relationship between mortality and exposure. Long-term studies (e.g., Pope et al., 1995) estimate the association between long-term (chronic) exposure to air pollution and the survival of members of a large study population over an extended period of time. Such studies examine the health endpoint of concern in relation to the general long-term level of the pollutant of concern, for example, relating annual mortality to some measure of annual pollutant level. Daily peak concentrations would impact the results only insofar as they affect the measure of long-term (e.g., annual) pollutant concentration. In contrast, short-term studies relate daily levels of the pollutant to daily mortality. By their basic design, daily studies can detect acute effects but cannot detect the effects of long-term exposures. A chronic exposure study design (a prospective cohort study, such as the Pope study) is best able to identify the long-term exposure effects, and may detect some of the short-term exposure effects as well. Because a long-term exposure study may detect some of the same short-term exposure effects detected by short-term studies, including both types of study in a benefit analysis would likely result in some degree of double counting of benefits. While the long-term study design is preferred, these types of studies are expensive to conduct and consequently there are relatively few well designed long-term studies.

5.1.2 Degree of Prematurity of Mortality

It is possible that the short-term studies are detecting an association between PM and mortality that is primarily occurring among terminally ill people. Critics of the use of short-term studies for policy analysis purposes correctly point out that an added risk factor that results in terminally ill people dying a few days or weeks earlier than they otherwise would have (referred to as "short-term harvesting") is potentially included in the measured PM mortality "signal" detected in such a study. While some of the detected excess deaths may have resulted in a substantial reduction in lifespan, others may have resulted in a relatively small decrease in lifespan. However, there is little evidence to bear on this question. Studies by Spix et al (1993) and Pope et al. (1992) yield conflicting evidence, suggesting that harvesting may represent anywhere from zero to 50

percent of the deaths estimated in short-term studies. A recent study by Zeger et al. (1999), that focused exclusively on this issue, reported that short-term harvesting may be a quite small fraction of mortality.[15]

It is not likely, however, that the excess mortality reported in a long-term prospective cohort study like Pope et al. (1995) contains any significant amount of this short-term harvesting. The Cox proportional hazard statistical model used in the Pope study examines the question of survivability throughout the study period (ten years). Deaths that are premature by only a few days or weeks within the ten-year study period (for example, the deaths of terminally ill patients, triggered by a short duration PM episode) are likely to have little impact on the calculation of the average probability of surviving the entire ten-year interval.

5.1.3 Estimating PM-Related Premature Mortality

The benefits analysis estimates $PM_{2.5}$ -related mortality using the C-R function estimated by Krewski et al. (2000). This study is a reanalysis of (Pope et al., 1995), which estimated the association between long-term (chronic) exposure to $PM_{2.5}$ and the survival of members of a large study population. Our decision to use Pope et al. (1995) in previous benefits analyses reflected the Science Advisory Board's explicit recommendation for modeling the mortality effects of PM in both the§812 Retrospective Report to Congress and the §812 Prospective Report (U.S. EPA, 1999a, p. 12). An advantage of Krewski et al. (2000) over Pope et al. (1995) is that Krewski et al.'s (2000) reanalysis of the Pope data uses the annual mean $PM_{2.5}$ concentration rather than the annual median. Because the mean is more readily affected by high PM values than is the median, if high PM days are actually important in causing premature mortality, the annual mean may be a preferable measure of long-term exposure than the median. However, estimates of annual mean levels are inherently less stable than annual median estimates, and are more sensitive to the estimates on the highly polluted days. Specifically, we use the Krewski results (Table 31, Krewski et al. (2000)) based on dichotomous samplers in 63 cities (rather than the 50 cities used in the Pope et al. $PM_{2.5}$ analysis)

The Krewski et al. (2000) long-term study is selected for use in the benefits analysis instead of short-term (daily pollution) studies for a number of reasons. It is used alone– rather than considering the total effect to be the sum of estimated short-term and long-term effects– because summing creates the possibility of double-counting a portion of PM-related mortality. The Krewski et al. study and the Pope study it reanalyzes are considered preferable to other available long-term studies because they use better statistical methods, have a much larger sample size, and more locations (63 cities) in the United States, than other studies. We also consider the Krewski study preferable to the original Pope et al. (1995) study because it uses the annual mean $PM_{2.5}$ rather than the median, which makes it more compatible with results from the S-R Matrix air quality model.

It is unlikely that the Krewski et al. study contains any significant amount of short-term harvesting. First, the health status of each individual tracked in the study is known at the beginning of the study period. Persons with known pre-existing serious illnesses were excluded from the study population. Second, the statistical model used in the Krewski and Pope studies examines the question of survivability throughout the study period (ten years). Deaths that are premature by only a few days or weeks within the ten-year study period (for example, the deaths of terminally ill patients, triggered by a short duration PM episode) are likely to have little impact on the calculation of the average probability of surviving the entire ten year interval. In

[15]Zeger et al. (1999, p. 171) reported that: "The TSP-mortality association in Philadelphia is inconsistent with the harvesting-only hypothesis, and the harvesting-resistant estimates of the TSP relative risk are actually larger – not smaller – than the ordinary estimates."

relation to the "Six-cities" study by Dockery et al. (1993), both the Krewski et al. study and the Pope et al. study found smaller increases in excess mortality for a given PM air quality change.

It is currently unknown whether there is a time lag (a delay between changes in PM exposures and changes in mortality rates) in the chronic PM/premature mortality relationship. The existence of such a lag is important for the valuation of premature mortality incidences because economic theory suggests that benefits occurring in the future should be discounted. Although there is no specific scientific evidence of the existence or structure of a PM effects lag, current scientific literature on adverse health effects, such as those associated with PM (e.g., smoking related disease) and the difference in the effect size between chronic exposure studies and daily mortality studies suggest that it is likely that not all incidences of premature mortality reduction associated with a given incremental change in PM exposure would occur in the same year as the exposure reduction. This same smoking-related literature implies that lags of up to a few years are plausible. Following explicit advice from the SAB, we assume a five-year lag structure, with 25 percent of premature deaths occurring in the first year, another 25 percent in the second year, and 16.7 percent in each of the remaining three years (EPA-SAB-COUNCIL-ADV-00-001, 1999). It should be noted that the selection of a five-year lag structure is not directly supported by any PM-specific literature. Rather, it is intended to be a best guess at the appropriate distribution of avoided incidences of PM-related mortality.

Alternative Calculations: PM-Related Mortality Based on the Original Pope et al. (1995) and on a Reanalysis of Pope et al. (1995) using the Annual Median PM concentration[16]

Although we believe that the annual mean PM is probably a superior measure of long-term PM exposure, for purposes of comparison we calculated PM-related mortality based on two studies which used the annual median PM: (1) the original study by Pope et al. (1995) and (2) Krewski et al. (2000), a reanalysis of Pope et al. (1995), also using the annual median PM.[17]

Alternative Calculation: PM-Related Mortality Based on Dockery et al. (1993)

As an alternative to Pope et al. (1995), this analysis calculates the impact of PM on mortality using Dockery et al. (1993), another long-term PM-mortality study. Dockery et al. (1993) examined the relationship between PM exposure and mortality in a cohort of 8,111 individuals aged 25 and older, living in six U.S. cities. They surveyed these individuals in 1974-1977 and followed their health status until 1991. While they used a smaller sample of individuals from fewer cities than the study by Pope et al., they used improved exposure estimates, a slightly broader study population (adults aged 25 and older), and a follow-up period nearly twice as long as that of Pope et al. (1995). Perhaps because of these differences, Dockery et al. (1993) found a larger effect of PM on premature mortality than that found by Pope et al.

[16] The annual median is the median value of the daily average PM concentrations over the entire four-year period that characterizes PM levels in each of the 51 cities in the Pope et al. (1995) study.

[17] Krewski et al. (2000) reanalyzed Pope et al. (1995) both using the annual median PM concentration and the annual mean PM concentration. The latter reanalysis, using the mean, is used in our primary analysis.

5.1.4 Valuing Premature Mortality

The "statistical lives lost" approach to valuing premature mortality estimates the value of a statistical death to be $6.12 million (in 1999 $). We assume for this analysis that some of the incidences of premature mortality related to PM exposures occur in a distributed fashion over the five years following exposure (the five-year mortality lag). To take this into account in the valuation of reductions in premature mortalities, we apply an annual five percent discount rate to the value of premature mortalities occurring in future years.[18]

Statistical Lives Lost

The "statistical lives lost" value of $6.12 million represents an intermediate value from a variety of estimates that appear in the economics literature, and is a value that EPA has frequently used. This estimate is the mean of a distribution fitted to the estimates from 26 value-of-life studies identified in the §812 study as "applicable to policy analysis." The approach and set of selected studies mirrors that of Viscusi (1992) (with the addition of two studies), and uses the same criteria used by Viscusi in his review of value-of-life studies. The $6.12 million estimate is consistent with Viscusi's conclusion (updated to 1999 $) that "most of the reasonable estimates of the value of life are clustered in the $3.84 to $8.93 million range." Uncertainty associated with the valuation of premature mortality is expressed through a Weibull distribution with a standard deviation of $4.13 million (IEc 1992, p. 2).

Five of the 26 studies are contingent valuation (CV) studies, which directly solicit WTP information from subjects; the rest are wage-risk studies, which base WTP estimates on estimates of the additional compensation demanded in the labor market for riskier jobs. The 26 studies are listed in Exhibit 5-4. The references for all but Gegax et al. (1985) and V.K. Smith (1983) may be found in Viscusi (1992). Although each of the studies estimated the mean WTP (MWTP) for a given reduction in mortality risk, the amounts of reduction in risk being valued were not necessarily the same across studies, nor were they necessarily the same as the amounts of reduction in mortality risk that would actually be conferred by a given reduction in ambient concentrations. The transferability of estimates of the value of a statistical life from the 26 studies to this analysis rests on the assumption that, within a reasonable range, WTP for reductions in mortality risk is linear in risk reduction, or equivalently, that the marginal willingness to pay curve is horizontal within a reasonable range. For example, suppose a study estimates that the average WTP for a reduction in mortality risk of 1/100,000 is $30. Suppose, however, that the actual mortality risk reduction resulting from a given air quality improvement is 1/10,000. If WTP for reductions in mortality risk is linear in risk reduction, then a WTP of $30 for a reduction of 1/100,000 implies a WTP of $300 for a risk reduction of 1/10,000 (which is ten times the risk reduction valued in the study). Under the assumption of linearity, the estimate of the value of a statistical life does not depend on the particular amount of risk reduction being valued.

[18]The choice of a five percent discount rate is based on the technical recommendation of the SAB for computing the value of a statistical life-year (U.S. EPA, 1999c, p. 14).

Exhibit 5-4 Summary of Mortality Valuation Estimates

Study	Type of Estimate	Valuation (millions 1999 $)
Kneisner and Leeth (1991) (US)	Labor Market	0.7
Smith and Gilbert (1984)	Labor Market	0.9
Dillingham (1985)	Labor Market	1.1
Butler (1983)	Labor Market	1.5
Miller and Guria (1991)	Contingent Valuation	1.6
Moore and Viscusi (1988)	Labor Market	3.2
Viscusi et al. (1991)	Contingent Valuation	3.4
Gegax et al. (1985; 1991)	Contingent Valuation	4.3
Marin and Psacharopoulos (1982)	Labor Market	3.5
Kneisner and Leeth (1991) (Australia)	Labor Market	4.3
Gerking et al. (1988)	Contingent Valuation	4.4
Cousineau et al. (1988; 1992)	Labor Market	4.6
Jones-Lee (1989)	Contingent Valuation	4.9
Dillingham (1985)	Labor Market	5.1
Viscusi (1978; 1979)	Labor Market	5.2
R.S. Smith (1976)	Labor Market	5.8
V.K. Smith (1983)	Labor Market	6.0
Olson (1981)	Labor Market	6.6
Viscusi (1981)	Labor Market	8.3
R.S. Smith (1974)	Labor Market	9.1
Moore and Viscusi (1988)	Labor Market	9.3
Kneisner and Leeth (1991) (Japan)	Labor Market	9.7
Herzog and Schlottman (1987; 1990)	Labor Market	11.6
Leigh and Folson (1984)	Labor Market	12.4
Leigh (1987)	Labor Market	13.3
Garen (1988)	Labor Market	17.2

Source: Viscusi (1992, Table 4.1).

Although the particular amount of mortality risk reduction being valued in a study may not affect the transferability of the WTP estimate from the study to this analysis, the characteristics of the study subjects and the nature of the mortality risk being valued in the study could be important. Certain characteristics of both the population affected and the mortality risk facing that population are believed to affect the MWTP to reduce the risk. The appropriateness of the MWTP estimates from the 26 studies for valuing the mortality-related benefits of reductions in ambient air 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 (1) the extent to which the subjects in the studies are similar to the population affected by changes in ambient air concentrations and (2) the extent to which the risks being valued are similar.

Focusing on the wage-risk studies, which make up the substantial majority of the 26 studies relied upon, the likely differences between (1) the subjects in these studies and the population affected by changes in air concentrations and (2) the nature of the mortality risks being valued in these studies and the nature of air pollution-related mortality risk are considered. The direction of bias in which each difference is likely to result is also considered.

Compared with the subjects in wage-risk studies, the population believed to be most affected by air pollution (i.e., the population that would receive the greatest mortality risk reduction associated with a given reduction in air concentrations) is, on average, older and probably more risk averse. For example, citing Schwartz and Dockery (1992) and Ostro et al. (1996), Chestnut (1995) estimated that approximately 85 percent of those who die prematurely from ambient air pollution-related causes are over 65. The average age of subjects in wage-risk studies, in contrast, is well under 65.

There is also reason to believe that those over 65 are, in general, more risk averse than the general population while workers in wage-risk studies are likely to be less risk averse than the general population. Although Viscusi's (1992) list of recommended studies excludes studies that consider only much-higher-than-average occupational risks, there is nevertheless likely to be some selection bias in the remaining studies -- that is, these studies are likely to be based on samples of workers who are, on average, more risk-loving than the general population. In contrast, older people as a group exhibit more risk averse behavior.

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

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

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

There is substantial evidence that the income elasticity of WTP for health risk reductions is positive (Loehman and De, 1982; Jones-Lee et al., 1985; Mitchell and Carson, 1986; Gerking et al., 1988; Alberini et al., 1997), although there is uncertainty about the exact value of this elasticity). Individuals with higher incomes (or greater wealth) should, then, be willing to pay more to reduce risk, all else equal, than individuals with lower incomes or wealth. Whether the average income or level of wealth of the population affected by ambient air pollution reductions is likely to be significantly different from that of subjects in wage-risk studies, however, is unclear.

Finally, although there may be several ways in which job-related mortality risks differ from air pollution-related mortality risks, the most important difference may be that job-related risks are incurred voluntarily whereas air pollution-related risks are incurred involuntarily.

There is some evidence that people will pay more to reduce involuntarily incurred risks than risks incurred voluntarily (e.g., Violette and Chestnut, 1983). Job-related risks are incurred voluntarily whereas air pollution-related risks are incurred involuntarily. If this is the case, WTP estimates based on wage-risk studies may be downward biased estimates of WTP to reduce involuntarily incurred ambient air pollution-related mortality risks.

The potential sources of bias in an estimate of MWTP to reduce the risk of air pollution related mortality based on wage-risk studies are summarized in Exhibit 5-5. Although most of the individual factors tend to have a downward bias, the overall effect of these biases is unclear.

Exhibit 5-5 Potential Sources of Bias in Estimates of Mean WTP to Reduce the Risk of PM Related Mortality Based on Wage-Risk Studies

Factor	Likely Direction of Bias in Mean WTP Estimate
Age	Uncertain
Degree of Risk Aversion	Downward
Income	Downward, if the elderly affected are a random sample of the elderly. It is unclear, if the elderly affected are the poor elderly.
Risk Perception: Voluntary vs. Involuntary risk	Downward

5.2 CHRONIC ILLNESS

Onset of bronchitis has been associated with exposure to air pollutants. Three studies have linked the onset of chronic bronchitis in adults to particulate matter. These results are consistent with research that has found chronic exposure to pollutants leads to declining pulmonary functioning (Detels et al., 1991; Ackermann-Liebrich et al., 1997; Abbey et al., 1998).

5.2.1 Chronic Bronchitis

We estimate the changes in the number of new cases of PM-related chronic bronchitis using studies by Schwartz (1993) and Abbey et al. (1995b). The Schwartz study is somewhat older and uses a cross-sectional design; however, it is based on a national sample, unlike the Abbey et al. study which is based on a sample of California residents. The estimates from Schwartz (1993) and Abbey et al. (1995b) are pooled. A second study by Abbey et al. (1993) is based on the same sample population as Abbey et al. (1995b) but used TSP as the measure of particulate matter. Because the two Abbey et al. studies used the same population, but the more recent study used a preferable measure of particulate matter, we did not include the earlier study in our pooling. The two studies used in our pooled estimate are listed in Exhibit 5-6.

Exhibit 5-6 Chronic Bronchitis Studies

Location	Study	Pollutants Used in Final Model	Age of Study Population
California	Abbey et al. (1995b)	$PM_{2.5}$	>26
United States	Schwartz (1993)	PM_{10}	>29

Schwartz (1993) examined survey data collected from 3,874 adults ranging in age from 30 to 74, and living in 53 urban areas in the U.S. The survey was conducted between 1971 and 1975, as part of the National Health and Nutrition Examination Survey, and is representative of the non-institutionalized U.S. population. Schwartz (1993, Table 3) reported chronic bronchitis prevalence rates in the study population by age, race, and gender. Non-white males under 52 years old had the lowest rate (1.7%) and white males 52 years and older had the highest rate (9.3%). The study examined the relationship between the prevalence of reported chronic bronchitis and annual levels of total suspended particulates (TSP), collected in the year prior to the survey.

The study by Abbey et al. (1995b) examined the relationship between estimated $PM_{2.5}$ (annual mean from 1966 to 1977), PM_{10} (annual mean from 1973 to 1977) and TSP (annual mean from 1973 to 1977) and the same chronic respiratory symptoms in a sample population of 1,868 Californian Seventh-Day Adventists. The initial survey was conducted in 1977 and the final survey in 1987. To ensure a better estimate of exposure, the study participants had to have been living in the same area for an extended period of time. In single-pollutant models, there was a statistically significant $PM_{2.5}$ relationship with development of chronic bronchitis, but not for airway obstructive disease (AOD) or asthma; PM_{10} was significantly associated with chronic bronchitis and AOD; and TSP was significantly associated with all cases of all three chronic symptoms. Other pollutants were not examined.

Valuing Chronic Bronchitis

PM-related chronic bronchitis is expected to last from the initial onset of the illness throughout the rest of the individual's life. WTP to avoid chronic bronchitis would therefore be expected to incorporate the present discounted value of a potentially long stream of costs (e.g., medical expenditures and lost earnings) and pain and suffering associated with the illness. Two studies, Viscusi et al. (1991) and Krupnick and Cropper (1992), provide estimates of WTP to avoid a case of chronic bronchitis.

The Viscusi et al. (1991) and the Krupnick and Cropper (1992) studies were experimental studies intended to examine new methodologies for eliciting values for morbidity endpoints. Although these studies were not specifically designed for policy analysis, we believe the studies provide reasonable estimates of the WTP for chronic bronchitis. As with other contingent valuation studies, the reliability of the WTP estimates depends on the methods used to obtain the WTP values. The Viscusi et al. and the Krupnick and Cropper studies are broadly consistent with current contingent valuation practices, although specific attributes of the studies may not be.

The study by Viscusi et al. uses a sample that is larger and more representative of the general population than the study by Krupnick and Cropper (which selects people who have a relative with the disease). Thus, the valuation for the high-end estimate is based on the distribution of WTP responses from Viscusi et al. The WTP to avoid a case of pollution-related chronic bronchitis (CB) is derived by starting with the WTP to avoid a severe case of chronic bronchitis, as described by Viscusi et al. (1991), and adjusting it downward

to reflect (1) the decrease in severity of a case of pollution-related CB relative to the severe case described in the Viscusi et al. study, and (2) the elasticity of WTP with respect to severity reported in the Krupnick and Cropper study. Because elasticity is a marginal concept and because it is a function of severity (as estimated from Krupnick and Cropper, 1992), WTP adjustments were made incrementally, in one percent steps. A severe case of CB was assigned a severity level of 13 (following Krupnick and Cropper). The WTP for a one percent decrease in severity is given by:

$$WTP_{0.99sev} = WTP_{sev} \cdot (1 - 0.01 \cdot e) \ ,$$

where sev is the original severity level (which, at the start, is 13) and e is the elasticity of WTP with respect to severity. Based on the regression in Krupnick and Cropper (1992) (see below), the estimate of e is 0.18*sev. At the mean value of sev (6.47), e = 1.16. As severity decreases, however, the elasticity decreases. Using the regression coefficient of 0.18, the above equation can be rewritten as:

$$WTP_{0.99sev} = WTP_{sev} \cdot (1 - 0.01 \cdot 0.18sev) \ .$$

For a given WTP_{sev} and a given coefficient of sev (0.18), the WTP for a 50 percent reduction in severity can be obtained iteratively, starting with sev =13, as follows:

$$WTP_{12.87} = WTP_{0.99 \cdot 13} = WTP_{13} \cdot (1 - 0.01 \cdot 0.18 \cdot 13)$$

$$WTP_{12.74} = WTP_{0.99 \cdot 12.87} = WTP_{12.87} \cdot (1 - 0.01 \cdot 0.18 \cdot 12.87)$$

$$WTP_{12.61} = WTP_{0.99 \cdot 12.74} = WTP_{12.74} \cdot (1 - 0.01 \cdot 0.18 \cdot 12.74)$$

and so forth. This iterative procedure eventually yields $WTP_{6.5}$, or WTP to avoid a case of chronic bronchitis that is of "average" severity.

The derivation of the WTP to avoid a case of pollution-related chronic bronchitis is based on three components, each of which is uncertain: (1) the WTP to avoid a case of severe CB, as described in the Viscusi et al. (1991) study, (2) the severity level of an average pollution-related case of CB (relative to that of the case described by Viscusi et al.), and (3) the elasticity of WTP with respect to severity of the illness. Because of these three sources of uncertainty, the WTP is uncertain. Based on assumptions about the distributions of each of the three uncertain components, a distribution of WTP to avoid a pollution-related case of CB was derived by Monte Carlo methods. The mean of this distribution, which was about $319,000 ($331,000 in 1999$), is taken as the central tendency estimate of WTP to avoid a pollution-related case of CB. Each of the three underlying distributions is described briefly below.

1. The distribution of WTP to avoid a severe case of CB was based on the distribution of WTP responses in the Viscusi et al. (1991) study. Viscusi et al. derived respondents' implicit WTP to avoid a statistical case of chronic bronchitis from their WTP for a specified reduction in risk. The mean response implied a WTP of about $1,275,000 (1999 $)[19]; the median response implied a WTP of about $676,000 (1999 $). However, the extreme tails of distributions of WTP responses are usually considered unreliable. Because the mean is much more sensitive to extreme values, the median of WTP responses is often used rather than the mean. Viscusi et al. report not only the mean and median of their distribution of WTP responses, however, but the decile points as well. The distribution of reliable WTP responses from the Viscusi et al. study could therefore be approximated by a discrete uniform distribution giving a probability of 1/9 to each of the first nine decile points. This omits the first five and the last five percent of the responses (the extreme tails, considered unreliable). This trimmed distribution of WTP responses from the Viscusi et al. study was assumed to be the distribution of WTPs to avoid a severe case of CB. The mean of this distribution is about $918,000 (1999 $).

2. The distribution of the severity level of an average case of pollution-related CB was modeled as a triangular distribution centered at 6.5, with endpoints at 1.0 and 12.0. These severity levels are based on the severity levels used in Krupnick and Cropper (1992), which estimated the relationship between ln(WTP) and severity level, from which the elasticity is derived. The most severe case of CB in that study is assigned a severity level of 13. The mean of the triangular distribution is 6.5. This represents a 50 percent reduction in severity from a severe case.

3. The elasticity of WTP to avoid a case of CB with respect to the severity of that case of CB is a constant times the severity level. This constant was estimated by Krupnick and Cropper (1992) in the regression of ln(WTP) on severity, discussed above. This estimated constant (regression coefficient) is normally distributed with mean = 0.18 and standard deviation = 0.0669 (obtained from Krupnick and Cropper).

The distribution of WTP to avoid a case of pollution-related CB was generated by Monte Carlo methods, drawing from the three distributions described above. On each of 16,000 iterations (1) a value was selected from each distribution, and (2) a value for WTP was generated by the iterative procedure described above, in which the severity level was decreased by one percent on each iteration, and the corresponding WTP was derived. The mean of the resulting distribution of WTP to avoid a case of pollution-related CB was $331,000 (1999$).

This WTP estimate is reasonably consistent with full COI estimates derived for chronic bronchitis, using average annual lost earnings and average annual medical expenditures reported by Cropper and Krupnick (1990) Using a 5 percent discount rate and assuming that (1) lost earnings continue until age 65, (2) medical expenditures are incurred until death, and (3) life expectancy is unchanged by chronic bronchitis, the present discounted value of the stream of medical expenditures and lost earnings associated with an average case of chronic bronchitis is estimated to be about $113,000 for a 30 year old, about $109,000 for a 40 year old, about $100,000 for a 50 year old, and about $57,000 for a 60 year old. A WTP estimate would be expected to be greater than a full COI estimate, reflecting the willingness to pay to avoid the pain and suffering associated with the illness. The WTP estimate of $331,000 is from 2.9 times the full COI estimate (for 30 year olds) to 5.8 times the full COI estimate (for 60 year olds).

[19]There is an indication in the Viscusi et al. (1991) paper that the dollar values in the paper are in 1987 dollars. Under this assumption, the dollar values were converted to 1999 dollars.

5.3 HOSPITAL ADMISSIONS

We estimate the impact of ozone and PM on both respiratory and cardiovascular hospital admissions. In addition, we estimate the impact of these pollutants on emergency room visits for asthma. The respiratory and cardiovascular hospital admissions studies used in the primary analysis are listed in Exhibits 5-7 and 5-8, respectively. Appendix B provides details on each study. Although the benefits associated with respiratory and cardiovascular hospital admissions are estimated separately in the analysis, the methods used to estimate changes in incidence and to value those changes are the same for both broad categories of hospital admissions. The two categories of hospital admissions are therefore discussed together in this section.

Exhibit 5-7 Respiratory Hospital Admission Studies

Location	Study	Endpoints Estimated (ICD code)	Pollutants Used in Final Model	Age of Study Population
PM-Related Hospital Admissions				
Fourteen U.S. Cities*	Samet et al. (2000)	pneumonia (480-487); COPD (490-492, 494-6)	PM_{10}	>64
Seattle, WA	Sheppard et al. (1999)	asthma (493)	$PM_{2.5}$	<65

*Birmingham, Alabama; Boulder, Colorado; Canton, Ohio; Chicago, Illinois; Colorado Springs, Colorado; Detroit, Michigan; Minneapolis/St. Paul, Minnesota; Nashville, Tennessee; New Haven, Connecticut; Pittsburgh, Pennsylvania; Provo/Orem, Utah; Seattle, Washington; Spokane, Washington; and Youngstown, Ohio.

Exhibit 5-8 Cardiovascular Hospital Admission Study

Location	Study	Endpoints Estimated (ICD code)	Pollutants Used in Final Model	Age of Study Population
PM-Related Hospital Admissions				
Fourteen U.S. Cities*	Samet et al. (2000)	cardiovascular illness (390 - 429)	PM_{10}	>64

5.3.1 PM-Related Respiratory and Cardiovascular Hospital Admissions

Respiratory and cardiovascular hospital admissions are the two broad categories of hospital admissions that have been related to exposure to both PM and ozone. Several epidemiological studies have estimated C-R functions that included both PM and ozone. However, a recent study by the Health Effects Institute (HEI) (Samet et al., 2000) estimated separate models for PM_{10} and pneumonia, COPD and cardiovascular diseases in each of fourteen cities in the United States, as well as pooled estimates across these cities. The fourteen cities included in the HEI hospital admissions study are Birmingham, Alabama; Boulder, Colorado; Canton, Ohio; Chicago, Illinois; Colorado Springs, Colorado; Detroit, Michigan; Minneapolis/St. Paul, Minnesota;

Nashville, Tennessee; New Haven, Connecticut; Pittsburgh, Pennsylvania; Provo/Orem, Utah; Seattle, Washington; Spokane, Washington; and Youngstown, Ohio.

We believe the Samet et al. (2000) pooled estimates are preferable to previously estimated models for several reasons. First, the HEI models are distributed lag models that are designed to capture not only same-day effects of PM but the effects of PM on a series of days subsequent to exposure. This type of model therefore captures the full impact of PM on hospital admissions. Samet et al. (2000) note that because of serial correlation, the coefficients of the PM lags tend to be unstable (i.e., have large variances) in single-city models; however, the pooled estimates, based on all fourteen cities are more stable because they are based on much larger sample sizes. A second advantage of the HEI models is that they represent the PM effect across a range of cities in the United States. Although other studies have estimated C-R functions in various cities in the United States, many of these cities (e.g., Minneapolis/St. Paul, Birmingham, Detroit, Spokane, New Haven, and Seattle) are included in the HEI study, which is a more recent analysis of the PM-hospital admissions relationships in these cities.

Although the HEI models do not include other pollutants, they do investigate the impact of omitting other pollutants on the estimated PM effects on hospital admissions. The results of this investigation are shown graphically in Figures 33 and 34 of Samet et al. (2000). The study authors conclude that the omission of SO_2 and O_3 from the models had virtually no effect on the estimated PM effect in any of the three pooled estimates (for cardiovascular diseases, COPD, and pneumonia). While Figure 34 suggests that this is the case for CV diseases and pneumonia, the omission of ozone from the model appears to have resulted in a downward-biased estimate of the PM effect on hospital admissions for COPD. This suggests that using the HEI pooled estimate for COPD will tend to understate the PM effect.

The HEI study estimates separate C-R functions for pneumonia and COPD hospital admissions for people 65 years and older. In addition, another study by Sheppard et al. (1999) estimates a C-R function for asthma hospital admissions for people under 65. The results of these three non-overlapping PM-related respiratory C-R functions are aggregated using the relevant steps of a pooling procedure described below.

5.3.2 Valuing Respiratory and Cardiovascular Hospital Admissions

Society's WTP to avoid a hospital admission includes medical expenses, lost work productivity, the non-market costs of treating illness (i.e., air, water and solid waste pollution from hospitals and the pharmaceutical industry), and the pain and suffering of the affected individual as well as of that of relatives, friends, and associated caregivers.[20]

[20] Some people take action to avert the negative impacts of pollution. While the costs of successful averting behavior should be added to the sum of the health-endpoint-specific costs when estimating the total costs of pollution, these costs are not associated with any single health endpoint It is possible that in some cases the averting action was not successful, in which case it might be argued that the cost of the averting behavior should be added to the other costs listed (for example, it might be the case that an individual incurs the costs of averting behavior and in addition incurs the costs of the illness that the averting behavior was intended to avoid). Because averting behavior is generally not taken to avoid a particular health problem (such as a hospital admission for respiratory illness), but instead is taken to avoid the entire collection of adverse effects of pollution, it does not seem reasonable to ascribe the entire costs of averting behavior to any single health endpoint.

Because medical expenditures are to a significant extent shared by society, via medical insurance, Medicare, etc., the medical expenditures actually incurred by the individual are likely to be less than the total medical cost to society. The total value to society of an individual's avoidance of hospital admission, then, might be thought of as having two components: (1) the cost of illness (COI) to society, including the total medical costs plus the value of the lost productivity, as well as (2) the WTP of the individual, as well as that of others, to avoid the pain and suffering resulting from the illness.

In the absence of estimates of social WTP to avoid hospital admissions for specific illnesses (components 1 plus 2 above), estimates of total COI (component 1) are typically used as conservative (lower bound) estimates. Because these estimates do not include the value of avoiding the pain and suffering resulting from the illness (component 2), they are biased downward. Some analyses adjust COI estimates upward by multiplying by an estimate of the ratio of WTP to COI, to better approximate total WTP. Other analyses have avoided making this adjustment because of the possibility of over-adjusting -- that is, possibly replacing a known downward bias with an upward bias. The COI values used in this benefits analysis will not be adjusted to better reflect the total WTP.

Following the method used in the §812 analysis (U.S. EPA, 1999b), ICD-code-specific COI estimates used in our analysis consist of two components: estimated hospital charges and the estimated opportunity cost of time spent in the hospital (based on the average length of a hospital stay for the illness). The opportunity cost of a day spent in the hospital is estimated as the value of the lost daily wage, regardless of whether or not the individual is in the workforce. This is estimated at $106 (U.S. Bureau of the Census, 1992).

For all hospital admissions included in this analysis, estimates of hospital charges and lengths of hospital stays were based on discharge statistics provided by Elixhauser et al. (1993). The total COI for an ICD-code-specific hospital stay lasting n days, then, would be estimated as the mean hospital charge plus $106*n. Most respiratory hospital admissions categories considered in epidemiological studies consisted of sets of ICD codes. The unit dollar value for the set of ICD codes was estimated as the weighted average of the ICD-code-specific mean hospital charges of each ICD code in the set. The weights were the relative frequencies of the ICD codes among hospital discharges in the United States, as estimated by the National Hospital Discharge Survey [Owings, 1999 #1872]. The study-specific values for valuing respiratory and cardiovascular hospital admissions are shown in Exhibits 5-9 and 5-10, respectively.

The mean hospital charges and mean lengths of stay provided by Elixhauser et al. (1993) are based on a very large nationally representative sample of about seven million hospital discharges, and are therefore the best estimates of mean hospital charges and mean lengths of stay available, with negligible standard errors. However, because of distortions in the market for medical services, the hospital charge may exceed "the cost of a hospital stay." We use the example of a hospital visit to illustrate the problem. Suppose a patient is admitted to the hospital to be treated for an asthma episode. The patient's stay in the hospital (including the treatments received) costs the hospital a certain amount. This is the hospital cost – i.e., the short-term expenditures of the hospital to provide the medical services that were provided to the patient during his hospital stay. The hospital then charges the payer a certain amount – the hospital charge. If the hospital wants to make a profit, is trying to cover costs that are not associated with any one particular patient admission (e.g., uninsured patient services), and/or has capital expenses (building expansion or renovation) or other long term costs, it may charge an amount that exceeds the patient-specific short term costs of providing services. The payer (e.g., the health maintenance organization or other health insurer) pays the hospital a certain amount – the payment – for the services provided to the patient. The less incentive the payer has to keep costs down, the closer the payment will be to the charge. If, however, the payer has an incentive to keep costs down, the payment may be substantially less than the charge; it may still, however, exceed the short-term cost for services to the individual patient.

Although the hospital charge may exceed the short-term cost to the hospital of providing the medical services required during a patient's hospital stay, cost of illness estimates based on hospital charges are still likely to understate the total social WTP to avoid the hospitalization in the first place, because the omitted WTP to avoid the pain and suffering is likely to be quite large.

Exhibit 5-9 Unit Values for Respiratory Hospital Admissions*

Location	Study	Endpoints Estimated (ICD code)	Age of Study Population	COI [a] (1999 $)
PM-Related Hospital Admissions				
Fourteen U.S. Cities	Samet et al. (2000)	pneumonia (480-487)	>64	$14,693
		COPD (490-492, 494-6)		$12,378
Seattle, WA	Sheppard et al. (1999)	asthma (493)	<65	$6,634

* The unit value for a group of ICD-9 codes is the weighted average of ICD-9 code-specific values, from Elixhauser et al. (1993). The weights are the relative frequencies of hospital discharges in Elixhauser et al. for each ICD-9 code in the group. The monetized benefits of non-overlapping endpoints within each study were aggregated. Monetized benefits for asthma among people age <65 (Sheppard et al., 1999) were aggregated with the monetized benefits in Samet et al. (2000) of people age >64.

Exhibit 5-10 Unit Values for Cardiovascular Hospital Admissions*

Location	Study	Endpoints Estimated (ICD code)	Age of Study Population	COI [a] (1999 $)
PM-Related Hospital Admissions				
Fourteen U.S. Cities	Samet et al. (2000)	cardiovascular illness (390 - 429)	>64	$18,387

* The unit value for a group of ICD-9 codes is the weighted average of ICD-9 code-specific values, from Elixhauser et al. (1993). The weights are the relative frequencies of hospital discharges in Elixhauser et al. for each ICD-9 code in the group.

We were not able to estimate the uncertainty surrounding cost-of-illness estimates for hospital admissions because 1993 was the last year for which standard errors of estimates of mean hospital charges were reported . However, the standard errors reported in 1993 were very small because estimates of mean hospital charges were based on large sample sizes, and the overall sample size in 1997 was about ten times as large as that in 1993 (at about seven million hospital discharges in all). The standard errors of the current estimates of mean hospital charges will therefore be negligible. Therefore, although we cannot include the uncertainty surrounding these cost-of-illness estimates in our overall uncertainty analysis, the omission of this component of uncertainty will have virtually no impact on the overall characterization of uncertainty.

5.3.3 Asthma-Related Emergency Room (ER) Visits

We use one C-R function to estimate the effects of PM exposure to asthma-related ER visits. In a study of Seattle residents, Schwartz et al. (1993) found PM_{10} to be significantly related to asthma-related ER visits.

Because we are estimating ER visits as well as hospital admissions for asthma, we must avoid counting twice the ER visits for asthma that are subsequently admitted to the hospital. To avoid double-counting, the baseline incidence rate for emergency room visits is adjusted by subtracting the percentage of patients that are admitted into the hospital. Three studies provide some information to do this: Richards et al. (1981, p. 350) reported that 13% of children's ER visits ended up as hospital admissions; Lipfert (1993, p. 230) reported that ER visits (for all causes) are two to five times more frequent than hospital admissions; Smith et al. (1997, p. 789) reported 445,000 asthma-related hospital admissions in 1987 and 1.2 million asthma ER visits. The study by Smith et al. seems the most relevant since it is a national study and looks at all age groups. Assuming that air-pollution related hospital admissions first pass through the ER, the reported incidence rates suggest that 37% (=445,000/1,200,000) of ER visits are subsequently admitted to the hospital, or that ER visits for asthma occur 2.7 times as frequently as hospital admissions for asthma. The baseline incidence of asthma ER visits is therefore taken to be 2.7 times the baseline incidence of hospital admissions for asthma. To avoid double-counting, however, only 63% of the resulting change in asthma ER visits associated with a given change in pollutant concentrations is counted in the ER visit incidence change.

Valuing Asthma-Related Emergency Room (ER) Visits

The value of an avoided asthma-related ER visit was based on national data reported in Smith et al. (1997). Smith et al. reported that there were approximately 1.2 million asthma-related ER visits made in 1987, at a total cost of $186.5 million, in 1987$. The average cost per visit was therefore $155 in 1987$, or $298.62 in 1999 $ (using the CPI-U for medical care to adjust to 1999 $). The uncertainty surrounding this estimate, based on the uncertainty surrounding the number of ER visits and the total cost of all visits reported by Smith et al. was characterized by a triangular distribution centered at $298.62, on the interval [$221.65, $414.07].

5.4 ACUTE ILLNESSES AND SYMPTOMS NOT REQUIRING HOSPITALIZATION

We consider in this section a number of acute symptoms that do not require hospitalization, such as acute bronchitis, and upper and lower respiratory symptoms. Several of these illnesses and symptoms were considered in the §812 Prospective analysis as well. The unit values and the uncertainty distributions for those acute illnesses and symptoms that were also considered in the §812 Prospective analysis were obtained by adjusting the unit values used in that analysis from 1990 $ to 1999 $ by multiplying by 1.275 (based on the CPI-U for “all items”).

Exhibit 5-11 Studies of Symptoms/Illnesses Not Requiring Hospitalization

Endpoint	Study	Pollutants	Study Population
Acute bronchitis	Dockery et al. (1996)	$PM_{2.5}$	Ages 8-12
Upper respiratory symptoms (URS)	Pope et al. (1991)	PM_{10}	Asthmatics, ages 9-11
Lower respiratory symptoms (LRS)	Schwartz et al. (1994)	$PM_{2.5}$	Ages 7-14
Minor restricted activity day (MRAD)	Ostro and Rothschild (1989),	$PM_{2.5}$	Ages 18-65
Asthma Attacks	Whittemore and Korn (1980)	PM_{10}	asthmatics, all ages
Work loss days (WLDs)	Ostro (1987)	$PM_{2.5}$	Ages 18-65

5.4.1 Acute Bronchitis

Dockery et al. (1996) examined the relationship between PM and other pollutants on the reported rates of asthma, persistent wheeze, chronic cough, and bronchitis, in a study of 13,369 children ages 8-12 living in 24 communities in the U.S. and Canada. Health data were collected in 1988-1991, and single-pollutant models were used in the analysis to test a number of measures of particulate air pollution. Dockery et al. found that annual level of sulfates and particle acidity were significantly related to bronchitis, and $PM_{2.5}$ and PM_{10} were marginally significantly related to bronchitis.

Valuing Acute Bronchitis

Estimating WTP to avoid a case of acute bronchitis is difficult for several reasons. First, WTP to avoid acute bronchitis itself has not been estimated. Estimation of WTP to avoid this health endpoint therefore must be based on estimates of WTP to avoid symptoms that occur with this illness. Second, a case of acute bronchitis may last more than one day, whereas it is a day of avoided symptoms that is typically valued. Finally, the C-R function used in the benefit analysis for acute bronchitis was estimated for children, whereas WTP estimates for those symptoms associated with acute bronchitis were obtained from adults.

With these caveats in mind, the values used for acute bronchitis in this analysis were obtained by adjusting the values used in the §812 Prospective analysis from 1990 $ to 1999 $ by multiplying by 1.275. WTP to avoid a case of acute bronchitis was estimated as the midpoint between a low estimate and a high estimate. The low estimate is the sum of the midrange values recommended by IEc (1994) for two symptoms believed to be associated with acute bronchitis: coughing and chest tightness. The high estimate was taken to be twice the value of a minor respiratory restricted activity day. The unit value is the midpoint between the low and high estimates. The low, high, and midpoint estimates used in the §812 Prospective analysis were $13, $77, and $45, respectively, in 1990 $. The corresponding values in 1999 $ are $16.58, $98.18, and $57.38, respectively.

5.4.2 Upper Respiratory Symptoms (URS)

Using logistic regression, Pope et al. (1991) estimated the impact of PM_{10} on the incidence of a variety of minor symptoms in 55 subjects (34 "school-based" and 21 "patient-based") living in the Utah Valley from December 1989 through March 1990. The children in the Pope et al. study were asked to record respiratory symptoms in a daily diary, and the daily occurrences of URS and LRS, as defined above, were related to daily PM_{10} concentrations. Pope et al. describe URS as consisting of one or more of the following symptoms: runny or stuffy nose; wet cough; and burning, aching, or red eyes. Levels of ozone, NO_2, and SO_2 were reported low during this period, and were not included in the analysis.

The sample in this study is relatively small and is most representative of the asthmatic population, rather than the general population. The school-based subjects (ranging in age from 9 to 11) were chosen based on "a positive response to one or more of three questions: ever wheezed without a cold, wheezed for 3 days or more out of the week for a month or longer, and/or had a doctor say the 'child has asthma' (Pope et al., 1991, p. 669)." The patient-based subjects (ranging in age from 8 to 72) were receiving treatment for asthma and were referred by local physicians. Regression results for the school-based sample (Pope et al., 1991, Table 5) show PM_{10} significantly associated with both upper and lower respiratory symptoms. The patient-based sample did not find a significant PM_{10} effect. The results from the school-based sample are used here.

Valuing URS

Willingness to pay to avoid a day of URS is based on symptom-specific WTPs to avoid those symptoms identified by Pope et al. as part of the URS complex of symptoms. Three contingent valuation (CV) studies have estimated WTP to avoid various morbidity symptoms that are either within the URS symptom complex defined by Pope et al. (1991) or are similar to those symptoms identified by Pope et al. In each CV study, participants were asked their WTP to avoid a day of each of several symptoms. The WTP estimates corresponding to the morbidity symptoms valued in each study are presented in Exhibit 5-12. The three individual symptoms listed in Exhibit 5-12 that were identified as most closely matching those listed by Pope, et al. for URS are cough, head/sinus congestion, and eye irritation, corresponding to "wet cough," "runny or stuffy nose," and "burning, aching or red eyes," respectively. A day of URS could consist of any one of the seven possible "symptom complexes" consisting of at least one of these three symptoms. Using the symptom symbols in Exhibit 5-12, these seven possible symptom complexes are presented in Exhibit 5-13. It is assumed that each of these seven URS complexes is equally likely.[21] The point estimate of MWTP to avoid an occurrence of URS is just an average of the seven estimates of MWTP for the different URS complexes – \$18.70, or about \$19 in 1990 \$. This is \$24.23 (=\$19*1.275) in 1999 \$. In the absence of information surrounding the frequency with which each of the seven types of URS occurs within the URS symptom complex, an uncertainty analysis for WTP to avoid a day of URS is based on a continuous uniform distribution of MWTPs in Exhibit 5-13, with a range of [\$7, \$33], or [\$8.93, \$42.08] in 1999 \$.

[21] With empirical evidence, we could presumably improve the accuracy of the probabilities of occurrence of each type of URS. Lacking empirical evidence, however, a uniform distribution seems the most reasonable "default" assumption.

Exhibit 5-12 Median WTP Estimates and Derived Midrange Estimates (in 1999 $)

Symptom [a]	Dickie et al. (1987)	Tolley et al. (1986)	Loehman et al. (1979)	Mid-Range Estimate
Throat congestion	4.81	20.84	-	12.75
Head/sinus congestion	5.61	22.45	10.45	12.75
Coughing	1.61	17.65	6.35	8.93
Eye irritation	-	20.03	-	20.03
Headache	1.61	32.07	-	12.75
Shortness of breath	0.00	-	13.47	6.37
Pain upon deep inhalation (PDI)	5.63	-	-	5.63
Wheeze	3.21	-	-	3.21
Coughing up phlegm	3.51 [b]	-	-	3.51
Chest tightness	8.03	-	-	8.03

[a] All estimates are WTP to avoid one day of symptom. Midrange estimates were derived by IEc (1993).

[b] 10% trimmed mean.

Exhibit 5-13 Estimates of MWTP to Avoid Upper Respiratory Symptoms (1999 $)

Symptom Combinations Identified as URS by Pope et al. (1991)	MWTP to Avoid Symptom(s)
Coughing	$8.93
Head/Sinus Congestion	$12.75
Eye Irritation	$20.03
Coughing, Head/Sinus Congestion	$21.67
Coughing, Eye Irritation	$28.96
Head/Sinus Congestion, Eye Irritation	$32.78
Coughing, Head/Sinus Congestion, Eye Irritation	$41.71
	Average: $23.83

Based on values reported in Exhibit 5-12.

It is worth emphasizing that what is being valued here is URS *as defined by Pope et al. (1991).* While other definitions of URS are certainly possible, this definition of URS is used in this benefit analysis because it is the incidence of this specific definition of URS that has been related to PM exposure by Pope et al.

5.4.3 Lower Respiratory Symptoms (LRS)

Schwartz et al. (1994) used logistic regression to link lower respiratory symptoms in children with SO_2, NO_2, ozone, PM_{10}, $PM_{2.5}$, sulfate and H^+ (hydrogen ion). Children were selected for the study if they were exposed to indoor sources of air pollution: gas stoves and parental smoking. The study enrolled 1,844 children into a year-long study that was conducted in different years (1984 to 1988) in six cities. The students were in grades two through five at the time of enrollment in 1984. By the completion of the final study, the cohort would then be in the eighth grade (ages 13-14); this suggests an age range of 7 to 14.

In single pollutant models SO_2, NO_2, $PM_{2.5}$, and PM_{10} were significantly linked to cough. In two-pollutant models, PM_{10} had the most consistent relationship with cough; ozone was marginally significant, controlling for PM_{10}. In models for upper respiratory symptoms, they reported a marginally significant association for PM_{10}. In models for lower respiratory symptoms, they reported significant single-pollutant models, using SO_2, O_3, $PM_{2.5}$, PM_{10}, SO_4, and H^+.

Valuing LRS

The method for deriving a point estimate of mean WTP to avoid a day of LRS is the same as for URS. Schwartz et al. (1994, p. 1235) define LRS as at least two of the following symptoms: cough, chest pain, phlegm, and wheeze. The symptoms for which WTP estimates are available that reasonably match those listed by Schwartz et al. for LRS are cough (C), chest tightness (CT), coughing up phlegm (CP), and wheeze (W). A day of LRS, as defined by Schwartz et al., could consist of any one of the 11 combinations of at least two of these four symptoms, as displayed in Exhibit 5-14.[22]

[22] Because cough is a symptom in some of the URS clusters as well as some of the LRS clusters, there is the possibility of a very small amount of double counting – if the same individual were to have an occurrence of URS which included cough and an occurrence of LRS which included cough *both on exactly the same day*. Because this is probably a very small probability occurrence, the degree of double counting is likely to be very minor. Moreover, because URS is applied only to asthmatics ages 9-11 (a very small population), the amount of potential double counting should be truly negligible.

Exhibit 5-14 Estimates of MWTP to Avoid Lower Respiratory Symptoms (1999 $)

Symptom Combinations Identified as LRS by Schwartz et al. (1994)	MWTP to Avoid Symptom(s)
Coughing, Chest Tightness	$16.95
Coughing, Coughing Up Phlegm	$12.42
Coughing, Wheeze	$12.13
Chest Tightness, Coughing Up Phlegm	$11.53
Chest Tightness, Wheeze	$11.24
Coughing Up Phlegm, Wheeze	$6.72
Coughing, Chest Tightness, Coughing Up Phlegm	$20.46
Coughing, Chest Tightness, Wheeze	$20.17
Coughing, Coughing Up Phlegm, Wheeze	$15.64
Chest Tightness, Coughing Up Phlegm, Wheeze	$14.75
Coughing, Chest Tightness, Coughing Up Phlegm, Wheeze	$23.67
	Average: $15.07

Based on values reported in Exhibit 5-12.

We assumed that each of the eleven types of LRS is equally likely.[23] The mean WTP to avoid a day of LRS as defined by Schwartz et al. (1994) is therefore the average of the mean WTPs to avoid each type of LRS, – $11.82. This is $15.07 (=1.275*$11.82) in 1999 $. This is the point estimate used in the benefit analysis for the dollar value for LRS as defined by Schwartz et al. The WTP estimates are based on studies which considered the value of a *day* of avoided symptoms, whereas the Schwartz et al. study used as its measure a *case* of LRS. Because a case of LRS usually lasts at least one day, and often more, WTP to avoid a day of LRS should be a conservative estimate of WTP to avoid a case of LRS.

In the absence of information about the frequency of each of the seven types of LRS among all occurrences of LRS, the uncertainty analysis for WTP to avoid a day of URS is based on a continuous uniform distribution of MWTPs in Exhibit 5-12, with a range of [$5, $19], or [$6.37, $24.22] in 1999 $. This is the same procedure as that used in the URS uncertainty analysis.

As with URS, it is worth emphasizing that what is being valued here is LRS *as defined by Schwartz et al. (1994)*. While other definitions of LRS are certainly possible, this definition of LRS is used in this benefit analysis because it is the incidence of this specific definition of LRS that has been related to PM exposure by Schwartz et al.

[23] As with URS, if we had empirical evidence we could improve the accuracy of the probabilities of occurrence of each type of LRS. Lacking empirical evidence, however, a uniform distribution seems the most reasonable "default" assumption.

Issues in the Valuation of URS and LRS

The point estimates derived for mean WTP to avoid a day of URS and a case of LRS are based on the assumption that WTPs are additive. For example, if WTP to avoid a day of cough is $8.93, and WTP to avoid a day of shortness of breath is $6.37, then WTP to avoid a day of both cough and shortness of breath is $15.30. If there are no synergistic effects among symptoms, then it is likely that the marginal utility of avoiding symptoms decreases with the number of symptoms being avoided. If this is the case, adding WTPs would tend to overestimate WTP for avoidance of multiple symptoms. However, there may be synergistic effects– that is, the discomfort from two or more simultaneous symptoms may exceed the sum of the discomforts associated with each of the individual symptoms. If this is the case, adding WTPs would tend to underestimate WTP for avoidance of multiple symptoms. It is also possible that people may experience additional symptoms for which WTPs are not available, again leading to an underestimate of the correct WTP. However, for small numbers of symptoms, the assumption of additivity of WTPs is unlikely to result in substantive bias.

There are also three sources of uncertainty in the valuation of both URS and LRS: (1) an occurrence of URS or of LRS may be comprised of one or more of a variety of symptoms (i.e., URS and LRS are each potentially a "complex of symptoms"), so that what is being valued may vary from one occurrence to another; (2) for a given symptom, there is uncertainty about the mean WTP to avoid the symptom; and (3) the WTP to avoid an occurrence of multiple symptoms may be greater or less than the sum of the WTPs to avoid the individual symptoms.

Information about the degree of uncertainty from either the second or the third source is not available. The first source of uncertainty, however, is addressed because an occurrence of URS or LRS may vary in symptoms. For example, seven different symptom complexes that qualify as URS, as defined by Pope et al. (1991), were identified above. The estimates of MWTP to avoid these seven different kinds of URS range from $8.93 (to avoid an occurrence of URS that consists of only coughing) to $42.06 (to avoid an occurrence of URS that consists of coughing plus head/sinus congestion plus eye irritation). There is no information, however, about the frequency of each of the seven types of URS among all occurrences of URS.

Because of insufficient information to adequately estimate the distributions of the estimators of MWTP for URS and LRS, as a rough approximation, a continuous uniform distribution over the interval from the smallest point estimate to the largest is used. As was mentioned in the two previous sections, the interval for URS is [$8.93, $42.06], and for LRS, the interval is [$6.37, $24.22].

Alternatively, a discrete distribution of the seven unit dollar values associated with each of the seven types of URS identified could be used. This would provide a distribution whose mean is the same as the point estimate of MWTP. A continuous uniform distribution, however, is probably more reasonable than a discrete uniform distribution. The differences between the means of the discrete uniform distributions (the point estimates) and the means of the continuous uniform distributions are relatively small, as shown in Exhibit 5-15.

Exhibit 5-15 Comparison of the Means of Discrete and Continuous Uniform Distributions of MWTP Associated with URS and LRS (1990 $)

Health Endpoint	Mean of Discrete Uniform Distribution (Point Est.)	Mean of Continuous Uniform Distribution
URS (Pope et al., 1991)	18.70	19.86
LRS (Schwartz et al., 1994)	11.82	11.92

5.4.4 Minor Restricted Activity Days (MRADs)

Ostro and Rothschild (1989) estimated the impact of $PM_{2.5}$ on the incidence of minor restricted activity days (MRAD) in a national sample of the adult working population, ages 18 to 65, living in metropolitan areas. We developed separate coefficients for each year in the analysis (1976-1981), which were then combined for use in this analysis. The coefficient used in the C-R function is a weighted average of the coefficients in Ostro (Ostro, 1987, Table IV) using the inverse of the variance as the weight.

Valuing Minor Restricted Activity Days (MRADs)

The unit value and uncertainty distribution for MRADs for this analysis were obtained by adjusting the (rounded) values in 1990 $ used in the §812 Prospective analysis to 1999 $ by multiplying by 1.275. No studies are reported to have estimated WTP to avoid a minor restricted activity day (MRAD). However, IEc (1993) has derived an estimate of WTP to avoid a minor respiratory restricted activity day (MRRAD), using WTP estimates from Tolley et al. (1986) for avoiding a three-symptom combination of coughing, throat congestion, and sinusitis. This estimate of WTP to avoid a MRRAD, so defined, is $38.37 (1990 $), or about $38. Although Ostro and Rothschild (1989) estimated the relationship between $PM_{2.5}$ and MRADs, rather than MRRADs (a component of MRADs), it is likely that most of the MRADs associated with exposure to $PM_{2.5}$ are in fact MRRADs. For the purpose of valuing this health endpoint, then, we assumed that MRADs associated with PM exposure may be more specifically defined as MRRADs, and therefore used the estimate of mean WTP to avoid a MRRAD.

Any estimate of mean WTP to avoid a MRRAD (or any other type of restricted activity day other than WLD) will be somewhat arbitrary because the endpoint itself is not precisely defined. Many different combinations of symptoms could presumably result in some minor or less minor restriction in activity. Krupnick and Kopp (1988) argued that mild symptoms will not be sufficient to result in a MRRAD, so that WTP to avoid a MRRAD should exceed WTP to avoid any single mild symptom. A single severe symptom or a combination of symptoms could, however, be sufficient to restrict activity. Therefore WTP to avoid a MRRAD should, these authors argue, not necessarily exceed WTP to avoid a single severe symptom or a combination of symptoms. The "severity" of a symptom, however, is similarly not precisely defined; moreover, one level of severity of a symptom could induce restriction of activity for one individual while not doing so for another. The same is true for any particular combination of symptoms.

Given that there is inherently a substantial degree of arbitrariness in any point estimate of WTP to avoid a MRRAD (or other kinds of restricted activity days), the reasonable bounds on such an estimate must be considered. By definition, a MRRAD does not result in loss of work. WTP to avoid a MRRAD should therefore be less than WTP to avoid a WLD. At the other extreme, WTP to avoid a MRRAD should exceed

WTP to avoid a single mild symptom. The highest IEc midrange estimate of WTP to avoid a single symptom is $15.72 (1990 $), or about $16, for eye irritation. The point estimate of WTP to avoid a WLD in the benefit analysis is $83 (1990 $). If all the single symptoms evaluated by the studies are not severe, then the estimate of WTP to avoid a MRRAD should be somewhere between $16 and $83. Because the IEc estimate of $38 falls within this range (and acknowledging the degree of arbitrariness associated with any estimate within this range), the IEc estimate is used as the mean of a triangular distribution centered at $38, ranging from $16 to $61. Adjusting to 1999 $, this is a triangular distribution centered at $48.43, ranging from $20.34 to $77.76.

5.4.5 Asthma Attacks

Whittemore and Korn (1980) examined the relationship between air pollution and asthma attacks in a survey of 443 children and adults, living in six communities in southern California during three 34-week periods in 1972-1975. The analysis focused on TSP and ozone. Respirable PM, NO_2, SO_2 were highly correlated with TSP and excluded from the analysis. In a two pollutant model, daily levels of both TSP and O_x were significantly related to reported asthma attacks. The value of an asthma attack is assumed to be the same as for a day in which asthma is moderate or worse.

Valuing Asthma Attacks

The value of avoiding an asthma attack is estimated as the mean of four WTP estimates obtained in a study by Rowe and Chestnut (1986). The four WTP estimates correspond to four severity definitions of a "bad asthma day." The mean of the four average WTPs is $32 (1990 $), or $40.79 in 1999 $. The uncertainty surrounding this estimate was characterized by a continuous uniform distribution on the range defined by the lowest and highest of the four average WTP estimates from Rowe and Chestnut, [$12, $54], or [$15.30, $68.83] in 1999 $.

5.4.6 Work Loss Days (WLD)

Ostro (1987) estimated the impact of $PM_{2.5}$ on the incidence of work-loss days (WLDs), restricted activity days (RADs), and respiratory-related RADs (RRADs) in a national sample of the adult working population, ages 18 to 65, living in metropolitan areas. The annual national survey results used in this analysis were conducted in 1976-1981. Ostro reported that two-week average $PM_{2.5}$ levels were significantly linked to work-loss days, RADs, and RRADs, however there was some year-to-year variability in the results. Separate coefficients were developed for each year in the analysis (1976-1981); these coefficients were pooled. The coefficient used in the concentration-response function used here is a weighted average of the coefficients in Ostro (1987, Table III) using the inverse of the variance as the weight.

Valuing WLD

Willingness to pay to avoid the loss of one day of work was estimated by dividing the median weekly wage for 1990 (U.S. Bureau of the Census, 1992) by five (to get the median daily wage). This values the loss of a day of work at the national median wage for the day lost. To account for regional variations in median wages, the national daily median wage was adjusted on a county-by-county basis using a factor based on the ratio of national median household income divided by each county's median income. Each county's income-adjusted willingness to pay to avoid the loss of one day of work was then used to value the number of work loss

days attributed to that county. Valuing the loss of a day's work at the wages lost is consistent with economic theory, which assumes that an individual is paid exactly the value of his labor.

The use of the median rather than the mean, however, requires some comment. If all individuals in society were equally likely to be affected by air pollution to the extent that they lose a day of work because of it, then the appropriate measure of the value of a work loss day would be the mean daily wage. It is highly likely, however, that the loss of work days due to pollution exposure does not occur with equal probability among all individuals, but instead is more likely to occur among lower income individuals than among high income individuals. It is probable, for example, that individuals who are vulnerable enough to the negative effects of air pollution to lose a day of work as a result of exposure tend to be those with generally poorer health care. Individuals with poorer health care have, on average, lower incomes. To estimate the average lost wages of individuals who lose a day of work because of exposure to PM pollution, then, would require a weighted average of all daily wages, with higher weights on the low end of the wage scale and lower weights on the high end of the wage scale. Because the appropriate weights are not known, however, the median wage was used rather than the mean wage. The median is more likely to approximate the correct value than the mean because means are highly susceptible to the influence of large values in the tail of a distribution (in this case, the small percentage of very large incomes in the United States), whereas the median is not susceptible to these large values. The median daily wage in 1990 was $83, or $105.8 in 1999 $. This is the value that was used to represent work loss days (WLD). An uncertainty distribution for this endpoint was unavailable, therefore the same central estimate ($105.8) was used to value incidence changes at the fifth, mean, and ninety-fifth percentiles.

6. RESULTS

This chapter provides estimates of the magnitude and value of changes in adverse health effects associated with the different policy scenarios that we considered.

To place estimated incidence changes into context with predicted baseline incidence, Exhibit 6-1 displays the baseline incidence figures for those endpoints for which one can be calculated. Due to the nature of the endpoints, baseline incidence can only be calculated for PM-related health effects. In addition to baseline incidence, for each health effect, both the mean estimated incidence change and corresponding percent change between post-control incidence reductions and the predicted incidence baseline is presented. We calculated baseline incidence and the corresponding percentage changes for both national air quality changes.

Exhibits 6-2 and 6-3 present the 5^{th} percentile, mean, and 95^{th} percentile estimate for the incidence and benefit estimates for each endpoint and for the total. Exhibits 6-4 and 6-5 present the weights we used to pool the chronic bronchitis studies. Exhibit 6-6 presents several alternative mortality estimates. Exhibits 6-7 and 6-8 present state-level estimates for the "75 Percent Reduction" and the "All Power Plant" scenarios. Finally, Exhibits 6-9 and 6-10 present MSA-level estimates for the "75 Percent Reduction" and the "All Power Plant" scenarios.

Exhibit 6-1 PM-Related Health Effects as a Percentage of Health Effects Due to All Causes

		"75 Percent Reduction" Scenario		"Power Plant" Scenario	
Endpoint	**Reference**	**Mean**	**% of Baseline**	**Mean**	**% of Baseline**
Ages 30+	Krewski et al. (2000)	18,700	0.8%	30,100	1.3%
Chronic Bronchitis	Pooled Analysis	11,400	1.8%	18,600	3.0%
COPD-Related Hospital Admissions	Samet et al. (2000)	2,000	0.5%	3,320	1.4%
Pneumonia-Related Hospital Admissions	Samet et al. (2000)	2,440	0.3%	4,040	0.8%
Asthma-Related Hospital Admissions	Sheppard et al. (1999)	1,860	0.4%	3,020	1.1%
Cardiovascular-Related Hospital Admissions	Samet et al. (2000)	5,880	0.2%	9,720	0.4%
Asthma-Related ER Visits	Schwartz et al. (1993)	4,320	0.6%	7,160	1.6%
Acute Bronchitis	Dockery et al. (1996)	37,100	4.1%	59,000	12.8%
Upper Respiratory Symptoms	Pope et al. (1991)	412,000	0.4%	679,000	1.0%
Lower Respiratory Symptoms	Schwartz et al. (1994)	397,000	2.8%	630,000	6.6%
Asthma Attacks	Whittemore and Korn (1980)	366,000	0.2%	603,000	0.6%
Work Loss Days	Ostro (1987)	3,190,000	0.7%	5,130,000	1.3%
MRAD (adjusted for Asthma Attacks)	Ostro and Rothschild (1989)	16,400,000	1.3%	26,300,000	2.4%

Exhibit 6-2 Estimated PM-Related Health Benefits Associated with Air Quality Changes Resulting from the REMSAD-Based "75 Percent Reduction" Scenario

Endpoint	Reference	Avoided Incidence (cases/year)			Monetary Benefits (millions 1999$)		
		5th %ile	Mean	95th %ile	5th %ile	Mean	95th %ile
MORTALITY							
Ages 30+	Krewski et al. (2000)	10,500	18,700	26,500	14,900	106,000	258,000
CHRONIC ILLNESS							
Chronic Bronchitis	Pooled Analysis	3,940	11,400	19,600	356	3,770	12,300
HOSPITALIZATION							0
COPD-Related	Samet et al. (2000)	454	2,000	3,580	6	25	44
Pneumonia-Related	Samet et al. (2000)	1,340	2,440	3,540	20	36	52
Asthma-Related	Sheppard et al. (1999)	748	1,860	2,920	5	13	20
Cardiovascular-Related	Samet et al. (2000)	5,010	5,880	6,810	92	108	125
Asthma-Related ER Visits	Schwartz et al. (1993)	1,790	4,320	6,740	1	1	2
MINOR ILLNESS							
Acute Bronchitis	Dockery et al. (1996)	-190	37,100	74,100	0	2	5
Upper Respiratory Symptoms	Pope et al. (1991)	138,000	412,000	685,000	3	10	22
Lower Respiratory Symptoms	Schwartz et al. (1994)	186,000	397,000	596,000	2	6	11
Asthma Attacks	Whittemore and Korn (1980)	127,000	366,000	604,000	4	15	32
Work Loss Days	Ostro (1987)	2,770,000	3,190,000	3,580,000	294	338	379
MRAD	Ostro and Rothschild (1989)	14,000,000	16,400,000	18,700,000	479	796	1,150
TOTAL PRIMARY PM-RELATED BENEFITS					na	111,000	na

Exhibit 6-3 Estimated PM-Related Health and Welfare Benefits Associated with Air Quality Changes Resulting from the REMSAD-Based "All Power Plant" Scenario

Endpoint	Reference	Attributable Incidence (cases/year)			Monetary Benefits (millions 1999$)		
		5th %ile	Mean	95th %ile	5th %ile	Mean	95th %ile
MORTALITY							
Ages 30+	Krewski et al. (2000)	16,900	30,100	42,500	24,000	170,000	415,000
CHRONIC ILLNESS							
Chronic Bronchitis	Pooled Analysis	6,470	18,600	31,600	575	6,130	20,000
HOSPITALIZATION							
COPD-Related	Samet et al. (2000)	750	3,320	5,940	9	41	74
Pneumonia-Related	Samet et al. (2000)	2,220	4,040	5,870	33	59	86
Asthma-Related	Sheppard et al. (1999)	1,210	3,020	4,740	8	21	32
Cardiovascular-Related	Samet et al. (2000)	8,280	9,720	11,300	152	179	207
Asthma-Related ER Visits	Schwartz et al. (1993)	2,960	7,160	11,200	1	2	4
MINOR ILLNESS							
Acute Bronchitis	Dockery et al. (1996)	-307	59,000	116,000	0	3	8
Upper Respiratory Symptoms	Pope et al. (1991)	228,000	679,000	1,130,000	4	16	36
Lower Respiratory Symptoms	Schwartz et al. (1994)	299,000	630,000	935,000	3	10	18
Asthma Attacks	Whittemore and Korn (1980)	209,000	603,000	993,000	7	25	52
Work Loss Days	Ostro (1987)	4,460,000	5,130,000	5,750,000	472	543	609
MRAD	Ostro and Rothschild (1989)	22,500,000	26,300,000	29,800,000	767	1,270	1,840
TOTAL PRIMARY PM-RELATED BENEFITS					na	178,000	na

Exhibit 6-4 Alternative Mortality Calculations for the REMSAD-Based "75 Percent Reduction" and "All Power Plant" Scenarios

Age Group	Statistic	Mortality	Reference	"75 Percent Reduction" Scenario (avoided cases/year)			"All Power Plant" Scenario (attributable cases/year)		
				5th %ile	Mean	95th %ile	5th %ile	Mean	95th %ile
Age 30+	Median	Non-Accidental	Pope et al. (1995)	12,200	19,600	26,900	21,200	33,900	46,500
Age 30+	Median	Non-Accidental	Krewski et al. (2000)	9,220	16,400	23,500	16,000	28,400	40,600
Age 30+	Mean	Non-Accidental	Krewski et al. (2000)	10,500	17,900	25,200	16,800	28,700	40,600
Age 30+	Mean	All-Cause	Krewski et al. (2000)	10,500	18,700	26,500	16,900	30,100	42,500
Age 30+	Median	All-Cause	Krewski et al. (2000) - Random Effects, Independent Cities	17,600	33,200	47,700	30,400	57,300	82,200
Age 30+	Median	All-Cause	Krewski et al. (2000) - Random Effects, Regional Adjustment	1,040	19,400	36,500	1,810	33,600	63,100
Age 25+	Mean	Non-Accidental	Dockery et al. (1993)	20,600	48,500	75,800	33,200	77,600	121,000
Age 25+	Mean	Non-Accidental	Krewski et al. (2000)	26,600	51,800	78,100	42,800	82,900	124,000
Age 25+	Mean	All-Cause	Krewski et al. (2000)	28,100	54,600	80,700	45,100	87,300	128,000

Exhibit 6-5 Underlying Estimates and Weights for Pooled Estimate of PM-Related Chronic Bronchitis Studies "75 Percent Reduction" Scenario

Study	Ages Affected	Study Weights	5th %ile	mean	95th %ile
Abbey et al. (1995b)	>26	0.24	1,700	13,300	24,000
Schwartz (1993)	>29	0.76	4,390	10,800	16,800
Pooled estimate of chronic bronchitis			3,940	11,400	19,600

Exhibit 6-6 Underlying Estimates and Weights for Pooled Estimate of PM-Related Chronic Bronchitis Studies "All Power Plant Scenario"

Study	Ages Affected	Study Weights	5th %ile	mean	95th %ile
Abbey et al. (1995b)	>26	0.25	2,750	21,400	38,100
Schwartz (1993)	>29	0.75	7,200	17,700	27,300
Pooled estimate of chronic bronchitis			6,470	18,600	31,600

Exhibit 6-7 PM-Related Adverse Health Effects by State: "75 Percent Reduction" Scenario

State	Mortality	Chronic Bronchitis	Hospital Admissions	Asthma ER Visits	Acute Bronchitis	URS	LRS	Asthma Attacks	Work Loss Days	MRAD
Alabama	738	416	459	160	1,420	16,000	15,200	13,500	116,000	594,000
Arizona	11	8	8	3	27	297	299	251	2,150	11,200
Arkansas	277	144	174	53	503	5,560	5,530	4,610	38,400	198,000
California	49	38	36	15	132	1,450	1,440	1,280	11,200	58,400
Colorado	23	20	17	8	68	740	748	640	5,840	30,400
Connecticut	197	128	137	46	346	3,790	3,630	3,890	34,900	179,000
Delaware	80	51	53	20	159	1,780	1,680	1,640	14,600	74,900
District of Columbia	80	40	42	15	90	1,020	945	1,250	11,800	60,800
Florida	1,050	582	760	192	1,540	17,000	16,800	17,300	148,000	763,000
Georgia	1,090	747	688	309	2,620	29,800	28,000	25,200	223,000	1,140,000
Idaho	5	4	4	1	15	167	169	117	965	5,010
Illinois	981	589	635	222	1,980	21,900	21,400	19,000	164,000	848,000
Indiana	585	354	379	136	1,230	13,700	13,200	11,500	99,300	512,000
Iowa	183	106	128	38	366	4,040	3,990	3,330	27,800	144,000
Kansas	162	96	108	36	345	3,810	3,760	3,120	26,500	137,000
Kentucky	578	335	360	129	1,150	13,000	12,300	10,900	93,500	480,000
Louisiana	306	180	183	74	753	8,310	8,170	6,190	52,300	270,000
Maine	37	23	24	8	73	796	786	707	6,160	31,800
Maryland	619	428	397	166	1,280	14,300	13,500	13,700	124,000	638,000
Massachusetts	278	175	193	64	482	5,250	5,090	5,450	49,100	253,000
Michigan	523	338	343	131	1,180	13,000	12,700	11,000	95,600	493,000
Minnesota	153	108	111	42	391	4,310	4,240	3,530	30,600	159,000
Mississippi	318	171	192	69	705	7,850	7,640	5,880	48,400	249,000
Missouri	519	284	324	104	959	10,600	10,400	9,020	77,200	399,000
Montana	3	2	2	1	8	87	89	66	548	2,840
Nebraska	69	42	47	16	151	1,660	1,650	1,350	11,400	59,100
Nevada	5	3	3	1	10	116	115	109	982	5,110

Exhibit 6-7 PM-Related Adverse Health Effects by State: "75 Percent Reduction" Scenario (cont.)

State	Mortality	Chronic Bronchitis	Hospital Admissions	Asthma ER Visits	Acute Bronchitis	URS	LRS	Asthma Attacks	Work Loss Days	MRAD
New Hampshire	45	32	30	12	102	1,120	1,080	1,020	9,090	47,000
New Jersey	718	453	481	163	1,270	13,900	13,400	13,900	123,000	634,000
New Mexico	7	5	5	2	22	236	237	175	1,470	7,640
New York	1,200	744	792	273	2,180	23,900	23,300	23,200	206,000	1,060,000
North Carolina	1,190	744	771	287	2,250	25,300	24,100	24,000	213,000	1,100,000
North Dakota	10	6	7	2	24	260	260	207	1,730	8,950
Ohio	1,200	712	768	269	2,390	26,600	25,300	22,800	196,000	1,010,000
Oklahoma	250	138	154	51	488	5,370	5,330	4,420	37,500	194,000
Oregon	31	20	21	7	67	737	732	631	5,430	28,200
Pennsylvania	1,460	791	947	278	2,260	25,200	23,900	24,200	207,000	1,060,000
Rhode Island	57	34	40	12	95	1,040	1,000	1,060	9,380	48,300
South Carolina	515	318	324	127	1,110	12,500	11,900	10,600	91,900	472,000
South Dakota	19	11	14	4	42	461	460	354	2,880	14,900
Tennessee	857	500	533	188	1,570	17,800	17,000	15,900	139,000	715,000
Texas	805	565	534	229	2,160	23,600	23,500	19,100	168,000	868,000
Utah	7	6	6	3	40	436	436	246	1,900	9,820
Vermont	21	14	14	5	47	511	498	450	3,970	20,500
Virginia	828	571	542	223	1,770	19,900	18,800	18,400	166,000	855,000
Washington	31	23	23	9	81	895	879	744	6,390	33,200
West Virginia	296	153	181	55	488	5,450	5,170	4,700	39,700	203,000
Wisconsin	268	172	188	65	606	6,670	6,560	5,550	47,600	246,000
Wyoming	3	2	2	1	8	92	93	66	563	2,920

Exhibit 6-8 PM-Related Adverse Health Effects by State: "All Power Plant" Scenario

State	Mortality	Chronic Bronchitis	Hospital Admissions	Asthma ER Visits	Acute Bronchitis	URS	LRS	Asthma Attacks	Work Loss Days	MRAD
Alabama	1,110	627	701	246	2,090	24,300	22,300	20,600	173,000	886,000
Arizona	52	37	41	14	126	1,460	1,380	1,230	9,880	51,200
Arkansas	479	250	304	93	858	9,710	9,380	8,050	66,400	341,000
California	259	215	200	89	719	8,370	7,900	7,410	62,100	322,000
Colorado	64	56	48	22	189	2,100	2,060	1,800	16,000	82,800
Connecticut	299	197	213	71	522	5,880	5,430	6,040	52,800	271,000
Delaware	126	84	88	33	247	2,990	2,600	2,760	22,900	117,000
District of Columbia	118	60	64	23	132	1,550	1,380	1,900	17,500	89,900
Florida	1,740	1,010	1,350	342	2,530	30,000	27,400	30,800	245,000	1,260,000
Georgia	1,630	1,120	1,050	472	3,850	45,100	41,000	38,200	333,000	1,700,000
Idaho	8	6	6	2	25	280	276	192	1,530	7,950
Illinois	1,700	1,020	1,110	391	3,360	38,200	36,200	33,100	283,000	1,450,000
Indiana	1,030	623	679	244	2,110	24,300	22,600	20,500	173,000	886,000
Iowa	299	173	211	63	594	6,660	6,450	5,490	45,500	235,000
Kansas	274	163	185	62	577	6,470	6,280	5,300	44,600	230,000
Kentucky	997	578	635	229	1,940	22,700	20,600	19,000	161,000	819,000
Louisiana	481	284	291	118	1,170	13,200	12,600	9,800	81,900	422,000
Maine	55	34	36	12	108	1,190	1,150	1,060	9,090	46,900
Maryland	927	648	608	256	1,890	21,900	19,800	20,900	185,000	947,000
Massachusetts	441	283	313	104	760	8,550	7,990	8,880	78,000	401,000
Michigan	871	566	579	221	1,950	21,900	20,800	18,500	159,000	817,000
Minnesota	249	178	182	69	633	7,100	6,850	5,820	49,900	258,000
Mississippi	489	264	299	108	1,070	12,200	11,500	9,110	74,200	380,000
Missouri	896	494	569	184	1,630	18,600	17,600	15,800	133,000	684,000
Montana	6	4	4	1	14	154	154	116	954	4,950
Nebraska	122	73	84	28	264	2,930	2,880	2,390	19,900	103,000

Exhibit 6-8 PM-Related Adverse Health Effects by State: All Power Plant scenario (cont.)

State	Mortality	Chronic Bronchitis	Hospital Admissions	Asthma ER Visits	Acute Bronchitis	URS	LRS	Asthma Attacks	Work Loss Days	MRAD
Nevada	16	13	12	5	36	454	391	425	3,360	17,400
New Hampshire	67	48	46	18	152	1,700	1,600	1,540	13,500	69,800
New Jersey	1,100	708	758	259	1,910	22,100	20,200	21,900	189,000	967,000
New Mexico	23	17	17	7	74	831	804	599	4,880	25,300
New York	1,870	1,180	1,260	437	3,380	38,100	35,800	37,000	321,000	1,650,000
North Carolina	1,800	1,140	1,200	447	3,330	39,000	35,700	37,100	322,000	1,640,000
North Dakota	18	11	13	4	41	454	445	360	2,950	15,300
Ohio	1,920	1,150	1,250	442	3,770	43,400	39,700	37,100	313,000	1,600,000
Oklahoma	412	228	256	85	795	8,930	8,670	7,340	61,800	318,000
Oregon	43	29	31	11	95	1,060	1,040	912	7,740	40,100
Pennsylvania	2,250	1,240	1,510	445	3,430	40,100	36,000	38,400	318,000	1,620,000
Rhode Island	88	53	63	19	145	1,630	1,510	1,660	14,300	73,400
South Carolina	791	493	509	201	1,680	19,600	17,900	16,600	141,000	721,000
South Dakota	33	19	24	7	74	815	803	622	5,010	25,900
Tennessee	1,440	839	910	323	2,580	30,200	27,700	27,100	232,000	1,190,000
Texas	1,310	929	885	382	3,500	39,200	38,000	31,700	274,000	1,410,000
Utah	17	16	16	8	93	1,160	1,020	656	4,450	22,900
Vermont	32	22	22	8	71	786	749	692	6,030	31,100
Virginia	1,240	856	823	341	2,590	30,100	27,400	27,900	246,000	1,260,000
Washington	44	34	34	13	116	1,310	1,270	1,100	9,250	48,000
West Virginia	459	238	286	87	742	8,580	7,740	7,390	61,000	310,000
Wisconsin	448	288	317	109	1,000	11,200	10,800	9,340	79,300	409,000
Wyoming	7	5	5	2	23	262	249	183	1,490	7,710

Exhibit 6-9 PM-Related Adverse Health Effects by Metropolitan Statistical Area: "75 Percent Reduction" Scenario

MSA	Mortality	Chronic Bronchitis	Hospital Admissions	Asthma ER Visits	Acute Bronchitis	URS	LRS	Asthma Attacks	Work Loss Days	MRAD
Akron	283	166	185	60	520	5,780	5,540	5,160	44,500	229,000
Atlanta	431	366	283	154	1,240	14,100	13,200	12,300	113,000	581,000
Austin-SanMarcos	24	22	18	10	83	900	900	797	7,600	39,500
Boston	287	188	198	69	535	5,830	5,650	5,880	53,200	274,000
Boulder-Longmont	17	15	12	6	50	540	546	476	4,390	22,800
Buffalo-NiagaraFalls	99	54	64	19	155	1,710	1,630	1,660	14,300	73,400
Charlotte-Gastonia-RockHill	191	131	125	51	401	4,550	4,250	4,240	37,900	194,000
Chicago	572	373	368	145	1,270	14,100	13,800	12,200	107,000	553,000
Cincinnati	223	139	144	55	495	5,580	5,220	4,590	39,500	203,000
Columbus	128	90	83	37	298	3,320	3,140	3,020	27,400	141,000
Dallas	228	187	151	78	686	7,550	7,460	6,390	58,200	302,000
Dayton-Springfield	109	65	68	25	214	2,390	2,270	2,090	18,300	94,200
Detroit	322	209	207	80	702	7,730	7,520	6,740	59,100	305,000
FortLauderdale	40	22	31	6	49	526	533	610	5,130	26,600
GrandRapids-Muskegon-Holland	41	30	30	12	118	1,290	1,260	1,010	8,610	44,500
Greensboro--Winston-Salem--HighPoint	207	134	137	50	366	4,120	3,920	4,180	37,700	193,000
Hartford	72	46	49	17	128	1,400	1,340	1,430	12,900	66,400
Houston	127	111	82	47	447	4,910	4,860	3,820	34,300	178,000
Indianapolis	145	92	91	36	315	3,490	3,360	3,000	26,500	137,000
Jacksonville	74	47	46	19	158	1,740	1,710	1,560	13,900	71,800
KansasCity	116	76	75	29	266	2,950	2,890	2,430	21,300	110,000
LasVegas	4	3	3	1	9	98	97	93	836	4,350
LosAngeles-LongBeach	23	19	17	8	67	732	728	653	5,760	29,900
Louisville	145	85	89	32	279	3,140	2,960	2,690	23,400	120,000
Memphis	109	65	62	27	247	2,760	2,680	2,210	19,200	99,100
Milwaukee-Waukesha	97	62	64	23	214	2,370	2,310	1,980	17,100	88,500
Minneapolis-St.Paul	83	69	60	27	242	2,670	2,630	2,270	20,400	106,000
Nashville	149	101	95	40	330	3,730	3,560	3,300	29,600	152,000

Exhibit 6-9 PM-Related Adverse Health Effects by Metropolitan Statistical Area: "75 Percent Reduction" Scenario (cont.)

MSA	Mortality	Chronic Bronchitis	Hospital Admissions	Asthma ER Visits	Acute Bronchitis	URS	LRS	Asthma Attacks	Work Loss Days	MRAD
NewOrleans	97	56	56	22	219	2,420	2,380	1,890	16,100	83,400
NewYork	1,470	945	991	341	2,620	28,700	27,800	29,000	259,000	1,330,000
Norfolk-VirginiaBeach-NewportNews	150	107	97	46	387	4,340	4,110	3,750	33,600	173,000
OklahomaCity	48	30	29	12	109	1,190	1,190	992	8,780	45,500
Orlando	88	61	65	23	183	2,010	1,980	1,930	17,400	89,800
Philadelphia	647	373	406	138	1,130	12,500	11,900	11,700	102,000	527,000
Phoenix-Mesa	7	5	5	2	17	190	191	164	1,430	7,410
Pittsburgh	371	192	241	63	493	5,510	5,210	5,620	48,000	246,000
Portland-Vancouver	21	15	15	6	51	560	554	474	4,100	21,300
Raleigh-Durham-ChapelHill	118	93	82	38	270	3,040	2,880	3,120	29,400	151,000
Richmond-Petersburg	138	86	85	33	255	2,870	2,690	2,730	24,600	126,000
Rochester	59	38	40	14	121	1,340	1,280	1,220	10,700	55,200
Sacramento	3	2	2	1	8	87	86	74	657	3,420
SaltLakeCity-Ogden	4	4	3	2	23	257	256	149	1,180	6,130
SanAntonio	54	39	38	16	162	1,740	1,760	1,360	11,800	61,300
SanDiego	3	2	2	1	8	84	85	81	739	3,840
SanFrancisco	9	7	7	3	21	232	230	230	2,100	10,900
Seattle-Bellevue-Everett	15	13	12	5	41	452	441	405	3,580	18,600
St.Louis	280	159	170	59	547	6,010	5,890	5,060	43,900	227,000
Tampa-St.Petersburg-Clearwater	291	143	211	43	323	3,570	3,510	4,040	33,400	172,000
Washington	762	585	501	231	1,750	19,600	18,400	18,800	173,000	890,000
WestPalmBeach-BocaRaton	37	19	30	5	40	434	435	522	4,200	21,700

Exhibit 6-10 PM-Related Adverse Health Effects by Metropolitan Statistical Area: "All Power Plant" Scenario

MSA	Mortality	Chronic Bronchitis	Hospital Admissions	Asthma ER Visits	Acute Bronchitis	URS	LRS	Asthma Attacks	Work Loss Days	MRAD
Akron	442	261	293	96	802	9,150	8,490	8,170	69,300	355,000
Atlanta	647	550	432	237	1,820	21,400	19,300	18,700	169,000	866,000
Austin-SanMarcos	41	39	31	17	140	1,560	1,510	1,390	12,900	66,700
Boston	454	302	320	113	839	9,420	8,820	9,540	84,000	432,000
Boulder-Longmont	40	37	29	14	121	1,340	1,320	1,180	10,700	55,400
Buffalo-NiagaraFalls	149	82	98	29	230	2,600	2,400	2,530	21,400	110,000
Charlotte-Gastonia-RockHill	298	206	201	83	614	7,290	6,480	6,780	59,200	302,000
Chicago	995	651	648	256	2,190	24,800	23,600	21,400	186,000	957,000
Cincinnati	377	236	248	95	820	9,590	8,580	7,870	66,400	339,000
Columbus	201	142	132	59	459	5,270	4,810	4,790	42,700	219,000
Dallas	369	304	247	129	1,100	12,400	11,900	10,500	94,100	486,000
Dayton-Springfield	181	109	115	42	349	4,030	3,690	3,520	30,300	155,000
Detroit	527	343	343	134	1,140	12,800	12,100	11,200	96,400	496,000
FortLauderdale	68	39	55	12	84	946	915	1,100	8,870	45,800
GrandRapids-Muskegon-Holland	72	52	53	21	203	2,290	2,160	1,790	15,000	77,200
Greensboro--Winston-Salem--HighPoint	309	201	210	77	535	6,280	5,700	6,380	56,000	286,000
Hartford	110	72	77	27	194	2,190	2,020	2,240	19,700	101,000
Houston	201	178	132	76	705	7,890	7,650	6,140	54,400	281,000
Indianapolis	250	161	161	64	531	6,170	5,650	5,300	45,400	233,000
Jacksonville	131	87	84	35	276	3,250	2,990	2,910	24,500	126,000
KansasCity	194	127	126	49	439	4,960	4,760	4,100	35,500	183,000
LasVegas	18	13	13	5	35	445	386	423	3,330	17,200
LosAngeles-LongBeach	184	156	143	65	520	6,080	5,730	5,440	45,400	236,000
Louisville	256	152	162	59	480	5,670	5,080	4,870	41,200	210,000
Memphis	185	110	107	46	412	4,720	4,460	3,780	32,500	167,000
Milwaukee-Waukesha	163	104	110	40	357	4,030	3,830	3,370	28,700	148,000
Minneapolis-St.Paul	135	113	99	45	392	4,420	4,240	3,750	33,200	172,000
Nashville	260	175	167	71	558	6,530	5,970	5,800	51,200	262,000

Exhibit 6-10 PM-Related Adverse Health Effects by Metropolitan Statistical Area: "All Power Plant" Scenario (cont.)

MSA	Mortality	Chronic Bronchitis	Hospital Admissions	Asthma ER Visits	Acute Bronchitis	URS	LRS	Asthma Attacks	Work Loss Days	MRAD
NewOrleans	152	89	89	36	340	3,830	3,670	2,990	25,200	130,000
NewYork	2,290	1,490	1,580	546	4,020	45,700	42,700	46,200	402,000	2,060,000
Norfolk-VirginiaBeach-NewportNews	217	158	144	69	555	6,460	5,870	5,580	48,600	249,000
OklahomaCity	81	51	50	20	182	2,030	1,980	1,690	14,800	76,500
Orlando	152	108	116	41	313	3,620	3,380	3,490	29,900	154,000
Philadelphia	997	593	654	225	1,720	20,300	18,100	19,000	158,000	808,000
Phoenix-Mesa	30	23	24	9	75	866	818	751	6,130	31,800
Pittsburgh	585	309	395	105	765	9,030	8,020	9,210	75,500	385,000
Portland-Vancouver	32	23	23	9	76	859	832	729	6,190	32,100
Raleigh-Durham-ChapelHill	174	139	125	58	392	4,590	4,170	4,700	43,300	222,000
Richmond-Petersburg	203	128	128	50	369	4,310	3,870	4,100	36,000	184,000
Rochester	90	59	62	23	185	2,090	1,940	1,900	16,300	84,000
Sacramento	5	4	4	2	14	161	154	136	1,180	6,110
SaltLakeCity-Ogden	10	10	9	5	55	705	597	410	2,760	14,200
SanAntonio	93	69	67	29	277	3,090	3,010	2,410	20,500	106,000
SanDiego	20	16	16	7	51	575	554	552	4,840	25,100
SanFrancisco	20	17	15	6	48	547	520	541	4,760	24,700
Seattle-Bellevue-Everett	23	19	18	7	60	684	652	613	5,310	27,500
St.Louis	494	285	309	109	947	10,900	10,200	9,200	77,300	397,000
Tampa-St.Petersburg-Clearwater	494	271	409	86	549	7,200	5,960	8,070	57,200	293,000
Washington	1,140	881	764	354	2,560	29,800	26,900	28,600	257,000	1,320,000
WestPalmBeach-BocaRaton	59	32	50	9	65	723	698	870	6,790	35,000

7. REFERENCES

Abbey, D.E., F. Petersen, P.K. Mills and W.L. Beeson. 1993. Long-Term Ambient Concentrations of Total Suspended Particulates, Ozone, and Sulfur Dioxide and Respiratory Symptoms in a Nonsmoking Population. Archives of Environmental Health. Vol. 48(1): 33-46.

Abbey, D.E., B.L. Hwang, R.J. Burchette, T. Vancuren and P.K. Mills. 1995a. Estimated Long-Term Ambient Concentrations of Pm(10) and Development of Respiratory Symptoms in a Nonsmoking Population. Archives of Environmental Health. Vol. 50(2): 139-152.

Abbey, D.E., B.E. Ostro, F. Petersen and R.J. Burchette. 1995b. Chronic Respiratory Symptoms Associated with Estimated Long-Term Ambient Concentrations of Fine Particulates Less Than 2.5 Microns in Aerodynamic Diameter (PM2.5) and Other Air Pollutants. J Expo Anal Environ Epidemiol. Vol. 5(2): 137-159.

Abbey, D.E., R.J. Burchette, S.F. Knutsen, W.F. McDonnell, M.D. Lebowitz and P.L. Enright. 1998. Long-term particulate and other air pollutants and lung function in nonsmokers. American Journal of Respiratory and Critical Care Medicine. Vol. 158(1): 289-298.

Abt Associates Inc. 1999. Co-Control Benefits of Greenhouse Gas Control Policies. Prepared for U.S. EPA, Office of Policy, under contract no. 68-W4-0029. Washington, DC. February.

Ackermann-Liebrich, U., P. Leuenberger, J. Schwartz, C. Schindler, C. Monn, C. Bolognini, J.P. Bongard, O. Brandli, G. Domenighetti, S. Elsasser, L. Grize, W. Karrer, R. Keller, H. KellerWossidlo, N. Kunzli, B.W. Martin, T.C. Medici, A.P. Perruchoud, M.H. Schoni, J.M. Tschopp, B. Villiger, B. Wuthrich, J.P. Zellweger and E. Zemp. 1997. Lung function and long term exposure to air pollutants in Switzerland. Study on Air Pollution and Lung Diseases in Adults (SAPALDIA) Team. Am J Respir Crit Care Med. Vol. 155(1): 122-129.

Adams, P.F. and M.A. Marano. 1995. Current Estimates from the National Health Interview Survey, 1994. National Center for Health Statistics. Hyattsville, MD. Vital Health Statistics, Series 10, No. 193. December.

Alberini, A., M. Cropper, T.T. Fu, A. Krupnick, J.T. Liu, D. Shaw and W. Harrington. 1997. Valuing health effects of air pollution in developing countries: The case of Taiwan. Journal of Environmental Economics and Management. Vol. 34(2): 107-126.

Barnard, W.R. 1996. Updates to Fugitive Emission Components of the National Particulate Inventory. Prepared for U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards. Durham, NC. January.

Butler, R.J. 1983. Wage and Injury Rate Responses to Shifting Levels of Workers' Compensation. In: Safety and the Work Force. Worrall, J.D., Ed. Cornell University, ILR Press: Ithaca, NY.

CARB (California Air Resources Board). 1982. California Ambient Air Quality Standard for Particulate Matter. Sacramento, CA. December.

Chestnut, L.G. 1995. Dollars and Cents: The Economic and Health Benefits of Potential Particulate Matter Reductions in the United States. Prepared for American Lung Association.

Collins, J.G. 1997. Prevalence of Selected Chronic Conditions: United States 1990-1992. National Center for Health Statistics. Hyattsville, MD. Vital Health Statistics, Series 10, No. 194.

Council of Economic Advisers. 2000. The Annual Report of the Council of Economic Advisers. In: Economic Report of the President. U.S. Government Printing Office: Washington, DC.

Cousineau, J., R. Lacroix and A. Girard. 1988. Occupational Hazard and Wage Compensating Differentials. University of Montreal Working Paper.

Cousineau, J., R. Lacroix and A. Girard. 1992. Occupational Hazard and Wage Compensating Differentials. The Review of Economics and Statistics. Vol. 74: 166-169.

Cropper, M.L. and A.J. Krupnick. 1990. The Social Costs of Chronic Heart and Lung Disease. Resources for the Future. Washington, DC. Discussion Paper QE 89-16-REV.

Decisioneering. 1996. Crystal Ball: Forecasting and Risk Analysis for Spreadsheet Users: User Manual. www.decisioneering .com.

Detels, R., D.P. Tashkin, J.W. Sayre, S.N. Rokaw, F.J. Massey, A.H. Coulson and D.H. Wegman. 1991. The Ucla Population Studies of Cord .10. a Cohort Study of Changes in Respiratory Function Associated With Chronic Exposure to Sox, Nox, and Hydrocarbons. American Journal of Public Health. Vol. 81(3): 350-359.

Dickie, M. and S. Gerking. 1987. Reconciling Averting Behavior and Contingent Valuation Benefit Estimates of Reducing Symptoms of Ozone Exposure (draft), as cited in Neumann, J.E., M. Dickie, and R.E. Unsworth. 1994. Prepared by Industrial Economics. Prepared for Jim DeMocker, U.S. EPA, Office of Air and Radiation. March 31.

Dillingham, A. 1985. The Influence of Risk Variable Definition on Value of Life Estimates. Economic Inquiry. Vol. 24: 277-294.

Dockery, D.W., F.E. Speizer, D.O. Stram, J.H. Ware, J.D. Spengler and B.G. Ferris, Jr. 1989. Effects of Inhalable Particles on Respiratory Health of Children. Am Rev Respir Dis. Vol. 139: 587-594.

Dockery, D.W., C.A. Pope, X.P. Xu, J.D. Spengler, J.H. Ware, M.E. Fay, B.G. Ferris and F.E. Speizer. 1993. An association between air pollution and mortality in six U.S. cities. N Engl J Med. Vol. 329(24): 1753-1759.

Dockery, D.W., J. Cunningham, A.I. Damokosh, L.M. Neas, J.D. Spengler, P. Koutrakis, J.H. Ware, M. Raizenne and F.E. Speizer. 1996. Health Effects of Acid Aerosols On North American Children - Respiratory Symptoms. Environmental Health Perspectives. Vol. 104(5): 500-505.

E.H. Pechan & Associates Inc. 1995. Updates to Fugitive Emission Components of the National Particulate Inventory. William Kuykendal, U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards, Innovative Strategies and Economics Group. July 17.

E.H. Pechan & Associates Inc. 1997a. Control Measure Analysis of Ozone and PM Alternatives -- Methodologies and Results. Prepared for U.S. EPA, Office of Air Quality Planning and Standards, Innovative Strategies and Economics Group. Research Triangle Park, NC. July 17.

E.H. Pechan & Associates Inc. 1997b. Ozone Transport Assessment Group (OTAG) Emissions Inventory Development Report, Volume III: Projections and Controls. Draft report prepared for U.S. EPA, Office of Air Quality Planning and Standards, Innovative Strategies and Economics Group. Research Triangle Park, NC. July 17.

E.H. Pechan & Associates Inc. 1999. Procedures for Developing Base Year and Future Year Mass and Modeling Inventories for the Tier 2 Final Rulemaking. Prepared for U.S. EPA, Office of Air Quality Planning and Standards. Springfield, VA. September.

E.H. Pechan & Associates Inc. 2000. Analysis of Air Quality Impacts of Limiting Emissions from Electricity Generating Units: Draft Report. Prepared for Abt Associates Inc. Springfield, VA. September.

Ehrlich, I. and H. Chuma. 1990. A Model of the Demand For Longevity and the Value of Life Extension. Journal of Political Economy. Vol. 98(4): 761-782.

Elixhauser, A., R.M. Andrews and S. Fox. 1993. Clinical Classifications for Health Policy Research: Discharge Statistics by Principal Diagnosis and Procedure. U.S. Department of Health Services, Center for General Health Services Intramural Research, Agency for Health Care Policy and Research.

Empire State Electric Energy Research Corporation (ESEERCO). 1994. New York State Environmental Externalities Cost Study. Report 2: Methodology. Prepared by RCG/Hagler, Bailly, Inc. November.

Garen, J. 1988. Compensating Wage Differentials and the Endogeneity of Job Riskiness. The Review of Economics and Statistics. Vol. 70(1): 9-16.

Gegax, D., S. Gerking and W. Shulze. 1985. Perceived Risk and the Marginal Value of Safety. Working paper prepared for the U. S. Environmental Protection Agency.

Gegax, D., S. Gerking and W. Shulze. 1991. Perceived Risk and the Marginal Value of Safety. The Review of Economics and Statistics. Vol. 73(4): 589-596.

Gerking, S., M. DeHaan and W. Schulze. 1988. The Marginal Value of Job Safety: A Contingent Valuation Study. Journal of Risk and Uncertainty. Vol. 1: 185-199.

Gery, M.W., G.Z. Whitten, J.P. Killus and M.C. Dodge. 1989. A photochemical kinetics mechanism for urban and regional scale computer modeling. J. Geophys. Res. Vol. 94(D10): 12,925 -12,956.

Gifford, F.A. 1982. Horizontal Diffusion in the Atomsphere: A Lagrangian-Dynamical Theory. Atmospheric Environment. Vol. 16(3): 505-512.

Graves, E.J. and B.S. Gillum. 1997. Detailed Diagnoses and Procedures, National Hospital Discharge Survey, 1994. National Center for Health Statistics. Hyattsville, MD. Vital Health Statistics, Series 13, No. 127. March.

Herzog, H.W., Jr., and A.M. Schlottmann. 1987. Valuing Risk in the Workplace: Market Price, Willingness to Pay, and the Optimal Provision of Safety. University of Tennessee Working Paper.

Herzog, H.W., Jr., and A.M. Schlottmann. 1990. Valuing Risk in the Workplace: Market Price, Willingness to Pay, and the Optimal Provision of Safety. The Review of Economics and Statistics. Vol. 72(3): 463-470.

ICF Consulting. 2000. Limiting SO2 and NOx Emissions from Electrical Generating Units. Prepared for Abt Associates Inc. July.

Industrial Economics Incorporated (IEc). 1992. Review of Existing Value of Life Estimates: Valuation Document. Memorandum to Jim DeMocker, U.S. Environmental Protection Agency, Office of Air and Radiation, Office of Policy Analysis and Review. November 6.

Industrial Economics Incorporated (IEc). 1993. Memorandum to Jim DeMocker, U.S. Environmental Protection Agency, Office of Air and Radiation, Office of Policy Analysis and Review. September 30.

Industrial Economics Incorporated (IEc). 1994. Linkage Between Health Effects Estimation and Morbidity Valuation in the Section 812 Analysis -- Draft Valuation Document. Memorandum to Jim DeMocker, U.S. Environmental Protection Agency, Office of Air and Radiation, Office of Policy Analysis and Review. Prepared by J.E. Neumann, M.T. Dickie, and R.E. Unsworth. March 31.

Jones-Lee, M.W., M. Hammerton and P.R. Philips. 1985. The Value of Safety: Result of a National Sample Survey. Economic Journal. Vol. 95(March): 49-72.

Jones-Lee, M.W. 1989. The Economics of Safety and Physical Risk. Basil Blackwell: Oxford.

Kennedy. 1990. A Guide to Econometrics. 2nd ed. MIT Press: Cambridge, MA.

Kniesner, T.J. and J.D. Leeth. 1991. Compensating Wage Differentials for Fatal Injury Risk in Australia, Japan, and the United States. Journal of Risk and Uncertainty. Vol. 4(1): 75-90.

Krewski, D., R. Burnett, M. Goldberg, K. Hoover, J. Siemiatycki, M. Jerrett, M. Abrahamowicz and W. White. 2000. Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality. Health Effects Institute. Cambridge. July.

Krupnick, A.J. 1988. An Analysis of Selected Health Benefits from Reductions in Photochemical Oxidants in the Northeastern United States: Final Report. Prepared for U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards. Washington, DC. EPA Contract No. 68-02-4323. September.

Krupnick, A.J. and R.J. Kopp. 1988. The Health and Agricultural Benefits of Reductions in Ambient Ozone in the United States. Resources for the Future. Washington, DC. Discussion Paper QE88-10. August.

Krupnick, A.J. and M.L. Cropper. 1992. The Effect of Information On Health Risk Valuations. Journal of Risk and Uncertainty. Vol. 5(1): 29-48.

Latimer, D.A. 1993. Development of Regional Haze Screening Models. 86th Annual Meeting and Exhibition. Air and Wast Management Association. Denver, CO. Vol. 93-TP-49.04:

Leigh, J.P. and R.N. Folsom. 1984. Estimates of the Value of Accident Avoidance at the Job Depend on Concavity of the Equalizing Differences Curve. The Quarterly Review of Economics and Business. Vol. 24(1): 56-66.

Leigh, J.P. 1987. Gender, Firm Size, Industry and Estimates of the Value-of-Life. Journal of Health Economics. Vol. 6: 255-273.

Lipfert, F.W. 1993. A Critical Review of Studies of the Association Between Demands For Hospital Services and Air Pollution. Environmental Health Perspectives. Vol. 101(S2): 229-268.

Loehman, E.T., S.V. Berg, A.A. Arroyo, R.A. Hedinger, J.M. Schwartz, M.E. Shaw, R.W. Fahien, V.H. De, R.P. Fishe, D.E. Rio, W.F. Rossley and A.E.S. Green. 1979. Distributional Analysis of Regional Benefits and Cost of Air Quality Control. Journal of Environmental Economics and Management. Vol. 6: 222-243.

Loehman, E.T. and V.H. De. 1982. Application of Stochastic Choice Modeling to Policy Analysis of Public Goods. The Review of Economics and Statistics. Vol. 64(3): 474-480.

Marin, A. and G. Psacharopoulos. 1982. The Reward for Risk in the Labor Market: Evidence from the United Kingdom and a Reconciliation with Other Studies. Journal of Political Economy. Vol. 90(4): 827-853.

McDonnell, W.F., D.E. Abbey, N. Nishino and M.D. Lebowitz. 1999. Long-term ambient ozone concentration and the incidence of asthma in nonsmoking adults: the AHSMOG study. Environ Res. Vol. 80(2 Pt 1): 110-21.

Medical Center Information Systems: Duke University Health Systems ICD-9-CM.; November 5: http://dumccss.mc.duke.edu/standards/termcode/icd9/index.html, 1999.

Miller, T. and J. Guria. 1991. The Value of Statistical Life in New Zealand. Report to the New Zealand Ministry of Transport, Land Transport Division.

Mitchell, R.C. and R.T. Carson. 1986. The Use of Contingent Valuation Data for Benefit/Cost Analysis in Water Pollution Control. Draft report submitted by Resources for the Future to Environmental Protection Agency, Office of Policy Analysis. Washington, DC. September.

Mood, A.M., F.A. Graybill and D.C. Boes. 1974. Introduction to the Theory of Statistics. 3rd ed. McGraw Hill Book Company: New York.

Moore, M.J. and W.K. Viscusi. 1988. Doubling the Estimated Value of Life: Results Using New Occupational Fatality Data. Journal of Policy Analysis and Management. Vol. 7(3): 476-490.

National Heart, L., and Blood Institute. 1997. Guidelines for the Diagnosis and Management of Asthma: Expert Panel Report 2. National Institutes of Health. Bethesda, MD. NIH Publication No. 97-4051. July.

Neas, L.M., D.W. Dockery, J.H. Ware, J.D. Spengler, B.G. Ferris and F.E. Speizer. 1994. Concentration of Indoor Particulate Matter As a Determinant of Respiratory Health in Children. American Journal of Epidemiology. Vol. 139(11): 1088-1099.

Olson, C.A. 1981. An Analysis of Wage Differentials Received by Workers on Dangerous Jobs. Journal of Human Resources. Vol. 16: 167-185.

Ostro, B.D. 1987. Air Pollution and Morbidity Revisited: A Specification Test. Journal of Environmental Economics and Management. Vol. 14: 87-98.

Ostro, B.D., M.J. Lipsett and N.P. Jewell. 1989. Predicting Respiratory Morbidity From Pulmonary Function Tests - a Reanalysis of Ozone Chamber Studies. Japca. Vol. 39(10): 1313-1318.

Ostro, B.D. and S. Rothschild. 1989. Air Pollution and Acute Respiratory Morbidity - an Observational Study of Multiple Pollutants. Environ Res. Vol. 50(2): 238-247.

Ostro, B.D., J.M. Sanchez, C. Aranda and G.S. Eskeland. 1996. Air Pollution and Mortality - Results From a Study of Santiago, Chile. J Expo Anal Environ Epidemiol. Vol. 6(1): 97-114.

Pope, C.A., D.W. Dockery, J.D. Spengler and M.E. Raizenne. 1991. Respiratory Health and Pm10 Pollution - a Daily Time Series Analysis. American Review of Respiratory Disease. Vol. 144(3): 668-674.

Pope, C.A., J. Schwartz and M.R. Ransom. 1992. Daily Mortality and PM10 Pollution in Utah Valley. Archives of Environmental Health. Vol. 47(3): 211-217.

Pope, C.A., M.J. Thun, M.M. Namboodiri, D.W. Dockery, J.S. Evans, F.E. Speizer and C.W. Heath. 1995. Particulate air pollution as a predictor of mortality in a prospective study of U.S. adults. Am J Respir Crit Care Med. Vol. 151(3): 669-674.

Richards, W., S.P. Azen, J. Weiss, S. Stocking and J. Church. 1981. Los Angeles air pollution and asthma in children. Ann Allergy. Vol. 47(5 Pt 1): 348-54.

Rossi, G., M.A. Vigotti, A. Zanobetti, F. Repetto, V. Gianelle and J. Schwartz. 1999. Air pollution and cause-specific mortality in Milan, Italy, 1980-1989. Arch Environ Health. Vol. 54(3): 158-64.

Rowe, R.D. and L.G. Chestnust. 1986. Oxidants and Asthmatics in Los Angeles: A Benefits Analysis -- Executive Summary. Prepared for U.S. Environmental Protection Agency, Office of Policy Analysis. Prepared by Energy and Resource Consultants, Inc. Washington, DC. EPA-230-09-86-018. March.

Samet, J., S. Zeger, F. Dominici, F. Curriero, I. Coursac, D. Dockery, J. Schwartz and A. Zanobetti. 2000. The National Morbidity, Mortality, and Air Pollution Study. Health Effects Institute. Cambridge, MA. 94. May.

Schwartz, J. and D.W. Dockery. 1992. Particulate Air Pollution and Daily Mortality in Steubenville, Ohio. American Journal of Epidemiology. Vol. 135(1): 12-19.

Schwartz, J. 1993. Particulate Air Pollution and Chronic Respiratory Disease. Environ Res. Vol. 62: 7-13.

Schwartz, J., D. Slater, T.V. Larson, W.E. Pierson and J.Q. Koenig. 1993. Particulate air pollution and hospital emergency room visits for asthma in Seattle. Am Rev Respir Dis. Vol. 147(4): 826-31.

Schwartz, J. 1994a. Air Pollution and Hospital Admissions For the Elderly in Detroit, Michigan. American Journal of Respiratory and Critical Care Medicine. Vol. 150(3): 648-655.

Schwartz, J. 1994b. Air Pollution and Hospital Admissions For the Elderly in Birmingham, Alabama. American Journal of Epidemiology. Vol. 139(6): 589-598.

Schwartz, J. 1994c. What Are People Dying of On High Air Pollution Days. Environmental Research. Vol. 64(1): 26-35.

Schwartz, J., D.W. Dockery, L.M. Neas, D. Wypij, J.H. Ware, J.D. Spengler, P. Koutrakis, F.E. Speizer and B.G. Ferris. 1994. Acute Effects of Summer Air Pollution On Respiratory Symptom Reporting in Children. Am J Respir Crit Care Med. Vol. 150(5): 1234-1242.

Schwartz, J., D.W. Dockery and L.M. Neas. 1996. Is Daily Mortality Associated Specifically With Fine Particles. Journal of the Air & Waste Management Association. Vol. 46(10): 927-939.

Sheppard, L., D. Levy, G. Norris, T.V. Larson and J.Q. Koenig. 1999. Effects of ambient air pollution on nonelderly asthma hospital admissions in Seattle, Washington, 1987-1994. Epidemiology. Vol. 10(1): 23-30.

Smith, D.H., D.C. Malone, K.A. Lawson, L.J. Okamoto, C. Battista and W.B. Saunders. 1997. A national estimate of the economic costs of asthma. Am J Respir Crit Care Med. Vol. 156(3 Pt 1): 787-93.

Smith, R.S. 1974. The Feasibility of an 'Injury Tax' Approach to Occupational Safety. Law and Contemporary Problems. Vol. 38(4): 730-744.

Smith, R.S. 1976. The Occupational Safety and Health Act: Its Goals and Achievements. American Enterprise Institute. Washington, DC.

Smith, V.K. 1983. The Role of Site and Job Characteristics in Hedonic Wage Models. Journal of Urban Economics. Vol. 13: 296-321.

Smith, V.K. and C. Gilbert. 1984. The Implicit Risks to Life: A Comparative Analysis. Economics Letters. Vol. 16: 393-399.

Spix, C., J. Heinrich, D. Dockery, J. Schwartz, G. Volksch, K. Schwinkowski, C. Collen and H.E. Wichmann. 1993. Air Pollution and Daily Mortality in Erfurt, East-Germany, 1980-1989. Environmental Health Perspectives. Vol. 101(6): 518-526.

Stella, G. 1999. U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards, Emission Factors and Inventory Group. Personal communication with Erica Laich, E.H. Pechan & Associates, Inc. Email providing information on monthly percentage profiles by state, prime mover, and fuel use in developing June and August daily heat input and emissions to EGU inventory. 21, J.

Tolley, G.S. and et al. 1986. Valuation of Reductions in Human Health Symptoms and Risks. Prepared for U.S. Environmental Protection Agency. January.

U.S. Bureau of Economic Analysis. 1995. BEA Regional Projections to 2045: Volume 1, States. U.S. Department of Commerce. Washington, DC. July.

U.S. Bureau of the Census. 1992. Statistical Abstract of the United States: 1992. 112 ed. Washington, DC.

U.S. Bureau of the Census. 1997. Statistical Abstract of the United States: 1997. 117 ed. Washington, DC.

U.S. Bureau of the Census. 1998. Statistical Abstract of the United States: 1998. 118 ed. Washington, DC.

U.S. Centers for Disease Control. 1999. CDC Wonder. Downloaded May. http://wonder.cdc.gov/.

U.S. EPA. 1986. Review of the National Ambient Air Quality Standards for Particulate Matter: Updated Assessment of Scientific and Technical Information Addendum to the 1982 OAQPS Staff Paper. U.S. EPA, Office of Air Quality Planning and Standards. Research Triangle Park, NC. EPA 450/05-86-012.

U.S. EPA. 1997. Regulatory Impact Analyses for the Particulate Matter and Ozone National Ambient Air Quality Standards and Proposed Regional Haze Rule. U.S. EPA, Office of Air Quality Planning and Standards. Research Triangle Park, NC. July.

U.S. EPA. 1998a. Regulatory Impact Analysis for the NOx SIP Call, FIP, and Section 126 Petitions. U.S. EPA, Office of Air and Radiation. Washington, DC. EPA-452/R-98-003. December.

U.S. EPA. 1998b. Analyzing Electric Power Generation Under the CAAA. U.S. EPA, Office of Air and Radiation. March.

U.S. EPA. 1999a. An SAB Advisory: The Clean Air Act Section 812 Prospective Study Health and Ecological Initial Studies. Prepared by the Health and Ecological Effects SubCommittee (HEES) of the Advisory Council on the Clean Air Compliance Analysis, Science Advisory Board, U.S. Environmental Protection Agency. Washington, DC. EPA-SAB-Council-ADV-99-005. February.

U.S. EPA. 1999b. The Benefits and Costs of the Clean Air Act: 1990 to 2010: EPA Report to Congress. U.S. EPA, Office of Air and Radiation, Office of Policy. Washington, DC. EPA 410-R-99-001. November.

U.S. EPA. 1999c. The Clean Air Act Amendments (CAAA) Section 812 Prospective Study of Costs and Benefits (1999): Advisory by the Advisory Council on Clean Air Compliance Analysis: Costs and Benefits of the CAAA. Prepared by the Advisory Council on the Clean Air Compliance Analysis, Science Advisory Board, U.S. Environmental Protection Agency. Washington, DC. EPA-SAB-COUNCIL-ADV-00-002. October 29.

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

Viscusi, W.K. 1978. Labor Market Valuations of Life and Limb: Empirical Estimates and Policy Implications. Public Policy. Vol. 26(3): 359-386.

Viscusi, W.K. 1979. Employment Hazards: An Investigation of Market Performance. Harvard University Press: Cambridge.

Viscusi, W.K. 1981. Occupational Safety and Health Regulation: Its Impact and Policy Alternatives. In: Research in Public Policy Analysis and Management. Crecine, J., Ed. JAI Press: Greenwich, CT. Vol: 2. p. 281-299.

Viscusi, W.K., W.A. Magat and J. Huber. 1991. Pricing Environmental Health Risks - Survey Assessments of Risk - Risk and Risk - Dollar Trade-Offs For Chronic Bronchitis. Journal of Environmental Economics and Management. Vol. 21(1): 32-51.

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

Whittemore, A.S. and E.L. Korn. 1980. Asthma and Air Pollution in the Los Angeles Area. Am J Public Health. Vol. 70: 687-696.

Yamartino, R.J. 1985. Atmospheric Pollutant Deposition Modeling. In: Handbook of Applied Meteorology. Houghton, D.D., Ed. John Wiley & Sons: New York.

Zeger, S.L., F. Dominici and J. Samet. 1999. Harvesting-resistant estimates of air pollution effects on mortality. Epidemiology. Vol. 10(2): 171-5.

APPENDIX A: METROPOLITAN STATISTICAL AREAS

Exhibits A-1 and A-2 present the REMSAD-based results for all metropolitan statistical areas (MSAs) in the continental U.S. Exhibit A-3 presents the counties that are in each MSA and the estimated 2007 population for these counties.

Exhibit A-1 PM-Related Adverse Health Effects by Metropolitan Statistical Area: "75 Percent Reduction" Scenario

MSA	State	Population	Mortality	Chronic Bronch.	Hospital Admis.	Asthma ER Visits	Acute Bronch.	URS	LRS	Asthma Attacks	Work Loss Days	MRAD
Abilene	TX	158,508	4	3	3	1	10	105	106	87	758	3,930
Akron	OH	3,038,800	283	166	185	60	520	5,780	5,540	5,160	44,500	229,000
Albany	GA	150,035	15	10	9	4	44	491	471	346	2,910	15,000
Albany-Schenectady-Troy	NY	906,376	43	26	30	10	77	834	814	825	7,280	37,500
Albuquerque	NM	818,229	2	2	2	1	7	71	71	59	523	2,720
Alexandria	LA	149,570	10	5	6	2	22	240	238	177	1,480	7,620
Allentown-Bethlehem-Easton	PA	627,627	63	37	43	13	100	1,110	1,050	1,100	9,490	48,700
Altoona	PA	136,868	21	11	13	4	32	356	339	317	2,610	13,400
Amarillo	TX	246,598	3	2	2	1	9	97	97	74	650	3,370
Anniston	AL	139,054	25	13	14	5	45	503	477	438	3,830	19,600
Appleton-Oshkosh-Neenah	WI	358,203	13	9	10	4	35	385	377	309	2,680	13,900
Asheville	NC	241,640	44	24	29	8	62	697	657	712	6,130	31,400
Athens	GA	175,139	19	15	15	7	49	567	525	573	5,500	28,300
Atlanta	GA	3,964,069	431	366	283	154	1,240	14,100	13,200	12,300	113,000	581,000
Auburn-Opelika	AL	97,423	10	7	7	3	24	271	255	277	2,680	13,800
Augusta-Aiken	GA-SC	540,766	74	47	43	20	180	2,050	1,930	1,620	14,000	72,100
Austin-San Marcos	TX	1,116,410	24	22	18	10	83	900	900	797	7,600	39,500
Bakersfield	CA	665,377	1	1	0	0	2	25	25	18	148	769
Bangor	ME	191,687	4	3	3	1	9	95	95	84	752	3,900
Barnstable-Yarmouth	MA	201,278	14	7	10	2	17	182	173	195	1,580	8,140
Baton Rouge	LA	571,222	38	27	24	12	114	1,270	1,230	966	8,520	44,000
Beaumont-Port Arthur	TX	475,399	24	14	15	5	53	582	572	444	3,730	19,300
Bellingham	WA	169,697	0	0	0	0	1	10	10	8	73	379
Benton Harbor	MI	168,958	11	7	8	2	24	265	258	214	1,800	9,290
Billings	MT	146,333	0	0	0	0	1	13	13	10	86	446
Biloxi-Gulfport-Pascagoula	MS	354,653	33	20	20	8	79	876	855	688	5,910	30,400

Exhibit A-1 PM-Related Adverse Health Effects by Metropolitan Statistical Area: "75 Percent Reduction" Scenario (cont.)

MSA	State	Population	Mortality	Chronic Bronch.	Hospital Admis.	Asthma ER Visits	Acute Bronch.	URS	LRS	Asthma Attacks	Work Loss Days	MRAD
Binghamton	NY	287,626	20	12	14	4	37	407	387	377	3,280	16,900
Birmingham	AL	992,053	174	100	109	38	322	3,650	3,410	3,170	27,300	140,000
Bismarck	ND	89,362	1	1	1	0	3	35	35	27	225	1,170
Bloomington	IN	124,212	8	7	7	3	20	228	211	271	2,750	14,200
Bloomington-Normal	IL	140,591	10	7	7	3	25	274	264	261	2,440	12,600
Boise City	ID	454,755	1	1	1	0	5	52	53	38	323	1,680
Boston	MA-NH	6,991,988	287	188	198	69	535	5,830	5,650	5,880	53,200	274,000
Boulder-Longmont	CO	2,752,567	17	15	12	6	50	540	546	476	4,390	22,800
Brownsville-Harlingen-SanBenito	TX	346,141	4	3	3	1	17	185	191	116	896	4,630
Bryan-College Station	TX	159,612	5	4	4	2	17	185	178	194	1,960	10,200
Buffalo-Niagara Falls	NY	1,218,010	99	54	64	19	155	1,710	1,630	1,660	14,300	73,400
Burlington	VT	204,108	4	4	3	2	12	132	130	123	1,150	5,960
Canton-Massillon	OH	409,288	46	27	30	10	86	954	916	828	7,020	36,100
Casper	WY	79,731	0	0	0	0	1	12	12	9	76	396
Cedar Rapids	IA	178,822	9	7	7	3	23	253	250	221	1,960	10,100
Champaign-Urbana	IL	188,093	13	9	9	4	29	325	311	347	3,370	17,400
Charleston	WV	261,765	44	23	27	8	69	779	726	692	5,930	30,300
Charleston-North Charleston	SC	601,847	47	34	29	15	136	1,540	1,450	1,240	11,000	56,600
Charlotte-Gastonia-Rock Hill	NC-SC	1,460,744	191	131	125	51	401	4,550	4,250	4,240	37,900	194,000
Charlottesville	VA	158,737	20	13	13	5	36	402	376	440	4,170	21,500
Chattanooga	TN-GA	545,611	100	57	61	21	179	2,030	1,910	1,800	15,700	80,400
Cheyenne	WY	95,813	1	1	1	0	2	25	25	20	176	912
Chicago	IL	9,003,216	572	373	368	145	1,270	14,100	13,800	12,200	107,000	553,000
Chico-Paradise	CA	225,033	1	0	0	0	1	12	12	11	93	481

Exhibit A-1 PM-Related Adverse Health Effects by Metropolitan Statistical Area: "75 Percent Reduction" Scenario (cont.)

MSA	State	Population	Mortality	Chronic Bronch.	Hospital Admis.	Asthma ER Visits	Acute Bronch.	URS	LRS	Asthma Attacks	Work Loss Days	MRAD
Cincinnati	OH-KY-IN	1,947,621	223	139	144	55	495	5,580	5,220	4,590	39,500	203,000
Clarksville-Hopkinsville	TN-KY	202,112	18	13	12	6	50	562	540	507	4,610	23,800
Colorado Springs	CO	551,833	1	1	1	0	3	31	32	26	239	1,240
Columbia	SC	536,258	56	41	36	17	136	1,520	1,470	1,400	12,800	66,000
Columbia	MO	128,525	6	5	4	2	17	190	184	189	1,840	9,520
Columbus	OH	1,415,994	128	90	83	37	298	3,320	3,140	3,020	27,400	141,000
Columbus	GA-AL	350,300	52	30	31	12	105	1,190	1,120	1,020	8,840	45,300
Corpus Christi	TX	450,775	10	7	6	3	31	331	331	232	1,950	10,100
Corvallis	OR	110,085	1	1	0	0	2	18	19	18	176	913
Cumberland	MD-WV	119,023	22	10	13	3	27	304	286	300	2,520	12,900
Dallas	TX	5,307,754	228	187	151	78	686	7,550	7,460	6,390	58,200	302,000
Danville	VA	129,401	24	12	14	4	34	378	358	360	3,060	15,700
Davenport-Moline-RockIsland	IA-IL	377,234	30	18	20	6	63	692	687	558	4,740	24,500
Daytona Beach	FL	520,341	46	22	34	6	48	528	526	616	5,070	26,200
Dayton-Springfield	OH	1,005,479	109	65	68	25	214	2,390	2,270	2,090	18,300	94,200
Decatur	AL	151,257	21	13	14	5	46	513	503	435	3,770	19,400
Decatur	IL	128,361	15	8	10	3	28	309	305	259	2,180	11,200
DesMoines	IA	420,540	18	13	13	5	45	500	493	432	3,830	19,900
Detroit	MI	5,463,996	322	209	207	80	702	7,730	7,520	6,740	59,100	305,000
Dothan	AL	158,661	17	11	11	4	40	447	436	365	3,130	16,100
Dover	DE	125,701	11	7	7	3	27	297	280	251	2,190	11,200
Dubuque	IA	58,471	4	2	3	1	9	98	96	76	632	3,270
Duluth-Superior	MN-WI	277,005	6	3	4	1	11	115	116	99	825	4,270
EauClaire	WI	156,214	6	4	4	1	14	152	150	125	1,070	5,560

Exhibit A-1 PM-Related Adverse Health Effects by Metropolitan Statistical Area: "75 Percent Reduction" Scenario (cont.)

MSA	State	Population	Mortality	Chronic Bronch.	Hospital Admis.	Asthma ER Visits	Acute Bronch.	URS	LRS	Asthma Attacks	Work Loss Days	MRAD
Elkhart-Goshen	IN	179,988	10	7	7	3	28	302	300	244	2,080	10,700
Elmira	NY	101,706	9	5	6	2	16	177	166	151	1,270	6,530
ElPaso	TX	787,748	2	2	2	1	9	101	103	71	590	3,060
Enid	OK	64,850	4	2	2	1	6	70	69	55	458	2,370
Erie	PA	286,310	23	13	15	5	45	501	478	432	3,660	18,800
Eugene-Springfield	OR	371,712	3	2	2	1	6	67	66	59	517	2,690
Evansville-Henderson	IN-KY	316,843	42	24	27	9	78	876	831	740	6,300	32,400
Fargo-Moorhead	ND-MN	167,977	2	2	2	1	7	75	74	66	598	3,110
Fayetteville	NC	356,984	32	25	19	13	105	1,180	1,120	995	9,120	46,900
Fayetteville-Springdale-Rogers	AR	251,086	22	15	18	6	48	530	532	479	4,130	21,400
Flagstaff	AZ-UT	147,812	0	0	0	0	1	10	10	7	60	311
Florence	AL	155,821	27	15	17	5	45	512	493	469	4,060	20,900
Florence	SC	141,037	17	10	10	4	39	435	418	330	2,790	14,400
Fort Collins-Loveland	CO	260,092	2	2	2	1	6	69	69	60	554	2,880
Fort Lauderdale	FL	1,555,266	40	22	31	6	49	526	533	610	5,130	26,600
Fort Myers-Cape Coral	FL	447,165	21	11	18	3	24	258	256	307	2,460	12,700
Fort Pierce-Port St. Lucie	FL	327,920	13	7	10	2	16	172	170	186	1,500	7,760
Fort Smith	AR-OK	217,070	23	12	14	5	45	498	498	400	3,360	17,300
Fort Walton Beach	FL	184,439	15	12	10	5	40	436	430	394	3,590	18,500
Fort Wayne	IN	515,716	35	23	24	9	90	997	969	772	6,510	33,600
Fresno	CA	922,367	1	1	1	0	4	48	48	34	277	1,440
Gadsden	AL	118,516	27	13	17	5	41	470	441	412	3,440	17,600
Gainesville	FL	239,196	14	10	9	4	31	346	340	362	3,520	18,200
Glens Falls	NY	88,874	4	2	3	1	7	79	79	72	618	3,190

Exhibit A-1 PM-Related Adverse Health Effects by Metropolitan Statistical Area: "75 Percent Reduction" Scenario (cont.)

MSA	State	Population	Mortality	Chronic Bronch.	Hospital Admis.	Asthma ER Visits	Acute Bronch.	URS	LRS	Asthma Attacks	Work Loss Days	MRAD
Goldsboro	NC	130,660	19	12	11	5	40	449	436	400	3,560	18,300
Grand Forks	ND-MN	113,333	1	1	1	0	3	36	37	31	269	1,400
Grand Junction	CO	128,755	1	0	0	0	1	13	13	10	83	431
Grand Rapids-Muskegon-Holland	MI	1,000,106	41	30	30	12	118	1,290	1,260	1,010	8,610	44,500
GreatFalls	MT	99,816	0	0	0	0	1	6	6	5	39	201
Green Bay	WI	218,748	7	5	5	2	20	213	213	177	1,550	8,050
Greensboro--Winston-Salem--High Point	NC	1,343,693	207	134	137	50	366	4,120	3,920	4,180	37,700	193,000
Greenville	NC	135,297	16	10	10	4	33	374	363	357	3,290	16,900
Greenville-Spartanburg-Anderson	SC	985,653	145	89	93	34	277	3,130	2,960	2,860	25,200	129,000
Harrisburg-Lebanon-Carlisle	PA	597,604	76	46	51	16	132	1,480	1,400	1,410	12,400	63,500
Hartford	CT	1,326,689	72	46	49	17	128	1,400	1,340	1,430	12,900	66,400
Hattiesburg	MS	114,222	11	6	7	3	26	288	274	225	1,940	9,980
Hickory-Morganton-Lenoir	NC	369,838	54	36	36	13	103	1,160	1,110	1,110	9,900	50,800
Houma	LA	195,895	10	7	6	3	33	367	357	247	2,090	10,800
Houston	TX	4,913,333	127	111	82	47	447	4,910	4,860	3,820	34,300	178,000
Huntington-Ashland	WV-KY-OH	337,895	55	28	32	10	90	1,010	947	871	7,450	38,100
Huntsville	AL	340,441	39	30	26	12	96	1,090	1,030	1,010	9,290	47,800
Indianapolis	IN	1,572,962	145	92	91	36	315	3,490	3,360	3,000	26,500	137,000
Iowa City	IA	101,591	3	3	3	2	11	118	115	133	1,350	7,010
Jackson	MS	452,696	41	25	25	11	101	1,120	1,090	872	7,530	38,900
Jackson	TN	112,035	13	7	9	3	25	284	276	239	2,020	10,400
Jackson	MI	155,830	10	6	6	2	19	212	207	183	1,600	8,240
Jacksonville	FL	1,180,206	74	47	46	19	158	1,740	1,710	1,560	13,900	71,800

Exhibit A-1 PM-Related Adverse Health Effects by Metropolitan Statistical Area: "75 Percent Reduction" Scenario (cont.)

MSA	State	Population	Mortality	Chronic Bronch.	Hospital Admis.	Asthma ER Visits	Acute Bronch.	URS	LRS	Asthma Attacks	Work Loss Days	MRAD
Jacksonville	NC	190,295	10	9	7	6	38	426	402	438	4,350	22,500
Jamestown	NY	144,849	14	7	9	3	23	260	246	223	1,850	9,500
Janesville-Beloit	WI	161,217	10	6	7	2	23	251	246	205	1,750	9,040
Johnson City-Kingsport-Bristol	TN-VA	537,478	93	51	58	18	134	1,510	1,450	1,530	13,400	69,000
Johnstown	PA	253,500	39	20	26	6	57	637	607	581	4,730	24,200
Jonesboro	AR	83,910	7	4	5	2	15	162	160	147	1,310	6,750
Joplin	MO	155,108	18	10	11	3	32	358	354	300	2,500	12,900
Kalamazoo-Battle Creek	MI	441,064	24	16	16	6	55	605	587	517	4,550	23,500
Kansas City	MO-KS	1,791,964	116	76	75	29	266	2,950	2,890	2,430	21,300	110,000
Killeen-Temple	TX	332,715	9	7	6	3	31	335	332	279	2,520	13,100
Knoxville	TN	737,786	114	70	76	26	198	2,260	2,120	2,200	19,400	99,800
Kokomo	IN	109,357	9	6	6	2	21	230	228	193	1,670	8,620
LaCrosse	WI-MN	131,031	7	4	5	2	14	158	154	136	1,190	6,160
Lafayette	LA	382,013	19	12	12	5	56	618	608	436	3,680	19,000
Lafayette	IN	184,425	14	9	9	4	29	319	313	328	3,090	16,000
Lake Charles	LA	184,810	9	6	6	2	24	264	260	191	1,620	8,370
Lakeland-Winter Haven	FL	526,755	36	19	26	6	56	607	607	580	4,770	24,600
Lancaster	PA	439,469	54	34	38	13	116	1,310	1,230	1,110	9,390	48,200
Lansing-East Lansing	MI	456,760	20	15	13	6	56	615	599	518	4,710	24,300
Laredo	TX	174,981	1	1	1	1	9	94	97	59	463	2,390
Las Cruces	NM	180,761	1	0	0	0	2	22	23	17	143	745
Las Vegas	NV-AZ	1,467,639	4	3	3	1	9	98	97	93	836	4,350
Lawrence	KS	95,395	4	3	3	2	10	113	109	123	1,240	6,420
Lawton	OK	125,946	4	3	2	1	11	121	123	98	877	4,550

Exhibit A-1 PM-Related Adverse Health Effects by Metropolitan Statistical Area: "75 Percent Reduction" Scenario (cont.)

MSA	State	Population	Mortality	Chronic Bronch.	Hospital Admis.	Asthma ER Visits	Acute Bronch.	URS	LRS	Asthma Attacks	Work Loss Days	MRAD
Lewiston-Auburn	ME	112,945	3	2	2	1	7	74	72	64	552	2,860
Lexington	KY	454,516	56	38	36	16	123	1,380	1,310	1,300	11,900	61,300
Lima	OH	166,864	14	8	9	3	30	338	329	264	2,190	11,300
Lincoln	NE	241,281	8	6	6	2	19	211	209	197	1,820	9,440
Little Rock-North Little Rock	AR	610,612	46	29	29	11	102	1,130	1,120	949	8,320	43,000
Longview-Marshall	TX	245,628	22	12	14	5	47	520	513	397	3,270	16,900
Los Angeles-Long Beach	CA	17,763,602	23	19	17	8	67	732	728	653	5,760	29,900
Louisville	KY-IN	1,072,938	145	85	89	32	279	3,140	2,960	2,690	23,400	120,000
Lubbock	TX	294,525	4	2	2	1	10	104	106	87	801	4,160
Lynchburg	VA	233,684	37	21	24	8	60	686	642	665	5,840	29,900
Macon	GA	391,495	53	33	32	13	119	1,340	1,260	1,090	9,450	48,500
Madison	WI	417,101	17	15	13	6	46	503	494	506	4,850	25,100
Mansfield	OH	188,285	20	11	12	4	38	417	407	350	2,980	15,300
McAllen-Edinburg-Mission	TX	501,759	4	4	4	2	24	256	265	155	1,180	6,080
Medford-Ashland	OR	191,802	1	1	1	0	2	25	26	22	182	945
Melbourne-Titusville-Palm Bay	FL	522,202	27	16	20	5	42	461	454	481	4,220	21,900
Memphis	TN-AR-MS	1,253,499	109	65	62	27	247	2,760	2,680	2,210	19,200	99,100
Merced	CA	216,576	0	0	0	0	1	14	14	10	75	388
Milwaukee-Waukesha	WI	1,820,294	97	62	64	23	214	2,370	2,310	1,980	17,100	88,500
Minneapolis-St.Paul	MN-WI	2,942,826	83	69	60	27	242	2,670	2,630	2,270	20,400	106,000
Missoula	MT	102,046	0	0	0	0	1	7	7	6	55	285
Mobile	AL	557,578	61	37	40	14	139	1,530	1,500	1,220	10,200	52,600
Modesto	CA	458,480	1	1	1	0	3	37	35	26	208	1,080
Monroe	LA	159,432	10	6	6	2	25	279	277	208	1,750	9,050

Exhibit A-1 PM-Related Adverse Health Effects by Metropolitan Statistical Area: "75 Percent Reduction" Scenario (cont.)

MSA	State	Population	Mortality	Chronic Bronch.	Hospital Admis.	Asthma ER Visits	Acute Bronch.	URS	LRS	Asthma Attacks	Work Loss Days	MRAD
Montgomery	AL	340,717	49	29	30	12	108	1,220	1,150	984	8,430	43,200
Muncie	IN	133,491	12	7	7	3	20	219	213	223	2,050	10,600
Myrtle Beach	SC	172,374	19	12	12	4	35	396	382	366	3,260	16,800
Naples	FL	194,829	6	4	6	1	9	93	92	107	883	4,560
Nashville	TN	1,228,389	149	101	95	40	330	3,730	3,560	3,300	29,600	152,000
New London-Norwich	CT-RI	109,790	5	4	4	1	12	129	123	124	1,130	5,810
New Orleans	LA	1,411,716	97	56	56	22	219	2,420	2,380	1,890	16,100	83,400
New York	NY	20,578,316	1,470	945	991	341	2,620	28,700	27,800	29,000	259,000	1,330,000
Norfolk-VirginiaBeach-NewportNews	VA-NC	1,750,317	150	107	97	46	387	4,340	4,110	3,750	33,600	173,000
Ocala	FL	259,484	27	13	20	4	33	361	355	366	2,930	15,100
Odessa-Midland	TX	295,814	3	2	2	1	11	117	116	81	684	3,540
Oklahoma City	OK	1,091,027	48	30	29	12	109	1,190	1,190	992	8,780	45,500
Omaha	NE-IA	702,937	30	21	21	8	78	862	853	697	6,050	31,400
Orlando	FL	1,590,485	88	61	65	23	183	2,010	1,980	1,930	17,400	89,800
Owensboro	KY	97,223	12	7	8	3	26	291	281	235	1,980	10,200
Panama City	FL	166,259	18	11	12	4	37	409	397	364	3,210	16,500
Parkersburg-Marietta	WV-OH	155,110	23	13	15	5	41	455	425	394	3,340	17,100
Pensacola	FL	459,703	49	31	31	12	102	1,140	1,110	1,010	8,940	46,100
Peoria-Pekin	IL	366,759	36	21	24	8	73	809	799	668	5,640	29,100
Philadelphia	PA-NJ	6,414,340	647	373	406	138	1,130	12,500	11,900	11,700	102,000	527,000
Phoenix-Mesa	AZ	3,298,411	7	5	5	2	17	190	191	164	1,430	7,410
Pine Bluff	AR	102,116	10	5	6	2	19	216	212	165	1,370	7,050
Pittsburgh	PA	2,459,427	371	192	241	63	493	5,510	5,210	5,620	48,000	246,000

Exhibit A-1 PM-Related Adverse Health Effects by Metropolitan Statistical Area: "75 Percent Reduction" Scenario (cont.)

MSA	State	Population	Mortality	Chronic Bronch.	Hospital Admis.	Asthma ER Visits	Acute Bronch.	URS	LRS	Asthma Attacks	Work Loss Days	MRAD
Pittsfield	MA	149,519	9	5	6	2	13	141	137	141	1,200	6,190
Pocatello	ID	103,235	0	0	0	0	1	10	10	6	50	261
Portland	ME	257,111	8	5	6	2	15	165	164	164	1,470	7,580
Portland-Vancouver	OR-WA	2,371,025	21	15	15	6	51	560	554	474	4,100	21,300
Providence-FallRiver-Warwick	RI-MA	930,547	52	30	36	11	84	912	880	936	8,250	42,500
Provo-Orem	UT	379,915	1	1	1	0	6	66	66	37	286	1,480
Pueblo	CO	170,854	1	0	0	0	1	14	14	12	96	501
Punta Gorda	FL	129,773	10	5	9	1	7	82	81	118	875	4,500
Raleigh-Durham-ChapelHill	NC	1,088,464	118	93	82	38	270	3,040	2,880	3,120	29,400	151,000
Rapid City	SD	90,759	0	0	0	0	1	15	16	12	103	537
Reading	PA	330,183	40	23	28	8	65	726	684	699	5,980	30,700
Redding	CA	178,718	1	0	0	0	1	15	15	12	101	526
Reno	NV	444,290	1	0	0	0	1	13	12	13	115	598
Richland-Kennewick-Pasco	WA	202,015	1	1	1	0	5	55	54	38	317	1,640
Richmond-Petersburg	VA	1,053,301	138	86	85	33	255	2,870	2,690	2,730	24,600	126,000
Roanoke	VA	276,309	47	26	29	9	65	730	706	754	6,640	34,100
Rochester	NY	1,075,023	59	38	40	14	121	1,340	1,280	1,220	10,700	55,200
Rochester	MN	125,308	4	4	3	1	13	147	142	118	1,040	5,380
Rockford	IL	352,573	24	15	16	6	53	584	569	488	4,190	21,600
Rocky Mount	NC	167,594	26	14	15	6	50	556	543	467	3,980	20,400
Sacramento	CA	1,808,831	3	2	2	1	8	87	86	74	657	3,420
Saginaw-BayCity-Midland	MI	428,009	20	13	13	5	48	520	512	418	3,570	18,400
Salinas	CA	463,926	0	0	0	0	1	13	13	11	98	511
Salt Lake City-Ogden	UT	1,558,644	4	4	3	2	23	257	256	149	1,180	6,130

Exhibit A-1 PM-Related Adverse Health Effects by Metropolitan Statistical Area: "75 Percent Reduction" Scenario (cont.)

MSA	State	Population	Mortality	Chronic Bronch.	Hospital Admis.	Asthma ER Visits	Acute Bronch.	URS	LRS	Asthma Attacks	Work Loss Days	MRAD
San Angelo	TX	129,131	3	2	2	1	7	75	75	61	522	2,710
San Antonio	TX	1,735,324	54	39	38	16	162	1,740	1,760	1,360	11,800	61,300
San Diego	CA	3,040,458	3	2	2	1	8	84	85	81	739	3,840
San Francisco	CA	7,613,985	9	7	7	3	21	232	230	230	2,100	10,900
San Luis Obispo-Atascadero-Paso Robles	CA	265,215	0	0	0	0	1	6	6	6	58	302
Santa Barbara-Santa Maria-Lompoc	CA	452,536	0	0	0	0	1	9	9	9	80	413
Santa Fe	NM	163,156	0	0	0	0	1	11	11	9	84	434
Sarasota-Bradenton	FL	655,162	64	30	54	7	52	571	562	758	5,720	29,500
Savannah	GA	343,725	30	19	20	8	69	796	750	649	5,420	27,900
Scranton--Wilkes-Barre--Hazleton	PA	674,477	82	38	52	12	98	1,080	1,030	1,110	9,260	47,500
Seattle-Bellevue-Everett	WA	3,965,480	15	13	12	5	41	452	441	405	3,580	18,600
Sharon	PA	121,878	13	7	9	2	19	213	209	206	1,720	8,850
Sheboygan	WI	116,523	6	4	4	1	13	143	143	117	969	5,010
Sherman-Denison	TX	127,379	12	6	8	2	20	222	223	191	1,580	8,180
Shreveport-BossierCity	LA	413,424	29	16	17	6	63	688	680	526	4,400	22,700
Sioux City	IA-NE	126,860	7	4	4	1	15	165	161	122	990	5,120
Sioux Falls	SD	163,717	4	3	3	1	12	129	128	103	888	4,600
South Bend	IN	262,727	18	11	13	4	37	405	399	357	3,060	15,800
Spokane	WA	482,077	2	1	2	1	5	56	56	47	398	2,070
Springfield	MO	301,726	25	15	16	6	47	518	517	477	4,250	22,000
Springfield	IL	206,972	24	15	17	5	49	551	530	465	3,980	20,500
Springfield	MA	225,475	9	6	7	2	18	195	186	209	1,960	10,100
St. Cloud	MN	180,320	3	3	3	1	12	133	132	100	864	4,480

Exhibit A-1 PM-Related Adverse Health Effects by Metropolitan Statistical Area: "75 Percent Reduction" Scenario (cont.)

MSA	State	Population	Mortality	Chronic Bronch.	Hospital Admis.	Asthma ER Visits	Acute Bronch.	URS	LRS	Asthma Attacks	Work Loss Days	MRAD
St. Joseph	MO	114,839	8	4	5	1	14	154	155	129	1,060	5,480
St. Louis	MO-IL	2,819,493	280	159	170	59	547	6,010	5,890	5,060	43,900	227,000
State College	PA	129,802	9	7	7	3	19	212	203	276	2,800	14,400
Steubenville-Weirton	OH-WV	142,373	22	11	13	4	31	339	326	313	2,640	13,500
Stockton-Lodi	CA	583,401	1	1	1	0	3	39	38	30	239	1,240
Sumter	SC	122,049	13	9	8	4	36	406	382	316	2,710	13,900
Syracuse	NY	759,823	40	24	26	9	82	897	864	801	7,000	36,100
Tallahassee	FL	311,795	22	16	15	7	58	641	624	595	5,610	29,000
Tampa-St.Petersburg-Clearwater	FL	2,713,403	291	143	211	43	323	3,570	3,510	4,040	33,400	172,000
TerreHaute	IN	167,232	25	12	15	4	38	422	406	385	3,290	16,900
Texarkana	TX-AR	154,990	17	9	10	3	33	364	358	285	2,350	12,100
Toledo	OH	667,377	54	31	34	12	109	1,220	1,170	1,040	9,020	46,400
Topeka	KS	189,989	12	7	8	3	24	270	263	224	1,930	10,000
Tucson	AZ	982,093	2	1	1	0	4	44	44	40	351	1,830
Tulsa	OK	806,563	66	42	41	16	147	1,630	1,610	1,360	11,900	61,400
Tuscaloosa	AL	172,189	21	13	14	5	43	488	465	452	4,110	21,200
Tyler	TX	197,408	17	10	11	4	34	384	375	317	2,670	13,800
Utica-Rome	NY	321,925	18	10	12	3	30	332	324	306	2,580	13,300
Victoria	TX	98,674	3	2	2	1	9	97	96	69	574	2,970
Visalia-Tulare-Porterville	CA	379,467	1	0	0	0	2	20	20	13	102	531
Waco	TX	251,395	17	9	11	4	33	363	360	313	2,690	13,900
Washington	DC-MD-VA-WV	7,788,827	762	585	501	231	1,750	19,600	18,400	18,800	173,000	890,000
Waterloo-CedarFalls	IA	131,508	8	5	5	2	17	184	181	152	1,310	6,790

Exhibit A-1 PM-Related Adverse Health Effects by Metropolitan Statistical Area: "75 Percent Reduction" Scenario (cont.)

MSA	State	Population	Mortality	Chronic Bronch.	Hospital Admis.	Asthma ER Visits	Acute Bronch.	URS	LRS	Asthma Attacks	Work Loss Days	MRAD
Wausau	WI	131,430	4	3	4	1	13	142	141	107	897	4,640
WestPalmBeach-BocaRaton	FL	1,133,763	37	19	30	5	40	434	435	522	4,200	21,700
Wheeling	WV-OH	168,076	30	14	19	5	40	451	423	419	3,460	17,700
Wichita	KS	553,183	21	15	15	6	55	597	595	479	4,110	21,300
WichitaFalls	TX	171,656	7	4	4	2	14	156	156	132	1,140	5,930
Williamsport	PA	122,232	14	8	9	3	24	268	250	234	1,970	10,100
Wilmington	NC	221,013	23	15	16	5	42	463	456	459	4,110	21,200
Yakima	WA	253,518	2	2	2	1	7	75	74	55	435	2,260
Youngstown-Warren	OH	654,327	78	40	49	14	126	1,390	1,330	1,220	10,200	52,200
YubaCity	CA	147,736	0	0	0	0	1	9	9	7	59	308
Yuma	AZ	160,239	0	0	0	0	1	6	6	5	39	200

Exhibit A-2 PM-Related Adverse Health Effects by Metropolitan Statistical Area: All Power Plant Scenario

MSA	State	Population	Mortality	Chronic Bronch.	Hospital Admis.	Asthma ER Visits	Acute Bronch.	URS	LRS	Asthma Attacks	Work Loss Days	MRAD
Abilene	TX	158,508	7	4	5	2	17	182	180	152	1,300	6,720
Akron	OH	3,038,800	442	261	293	96	802	9,150	8,490	8,170	69,300	355,000
Albany	GA	150,035	22	14	13	6	64	733	685	517	4,290	22,000
Albany-Schenectady-Troy	NY	906,376	66	41	46	15	118	1,310	1,240	1,300	11,200	57,700
Albuquerque	NM	818,229	8	6	6	2	22	247	241	203	1,750	9,090
Alexandria	LA	149,570	15	8	9	3	34	376	366	277	2,290	11,800
Allentown-Bethlehem-Easton	PA	627,627	94	56	67	20	149	1,720	1,560	1,700	14,200	72,800
Altoona	PA	136,868	32	16	21	6	47	548	496	487	3,900	19,900
Amarillo	TX	246,598	8	5	5	2	21	234	228	180	1,540	7,960
Anniston	AL	139,054	37	20	22	8	65	753	689	656	5,660	28,800
Appleton-Oshkosh-Neenah	WI	358,203	21	16	16	6	58	655	627	526	4,500	23,200
Asheville	NC	241,640	69	38	47	13	95	1,120	1,010	1,150	9,640	49,100
Athens	GA	175,139	29	22	22	11	72	853	761	861	8,140	41,700
Atlanta	GA	3,964,069	647	550	432	237	1,820	21,400	19,300	18,700	169,000	866,000
Auburn-Opelika	AL	97,423	15	10	10	5	34	396	362	406	3,880	19,900
Augusta-Aiken	GA-SC	540,766	112	71	66	31	266	3,130	2,850	2,470	21,100	108,000
Austin-San Marcos	TX	1,116,410	41	39	31	17	140	1,560	1,510	1,390	12,900	66,700
Bakersfield	CA	665,377	5	4	4	2	17	210	188	151	1,120	5,790
Bangor	ME	191,687	6	4	4	1	13	141	138	125	1,110	5,720
Barnstable-Yarmouth	MA	201,278	21	11	16	3	24	276	253	296	2,340	12,000
Baton Rouge	LA	571,222	59	42	38	19	175	1,980	1,880	1,510	13,200	67,900
Beaumont-Port Arthur	TX	475,399	37	21	23	8	81	905	874	689	5,750	29,600
Bellingham	WA	169,697	0	0	0	0	1	11	11	10	86	447
Benton Harbor	MI	168,958	19	12	13	4	40	457	432	369	3,060	15,700
Billings	MT	146,333	1	1	1	0	3	35	35	28	238	1,240
Biloxi-Gulfport-Pascagoula	MS	354,653	49	30	30	13	116	1,330	1,250	1,040	8,790	45,100

Exhibit A-2 PM-Related Adverse Health Effects by Metropolitan Statistical Area: All Power Plant scenario (cont.)

MSA	State	Population	Mortality	Chronic Bronch.	Hospital Admis.	Asthma ER Visits	Acute Bronch.	URS	LRS	Asthma Attacks	Work Loss Days	MRAD
Binghamton	NY	287,626	31	18	21	7	56	628	579	581	4,970	25,500
Birmingham	AL	992,053	257	148	164	57	467	5,480	4,910	4,760	40,200	205,000
Bismarck	ND	89,362	2	2	2	1	6	67	64	50	411	2,130
Bloomington	IN	124,212	14	11	11	6	33	391	348	464	4,640	23,800
Bloomington-Normal	IL	140,591	16	12	12	5	40	460	429	437	4,030	20,700
Boise City	ID	454,755	2	2	2	1	7	75	75	55	462	2,400
Boston	MA-NH	6,991,988	454	302	320	113	839	9,420	8,820	9,540	84,000	432,000
Boulder-Longmont	CO	2,752,567	40	37	29	14	121	1,340	1,320	1,180	10,700	55,400
Brownsville-Harlingen-SanBenito	TX	346,141	6	5	5	2	28	301	306	189	1,440	7,420
Bryan-College Station	TX	159,612	7	6	6	4	25	287	269	301	2,990	15,500
Buffalo-Niagara Falls	NY	1,218,010	149	82	98	29	230	2,600	2,400	2,530	21,400	110,000
Burlington	VT	204,108	7	6	5	2	19	209	201	195	1,800	9,270
Canton-Massillon	OH	409,288	73	43	49	16	134	1,540	1,420	1,340	11,100	56,800
Casper	WY	79,731	1	1	1	0	3	32	31	23	191	989
Cedar Rapids	IA	178,822	15	11	11	4	37	411	398	359	3,160	16,300
Champaign-Urbana	IL	188,093	21	15	14	7	48	548	509	585	5,620	28,900
Charleston	WV	261,765	69	37	43	13	107	1,240	1,100	1,100	9,240	46,900
Charleston-North Charleston	SC	601,847	71	53	45	24	205	2,420	2,180	1,950	16,700	85,600
Charlotte-Gastonia-Rock Hill	NC-SC	1,460,744	298	206	201	83	614	7,290	6,480	6,780	59,200	302,000
Charlottesville	VA	158,737	29	19	20	8	52	601	543	658	6,140	31,400
Chattanooga	TN-GA	545,611	154	89	96	34	270	3,170	2,880	2,820	24,200	123,000
Cheyenne	WY	95,813	2	1	1	1	5	57	57	46	395	2,050
Chicago	IL	9,003,216	995	651	648	256	2,190	24,800	23,600	21,400	186,000	957,000
Chico-Paradise	CA	225,033	1	0	1	0	1	16	16	15	123	639

Exhibit A-2 PM-Related Adverse Health Effects by Metropolitan Statistical Area: All Power Plant scenario (cont.)

MSA	State	Population	Mortality	Chronic Bronch.	Hospital Admis.	Asthma ER Visits	Acute Bronch.	URS	LRS	Asthma Attacks	Work Loss Days	MRAD
Cincinnati	OH-KY-IN	1,947,621	377	236	248	95	820	9,590	8,580	7,870	66,400	339,000
Clarksville-Hopkinsville	TN-KY	202,112	33	24	23	12	88	1,020	935	923	8,240	42,200
Colorado Springs	CO	551,833	4	4	3	2	15	164	159	137	1,200	6,240
Columbia	SC	536,258	87	64	57	27	206	2,390	2,210	2,200	19,700	101,000
Columbia	MO	128,525	10	8	8	4	28	320	304	317	3,060	15,800
Columbus	OH	1,415,994	201	142	132	59	459	5,270	4,810	4,790	42,700	219,000
Columbus	GA-AL	350,300	75	43	45	18	149	1,730	1,570	1,490	12,700	64,800
Corpus Christi	TX	450,775	16	11	11	5	50	554	541	389	3,220	16,600
Corvallis	OR	110,085	1	1	1	0	2	26	26	26	245	1,270
Cumberland	MD-WV	119,023	33	15	21	5	40	469	419	462	3,780	19,200
Dallas	TX	5,307,754	369	304	247	129	1,100	12,400	11,900	10,500	94,100	486,000
Danville	VA	129,401	35	18	22	6	49	568	519	541	4,520	23,100
Davenport-Moline-RockIsland	IA-IL	377,234	51	30	34	11	104	1,180	1,130	952	7,920	40,800
Daytona Beach	FL	520,341	77	38	58	11	80	907	866	1,060	8,450	43,500
Dayton-Springfield	OH	1,005,479	181	109	115	42	349	4,030	3,690	3,520	30,300	155,000
Decatur	AL	151,257	34	22	22	9	73	835	787	707	6,040	30,900
Decatur	IL	128,361	25	14	17	5	46	531	503	445	3,680	18,900
DesMoines	IA	420,540	30	22	22	8	74	823	799	711	6,270	32,400
Detroit	MI	5,463,996	527	343	343	134	1,140	12,800	12,100	11,200	96,400	496,000
Dothan	AL	158,661	26	16	16	7	58	665	632	544	4,610	23,700
Dover	DE	125,701	16	11	11	5	39	459	411	388	3,270	16,700
Dubuque	IA	58,471	7	4	5	1	14	164	157	127	1,050	5,400
Duluth-Superior	MN-WI	277,005	11	6	7	2	18	198	197	170	1,410	7,280
EauClaire	WI	156,214	10	6	7	2	22	250	243	206	1,750	9,040

Exhibit A-2 PM-Related Adverse Health Effects by Metropolitan Statistical Area: All Power Plant scenario (cont.)

MSA	State	Population	Mortality	Chronic Bronch.	Hospital Admis.	Asthma ER Visits	Acute Bronch.	URS	LRS	Asthma Attacks	Work Loss Days	MRAD
Elkhart-Goshen	IN	179,988	18	13	13	5	48	533	514	431	3,620	18,600
Elmira	NY	101,706	13	7	9	3	23	266	242	227	1,880	9,600
ElPaso	TX	787,748	4	4	4	2	20	219	221	155	1,270	6,600
Enid	OK	64,850	6	3	4	1	11	120	116	94	777	4,010
Erie	PA	286,310	36	20	24	8	68	776	714	668	5,560	28,400
Eugene-Springfield	OR	371,712	4	2	3	1	8	87	85	76	665	3,450
Evansville-Henderson	IN-KY	316,843	78	44	51	17	141	1,670	1,490	1,410	11,700	59,500
Fargo-Moorhead	ND-MN	167,977	4	3	3	1	11	122	120	108	968	5,020
Fayetteville	NC	356,984	47	37	29	19	154	1,800	1,640	1,520	13,600	69,900
Fayetteville-Springdale-Rogers	AR	251,086	37	24	29	9	78	877	858	792	6,770	34,900
Flagstaff	AZ-UT	147,812	1	1	1	0	4	49	45	34	280	1,450
Florence	AL	155,821	43	24	29	9	72	838	775	768	6,550	33,500
Florence	SC	141,037	27	15	16	6	59	691	635	524	4,320	22,100
Fort Collins-Loveland	CO	260,092	5	4	4	2	15	171	167	148	1,350	6,990
Fort Lauderdale	FL	1,555,266	68	39	55	12	84	946	915	1,100	8,870	45,800
Fort Myers-Cape Coral	FL	447,165	33	18	29	5	37	415	394	493	3,830	19,700
Fort Pierce-Port St. Lucie	FL	327,920	25	13	21	4	29	340	317	369	2,820	14,500
Fort Smith	AR-OK	217,070	37	20	23	8	73	822	802	661	5,500	28,300
Fort Walton Beach	FL	184,439	22	17	15	7	57	646	619	584	5,250	27,000
Fort Wayne	IN	515,716	60	40	42	16	151	1,720	1,620	1,330	11,000	56,700
Fresno	CA	922,367	4	3	3	1	14	163	157	115	909	4,710
Gadsden	AL	118,516	41	20	25	7	60	712	643	624	5,130	26,100
Gainesville	FL	239,196	23	16	16	8	51	586	553	614	5,800	29,900
Glens Falls	NY	88,874	6	4	4	1	11	123	118	111	942	4,840

Exhibit A-2 PM-Related Adverse Health Effects by Metropolitan Statistical Area: All Power Plant scenario (cont.)

MSA	State	Population	Mortality	Chronic Bronch.	Hospital Admis.	Asthma ER Visits	Acute Bronch.	URS	LRS	Asthma Attacks	Work Loss Days	MRAD
Goldsboro	NC	130,660	27	18	17	7	58	671	629	596	5,220	26,800
Grand Forks	ND-MN	113,333	2	1	1	1	5	60	60	50	437	2,270
Grand Junction	CO	128,755	2	1	1	0	4	44	43	34	280	1,450
Grand Rapids-Muskegon-Holland	MI	1,000,106	72	52	53	21	203	2,290	2,160	1,790	15,000	77,200
GreatFalls	MT	99,816	0	0	0	0	1	8	8	6	53	277
Green Bay	WI	218,748	12	9	9	4	33	367	360	305	2,650	13,700
Greensboro--Winston-Salem--High Point	NC	1,343,693	309	201	210	77	535	6,280	5,700	6,380	56,000	286,000
Greenville	NC	135,297	23	15	15	7	49	564	528	538	4,860	24,900
Greenville-Spartanburg-Anderson	SC	985,653	226	139	148	54	422	4,950	4,480	4,520	39,100	200,000
Harrisburg-Lebanon-Carlisle	PA	597,604	116	70	79	26	198	2,300	2,080	2,190	18,800	96,000
Hartford	CT	1,326,689	110	72	77	27	194	2,190	2,020	2,240	19,700	101,000
Hattiesburg	MS	114,222	16	9	10	4	38	437	403	341	2,900	14,900
Hickory-Morganton-Lenoir	NC	369,838	85	56	58	22	157	1,870	1,680	1,790	15,400	78,700
Houma	LA	195,895	15	11	10	5	51	573	545	385	3,220	16,600
Houston	TX	4,913,333	201	178	132	76	705	7,890	7,650	6,140	54,400	281,000
Huntington-Ashland	WV-KY-OH	337,895	86	45	52	17	140	1,620	1,450	1,400	11,700	59,600
Huntsville	AL	340,441	62	48	42	20	150	1,760	1,600	1,620	14,700	75,300
Indianapolis	IN	1,572,962	250	161	161	64	531	6,170	5,650	5,300	45,400	233,000
Iowa City	IA	101,591	5	5	5	3	17	191	182	214	2,170	11,200
Jackson	MS	452,696	62	38	38	16	150	1,700	1,620	1,320	11,300	58,200
Jackson	TN	112,035	23	13	15	5	43	496	465	416	3,480	17,800
Jackson	MI	155,830	16	10	10	4	32	357	341	308	2,660	13,700
Jacksonville	FL	1,180,206	131	87	84	35	276	3,250	2,990	2,910	24,500	126,000

Exhibit A-2 PM-Related Adverse Health Effects by Metropolitan Statistical Area: All Power Plant scenario (cont.)

MSA	State	Population	Mortality	Chronic Bronch.	Hospital Admis.	Asthma ER Visits	Acute Bronch.	URS	LRS	Asthma Attacks	Work Loss Days	MRAD
Jacksonville	NC	190,295	14	13	10	9	55	640	585	657	6,420	33,100
Jamestown	NY	144,849	21	11	14	4	35	398	363	342	2,780	14,200
Janesville-Beloit	WI	161,217	16	11	11	4	37	423	405	345	2,910	15,000
Johnson City-Kingsport-Bristol	TN-VA	537,478	154	84	98	30	217	2,540	2,320	2,580	22,200	113,000
Johnstown	PA	253,500	61	31	42	10	87	1,020	918	932	7,310	37,200
Jonesboro	AR	83,910	13	8	8	3	25	290	278	263	2,320	11,900
Joplin	MO	155,108	30	15	19	6	51	579	559	485	4,020	20,700
Kalamazoo-Battle Creek	MI	441,064	41	27	28	11	92	1,040	982	886	7,710	39,700
Kansas City	MO-KS	1,791,964	194	127	126	49	439	4,960	4,760	4,100	35,500	183,000
Killeen-Temple	TX	332,715	14	12	11	6	51	568	550	474	4,210	21,800
Knoxville	TN	737,786	190	118	130	44	321	3,840	3,420	3,730	32,200	164,000
Kokomo	IN	109,357	16	11	11	4	35	398	380	335	2,840	14,600
LaCrosse	WI-MN	131,031	11	7	8	3	23	262	251	226	1,960	10,100
Lafayette	LA	382,013	29	19	19	8	87	971	934	685	5,710	29,400
Lafayette	IN	184,425	24	15	17	7	50	564	532	581	5,390	27,700
Lake Charles	LA	184,810	14	9	9	4	37	411	397	297	2,500	12,900
Lakeland-Winter Haven	FL	526,755	68	38	52	13	102	1,210	1,110	1,150	8,830	45,400
Lancaster	PA	439,469	84	53	60	21	178	2,070	1,860	1,760	14,600	74,500
Lansing-East Lansing	MI	456,760	33	25	23	11	94	1,060	1,010	892	7,990	41,200
Laredo	TX	174,981	2	2	2	1	15	163	165	102	790	4,080
Las Cruces	NM	180,761	1	1	1	0	5	53	53	39	332	1,730
Las Vegas	NV-AZ	1,467,639	18	13	13	5	35	445	386	423	3,330	17,200
Lawrence	KS	95,395	6	5	5	2	16	184	174	199	1,990	10,300
Lawton	OK	125,946	7	5	4	2	19	212	210	171	1,520	7,840

Exhibit A-2 PM-Related Adverse Health Effects by Metropolitan Statistical Area: All Power Plant scenario (cont.)

MSA	State	Population	Mortality	Chronic Bronch.	Hospital Admis.	Asthma ER Visits	Acute Bronch.	URS	LRS	Asthma Attacks	Work Loss Days	MRAD
Lewiston-Auburn	ME	112,945	5	3	3	1	10	111	106	96	816	4,210
Lexington	KY	454,516	95	65	63	28	204	2,390	2,180	2,250	20,300	104,000
Lima	OH	166,864	24	14	16	5	51	581	548	455	3,720	19,100
Lincoln	NE	241,281	13	10	10	4	32	359	351	335	3,080	15,900
Little Rock-North Little Rock	AR	610,612	85	53	53	21	184	2,090	2,000	1,750	15,200	78,400
Longview-Marshall	TX	245,628	37	20	23	8	76	863	825	658	5,350	27,500
Los Angeles-Long Beach	CA	17,763,602	184	156	143	65	520	6,080	5,730	5,440	45,400	236,000
Louisville	KY-IN	1,072,938	256	152	162	59	480	5,670	5,080	4,870	41,200	210,000
Lubbock	TX	294,525	7	5	5	2	20	218	214	182	1,620	8,420
Lynchburg	VA	233,684	54	32	36	12	88	1,040	933	1,010	8,650	44,100
Macon	GA	391,495	76	47	47	20	169	1,970	1,770	1,600	13,600	69,600
Madison	WI	417,101	28	24	22	10	75	836	803	840	7,980	41,200
Mansfield	OH	188,285	32	18	20	7	60	683	646	574	4,810	24,600
McAllen-Edinburg-Mission	TX	501,759	7	7	7	3	40	439	444	265	1,980	10,200
Medford-Ashland	OR	191,802	2	1	1	0	3	30	30	26	214	1,110
Melbourne-Titusville-Palm Bay	FL	522,202	46	29	36	10	72	822	769	859	7,250	37,400
Memphis	TN-AR-MS	1,253,499	185	110	107	46	412	4,720	4,460	3,780	32,500	167,000
Merced	CA	216,576	1	1	1	0	4	46	43	30	228	1,180
Milwaukee-Waukesha	WI	1,820,294	163	104	110	40	357	4,030	3,830	3,370	28,700	148,000
Minneapolis-St.Paul	MN-WI	2,942,826	135	113	99	45	392	4,420	4,240	3,750	33,200	172,000
Missoula	MT	102,046	0	0	0	0	1	9	9	8	70	366
Mobile	AL	557,578	92	56	61	22	206	2,350	2,220	1,860	15,300	78,600
Modesto	CA	458,480	3	2	2	1	9	105	95	75	569	2,940
Monroe	LA	159,432	16	10	10	4	41	461	445	344	2,850	14,700

Exhibit A-2 PM-Related Adverse Health Effects by Metropolitan Statistical Area: All Power Plant scenario (cont.)

MSA	State	Population	Mortality	Chronic Bronch.	Hospital Admis.	Asthma ER Visits	Acute Bronch.	URS	LRS	Asthma Attacks	Work Loss Days	MRAD
Montgomery	AL	340,717	73	43	45	18	156	1,820	1,660	1,460	12,400	63,100
Muncie	IN	133,491	20	11	13	5	33	379	357	387	3,510	18,000
Myrtle Beach	SC	172,374	29	18	19	7	53	613	569	565	4,930	25,300
Naples	FL	194,829	10	6	9	2	13	148	141	170	1,370	7,050
Nashville	TN	1,228,389	260	175	167	71	558	6,530	5,970	5,800	51,200	262,000
New London-Norwich	CT-RI	109,790	8	6	6	2	17	195	179	187	1,660	8,500
New Orleans	LA	1,411,716	152	89	89	36	340	3,830	3,670	2,990	25,200	130,000
New York	NY	20,578,316	2,290	1,490	1,580	546	4,020	45,700	42,700	46,200	402,000	2,060,000
Norfolk-VirginiaBeach-NewportNews	VA-NC	1,750,317	217	158	144	69	555	6,460	5,870	5,580	48,600	249,000
Ocala	FL	259,484	43	21	32	6	52	598	563	606	4,690	24,100
Odessa-Midland	TX	295,814	6	4	4	2	19	207	203	143	1,200	6,190
Oklahoma City	OK	1,091,027	81	51	50	20	182	2,030	1,980	1,690	14,800	76,500
Omaha	NE-IA	702,937	52	36	36	14	133	1,490	1,450	1,210	10,400	53,800
Orlando	FL	1,590,485	152	108	116	41	313	3,620	3,380	3,490	29,900	154,000
Owensboro	KY	97,223	24	14	16	6	49	573	524	463	3,820	19,500
Panama City	FL	166,259	26	17	17	6	53	605	570	538	4,680	24,000
Parkersburg-Marietta	WV-OH	155,110	36	20	23	7	62	717	638	621	5,160	26,200
Pensacola	FL	459,703	72	46	46	18	150	1,720	1,610	1,510	13,200	67,900
Peoria-Pekin	IL	366,759	60	36	41	13	121	1,380	1,310	1,140	9,440	48,500
Philadelphia	PA-NJ	6,414,340	997	593	654	225	1,720	20,300	18,100	19,000	158,000	808,000
Phoenix-Mesa	AZ	3,298,411	30	23	24	9	75	866	818	751	6,130	31,800
Pine Bluff	AR	102,116	19	9	11	4	35	405	383	309	2,530	13,000
Pittsburgh	PA	2,459,427	585	309	395	105	765	9,030	8,020	9,210	75,500	385,000

Exhibit A-2 PM-Related Adverse Health Effects by Metropolitan Statistical Area: All Power Plant scenario (cont.)

MSA	State	Population	Mortality	Chronic Bronch.	Hospital Admis.	Asthma ER Visits	Acute Bronch.	URS	LRS	Asthma Attacks	Work Loss Days	MRAD
Pittsfield	MA	149,519	14	7	9	2	19	215	203	215	1,800	9,250
Pocatello	ID	103,235	1	0	0	0	2	23	22	14	110	570
Portland	ME	257,111	12	8	8	3	22	247	239	245	2,170	11,200
Portland-Vancouver	OR-WA	2,371,025	32	23	23	9	76	859	832	729	6,190	32,100
Providence-FallRiver-Warwick	RI-MA	930,547	80	47	57	17	128	1,430	1,340	1,470	12,600	64,900
Provo-Orem	UT	379,915	2	2	2	1	13	147	138	83	602	3,110
Pueblo	CO	170,854	3	2	2	1	7	77	76	63	512	2,650
Punta Gorda	FL	129,773	16	8	16	2	12	137	125	199	1,370	7,020
Raleigh-Durham-ChapelHill	NC	1,088,464	174	139	125	58	392	4,590	4,170	4,700	43,300	222,000
Rapid City	SD	90,759	1	1	1	0	4	42	41	32	271	1,410
Reading	PA	330,183	62	37	45	13	99	1,170	1,040	1,130	9,290	47,500
Redding	CA	178,718	1	0	1	0	2	19	19	15	123	637
Reno	NV	444,290	1	1	1	0	2	26	21	26	204	1,060
Richland-Kennewick-Pasco	WA	202,015	2	2	1	1	7	74	73	52	428	2,220
Richmond-Petersburg	VA	1,053,301	203	128	128	50	369	4,310	3,870	4,100	36,000	184,000
Roanoke	VA	276,309	70	39	44	13	97	1,110	1,040	1,150	9,970	50,900
Rochester	NY	1,075,023	90	59	62	23	185	2,090	1,940	1,900	16,300	84,000
Rochester	MN	125,308	6	6	5	2	21	234	223	188	1,640	8,490
Rockford	IL	352,573	39	25	27	10	85	964	918	807	6,860	35,300
Rocky Mount	NC	167,594	38	22	23	8	73	840	792	706	5,900	30,200
Sacramento	CA	1,808,831	5	4	4	2	14	161	154	136	1,180	6,110
Saginaw-BayCity-Midland	MI	428,009	34	22	23	9	80	899	859	723	6,080	31,300
Salinas	CA	463,926	1	1	1	0	4	48	46	41	346	1,800
Salt Lake City-Ogden	UT	1,558,644	10	10	9	5	55	705	597	410	2,760	14,200

Exhibit A-2 PM-Related Adverse Health Effects by Metropolitan Statistical Area: All Power Plant scenario (cont.)

MSA	State	Population	Mortality	Chronic Bronch.	Hospital Admis.	Asthma ER Visits	Acute Bronch.	URS	LRS	Asthma Attacks	Work Loss Days	MRAD
San Angelo	TX	129,131	5	3	3	1	11	125	123	102	863	4,470
San Antonio	TX	1,735,324	93	69	67	29	277	3,090	3,010	2,410	20,500	106,000
San Diego	CA	3,040,458	20	16	16	7	51	575	554	552	4,840	25,100
San Francisco	CA	7,613,985	20	17	15	6	48	547	520	541	4,760	24,700
San Luis Obispo-Atascadero-Paso Robles	CA	265,215	1	1	1	0	2	20	19	20	175	908
Santa Barbara-Santa Maria-Lompoc	CA	452,536	1	1	1	0	3	28	27	28	248	1,290
Santa Fe	NM	163,156	1	1	1	0	4	48	47	40	356	1,850
Sarasota-Bradenton	FL	655,162	105	52	98	13	84	1,050	905	1,390	9,340	47,800
Savannah	GA	343,725	46	29	31	12	104	1,220	1,120	992	8,180	42,100
Scranton--Wilkes-Barre--Hazleton	PA	674,477	122	57	79	19	143	1,630	1,490	1,680	13,700	69,700
Seattle-Bellevue-Everett	WA	3,965,480	23	19	18	7	60	684	652	613	5,310	27,500
Sharon	PA	121,878	21	11	14	4	30	338	316	326	2,660	13,600
Sheboygan	WI	116,523	10	6	7	2	22	243	236	198	1,620	8,330
Sherman-Denison	TX	127,379	20	10	13	3	32	358	350	308	2,520	13,000
Shreveport-BossierCity	LA	413,424	49	28	30	11	106	1,200	1,150	914	7,530	38,800
Sioux City	IA-NE	126,860	10	6	7	2	23	259	250	192	1,550	8,000
Sioux Falls	SD	163,717	7	5	5	2	19	213	209	170	1,460	7,550
South Bend	IN	262,727	32	19	22	7	62	702	672	619	5,240	26,900
Spokane	WA	482,077	3	2	2	1	8	87	85	73	605	3,140
Springfield	MO	301,726	41	24	27	9	76	856	838	789	6,980	36,000
Springfield	IL	206,972	40	25	28	9	79	915	850	773	6,510	33,400
Springfield	MA	225,475	14	10	11	4	27	299	277	322	2,950	15,200
St. Cloud	MN	180,320	6	4	5	2	20	216	211	163	1,390	7,210

Exhibit A-2 PM-Related Adverse Health Effects by Metropolitan Statistical Area: All Power Plant scenario (cont.)

MSA	State	Population	Mortality	Chronic Bronch.	Hospital Admis.	Asthma ER Visits	Acute Bronch.	URS	LRS	Asthma Attacks	Work Loss Days	MRAD
St. Joseph	MO	114,839	14	7	9	2	23	259	256	218	1,770	9,140
St. Louis	MO-IL	2,819,493	494	285	309	109	947	10,900	10,200	9,200	77,300	397,000
State College	PA	129,802	13	11	11	5	29	326	298	424	4,210	21,600
Steubenville-Weirton	OH-WV	142,373	34	17	21	6	46	533	487	492	4,030	20,500
Stockton-Lodi	CA	583,401	3	2	2	1	10	113	105	86	664	3,440
Sumter	SC	122,049	20	14	13	6	54	648	572	505	4,130	21,100
Syracuse	NY	759,823	65	41	44	16	132	1,500	1,390	1,340	11,400	58,700
Tallahassee	FL	311,795	33	25	23	11	85	974	921	905	8,410	43,300
Tampa-St.Petersburg-Clearwater	FL	2,713,403	494	271	409	86	549	7,200	5,960	8,070	57,200	293,000
TerreHaute	IN	167,232	44	21	28	8	65	765	700	696	5,830	29,800
Texarkana	TX-AR	154,990	29	15	18	6	54	622	593	487	3,960	20,400
Toledo	OH	667,377	87	51	56	21	176	2,020	1,870	1,730	14,700	75,300
Topeka	KS	189,989	21	12	13	4	40	453	435	376	3,220	16,700
Tucson	AZ	982,093	10	6	7	2	20	229	224	210	1,770	9,200
Tulsa	OK	806,563	108	69	68	27	236	2,680	2,570	2,230	19,300	99,300
Tuscaloosa	AL	172,189	31	19	21	8	64	743	684	688	6,170	31,600
Tyler	TX	197,408	28	16	19	6	56	642	607	530	4,400	22,600
Utica-Rome	NY	321,925	29	16	19	6	47	531	502	489	4,060	20,800
Victoria	TX	98,674	5	3	3	1	15	163	159	116	955	4,930
Visalia-Tulare-Porterville	CA	379,467	2	1	1	1	6	71	68	47	355	1,840
Waco	TX	251,395	27	15	18	6	53	601	580	519	4,390	22,600
Washington	DC-MD-VA-WV	7,788,827	1,140	881	764	354	2,560	29,800	26,900	28,600	257,000	1,320,000
Waterloo-CedarFalls	IA	131,508	13	7	9	3	26	295	286	244	2,090	10,800

Exhibit A-2 PM-Related Adverse Health Effects by Metropolitan Statistical Area: All Power Plant scenario (cont.)

MSA	State	Population	Mortality	Chronic Bronch.	Hospital Admis.	Asthma ER Visits	Acute Bronch.	URS	LRS	Asthma Attacks	Work Loss Days	MRAD
Wausau	WI	131,430	7	5	6	2	21	238	231	178	1,490	7,670
WestPalmBeach-BocaRaton	FL	1,133,763	59	32	50	9	65	723	698	870	6,790	35,000
Wheeling	WV-OH	168,076	46	22	29	7	60	699	624	650	5,240	26,600
Wichita	KS	553,183	36	25	26	10	92	1,020	1,000	822	6,990	36,100
WichitaFalls	TX	171,656	12	7	7	3	24	263	257	222	1,910	9,860
Williamsport	PA	122,232	21	11	14	4	35	403	361	353	2,900	14,800
Wilmington	NC	221,013	34	22	24	8	61	701	668	693	6,100	31,300
Yakima	WA	253,518	3	2	2	1	9	97	96	71	562	2,910
Youngstown-Warren	OH	654,327	120	63	77	22	191	2,200	2,000	1,920	15,600	79,500
YubaCity	CA	147,736	0	0	0	0	1	14	14	10	86	446
Yuma	AZ	160,239	1	1	1	0	3	38	37	29	219	1,130

Exhibit A-3 Population and Counties in Metropolitan Statistical Areas

Metropolitan Statistical Area	County	State	Population 2007
Abilene	Taylor	Texas	158,508
Akron	Ashtabula	Ohio	114,693
	Cuyahoga	Ohio	1,472,729
	Geauga	Ohio	97,289
	Lake	Ohio	246,524
	Lorain	Ohio	296,573
	Medina	Ohio	119,436
	Portage	Ohio	134,768
	Summit	Ohio	556,788
Albany	Dougherty	Georgia	113,529
	Lee	Georgia	36,506
Albany-Schenectady-Troy	Albany	New York	313,200
	Montgomery	New York	51,366
	Rensselaer	New York	97,794
	Saratoga	New York	254,505
	Schenectady	New York	157,771
	Schoharie	New York	31,740
Albuquerque	Bernalillo	New Mexico	657,395
	Sandoval	New Mexico	94,682
	Valencia	New Mexico	66,151
Alexandria	Rapides	Louisiana	149,570
Allentown-Bethlehem-Easton	Carbon	Pennsylvania	45,046
	Lehigh	Pennsylvania	340,129
	Northampton	Pennsylvania	242,452
Altoona	Blair	Pennsylvania	136,868
Amarillo	Potter	Texas	83,412
	Randall	Texas	163,186
Anniston	Calhoun	Alabama	139,054
Appleton-Oshkosh-Neenah	Calumet	Wisconsin	78,116
	Outagamie	Wisconsin	123,912
	Winnebago	Wisconsin	156,175
Asheville	Buncombe	North Carolina	220,145
	Madison	North Carolina	21,495
Athens	Clarke	Georgia	131,358
	Madison	Georgia	27,221
	Oconee	Georgia	16,560
Atlanta	Barrow	Georgia	39,483
	Bartow	Georgia	85,852
	Carroll	Georgia	99,306
	Cherokee	Georgia	173,706

Exhibit A-3 Population and Counties in Metropolitan Statistical Areas (cont.)

Metropolitan Statistical Area	County	State	Population 2007
	Clayton	Georgia	289,182
Atlanta (cont.)	Cobb	Georgia	594,855
	Coweta	Georgia	78,935
	De Kalb	Georgia	876,505
	Douglas	Georgia	99,290
	Fayette	Georgia	78,445
	Forsyth	Georgia	60,009
	Fulton	Georgia	651,408
	Gwinnett	Georgia	478,156
	Henry	Georgia	53,561
	Newton	Georgia	62,938
	Paulding	Georgia	49,329
	Pickens	Georgia	20,330
	Rockdale	Georgia	63,176
	Spalding	Georgia	67,451
	Walton	Georgia	42,152
Auburn-Opelika	Lee	Alabama	97,423
Augusta-Aiken	Aiken	South Carolina	124,816
	Columbia	Georgia	116,414
	Edgefield	South Carolina	24,063
	McDuffie	Georgia	24,766
	Richmond	Georgia	250,708
Austin-San Marcos	Bastrop	Texas	53,437
	Caldwell	Texas	34,226
	Hays	Texas	90,853
	Travis	Texas	763,121
	Williamson	Texas	174,775
Bakersfield	Kern	California	665,377
Bangor	Penobscot	Maine	156,649
	Waldo	Maine	35,039
Barnstable-Yarmouth	Barnstable	Massachusetts	201,278
Baton Rouge	Ascension	Louisiana	58,503
	East Baton Rouge	Louisiana	436,879
	Livingston	Louisiana	65,188
	West Baton Rouge	Louisiana	10,653
Beaumont-Port Arthur	Hardin	Texas	56,299
	Jefferson	Texas	311,017
	Orange	Texas	108,082

Exhibit A-3 Population and Counties in Metropolitan Statistical Areas (cont.)

Metropolitan Statistical Area	County	State	Population 2007
Bellingham	Whatcom	Washington	169,697
Benton Harbor	Berrien	Michigan	168,958
Billings	Yellowstone	Montana	146,333
Biloxi-Gulfport-Pascagoula	Hancock	Mississippi	32,949
Biloxi-Gulfport-Pascagoula (cont.)	Harrison	Mississippi	185,074
	Jackson	Mississippi	136,630
Binghamton	Broome	New York	217,197
	Tioga	New York	70,429
	Blount	Alabama	47,374
	Jefferson	Alabama	754,478
	Shelby	Alabama	128,871
	St. Clair	Alabama	61,330
Bismarck	Burleigh	North Dakota	65,601
	Morton	North Dakota	23,761
Bloomington	Monroe	Indiana	124,212
Bloomington-Normal	McLean	Illinois	140,591
Boise City	Ada	Idaho	311,776
	Canyon	Idaho	142,980
Boston	Bristol	Massachusetts	545,686
	Essex	Massachusetts	657,320
	Hampden	Massachusetts	481,485
	Hillsborough	New Hampshire	374,566
	Merrimack	New Hampshire	132,658
	Middlesex	Massachusetts	1,762,715
	Norfolk	Massachusetts	608,114
	Plymouth	Massachusetts	419,137
	Rockingham	New Hampshire	308,542
	Strafford	New Hampshire	128,780
	Suffolk	Massachusetts	551,493
	Windham	Connecticut	103,093
	Worcester	Massachusetts	735,339
	York	Maine	183,060
Boulder-Longmont	Adams	Colorado	223,223
	Arapahoe	Colorado	374,654
	Boulder	Colorado	289,971
	Denver	Colorado	565,002
	Douglas	Colorado	138,938
	Jefferson	Colorado	978,473

Exhibit A-3 Population and Counties in Metropolitan Statistical Areas (cont.)

Metropolitan Statistical Area	County	State	Population 2007
	Weld	Colorado	182,308
Brownsville-Harlingen-San Benito	Cameron	Texas	346,141
Bryan-College Station	Brazos	Texas	159,612
Buffalo-Niagara Falls	Erie	New York	1,004,933
	Niagara	New York	213,077
Burlington	Chittenden	Vermont	149,952
	Franklin	Vermont	48,338
Burlington (cont.)	Grand Isle	Vermont	5,819
Canton-Massillon	Carroll	Ohio	33,196
	Stark	Ohio	376,092
Casper	Natrona	Wyoming	79,731
Cedar Rapids	Linn	Iowa	178,822
Champaign-Urbana	Champaign	Illinois	188,093
Charleston	Kanawha	West Virginia	223,022
	Putnam	West Virginia	38,743
Charleston-North Charleston	Berkeley	South Carolina	170,398
	Charleston	South Carolina	310,803
	Dorchester	South Carolina	120,646
Charlotte-Gastonia-Rock Hill	Cabarrus	North Carolina	104,485
	Gaston	North Carolina	223,097
	Lincoln	North Carolina	64,096
	Mecklenburg	North Carolina	660,626
	Rowan	North Carolina	157,734
	Union	North Carolina	99,578
	York	South Carolina	151,129
Charlottesville	Albemarle	Virginia	61,408
	Charlottesville	Virginia	72,321
	Fluvanna	Virginia	14,495
	Greene	Virginia	10,513
Chattanooga	Catoosa	Georgia	65,830
	Dade	Georgia	19,923
	Hamilton	Tennessee	356,950
	Marion	Tennessee	29,631
	Walker	Georgia	73,278
Cheyenne	Laramie	Wyoming	95,813
Chicago	Cook	Illinois	5,546,833
	De Kalb	Illinois	88,527
	Du Page	Illinois	843,409

Exhibit A-3 Population and Counties in Metropolitan Statistical Areas (cont.)

Metropolitan Statistical Area	County	State	Population 2007
	Grundy	Illinois	34,819
	Kane	Illinois	297,662
	Kankakee	Illinois	105,316
	Kendall	Illinois	76,023
	Kenosha	Wisconsin	176,599
	Lake	Illinois	532,251
	Lake	Indiana	530,431
	McHenry	Illinois	200,771
	Porter	Indiana	173,552
	Will	Illinois	397,023
Chico-Paradise	Butte	California	225,033
Cincinnati	Boone	Kentucky	43,493
	Brown	Ohio	34,661
	Butler	Ohio	268,789
	Campbell	Kentucky	22,401
	Clermont	Ohio	147,362
	Dearborn	Indiana	40,286
	Gallatin	Kentucky	6,423
	Grant	Kentucky	18,522
	Hamilton	Ohio	992,171
	Kenton	Kentucky	193,944
	Ohio	Indiana	6,503
	Pendleton	Kentucky	14,906
	Warren	Ohio	158,160
Clarksville-Hopkinsville	Christian	Kentucky	76,936
	Montgomery	Tennessee	125,176
Colorado Springs	El Paso	Colorado	551,833
Columbia	Boone	Missouri	128,525
	Lexington	South Carolina	245,190
	Richland	South Carolina	291,068
Columbus	Chattahoochee	Georgia	3,804
	Harris	Georgia	22,268
	Muscogee	Georgia	272,628
	Russell	Alabama	51,600
	Delaware	Ohio	62,945
	Fairfield	Ohio	130,418
	Franklin	Ohio	1,008,368
	Licking	Ohio	122,459

Exhibit A-3 Population and Counties in Metropolitan Statistical Areas (cont.)

Metropolitan Statistical Area	County	State	Population 2007
	Madison	Ohio	40,363
	Pickaway	Ohio	51,441
Corpus Christi	Nueces	Texas	381,535
	San Patricio	Texas	69,240
Corvallis	Benton	Oregon	110,085
Cumberland	Allegany	Maryland	97,743
	Mineral	West Virginia	21,279
Dallas	Collin	Texas	370,337
	Dallas	Texas	2,261,393
	Denton	Texas	420,130
	Ellis	Texas	105,923
	Henderson	Texas	83,441
	Hood	Texas	39,335
Dallas (cont.)	Hunt	Texas	78,605
	Johnson	Texas	116,255
	Kaufman	Texas	62,115
	Parker	Texas	96,636
	Rockwall	Texas	43,954
	Tarrant	Texas	1,629,631
Danville	Danville	Virginia	62,950
	Pittsylvania	Virginia	66,451
Davenport-Moline-Rock Island	Henry	Illinois	48,603
	Rock Island	Illinois	180,208
	Scott	Iowa	148,423
Daytona Beach	Flagler	Florida	36,080
	Volusia	Florida	484,261
Dayton-Springfield	Clark	Ohio	167,034
	Greene	Ohio	142,418
	Miami	Ohio	102,899
	Montgomery	Ohio	593,128
Decatur	Lawrence	Alabama	39,734
	Morgan	Alabama	111,524
	Macon	Illinois	128,361
Des Moines	Dallas	Iowa	30,914
	Polk	Iowa	343,757
	Warren	Iowa	45,868
Detroit	Genesee	Michigan	456,229
	Lapeer	Michigan	82,123

Exhibit A-3 Population and Counties in Metropolitan Statistical Areas (cont.)

Metropolitan Statistical Area	County	State	Population 2007
	Lenawee	Michigan	102,224
	Livingston	Michigan	132,485
	Macomb	Michigan	735,549
	Monroe	Michigan	135,218
	Oakland	Michigan	912,937
	St. Clair	Michigan	159,019
	Washtenaw	Michigan	275,777
	Wayne	Michigan	2,472,434
Dothan	Dale	Alabama	60,950
	Houston	Alabama	97,711
Dover	Kent	Delaware	125,701
Dubuque	Dubuque	Iowa	58,471
Duluth-Superior	Douglas	Wisconsin	35,801
	St. Louis	Minnesota	241,204
Eau Claire	Chippewa	Wisconsin	67,717
	Eau Claire	Wisconsin	88,496
El Paso	El Paso	Texas	787,748
Elkhart-Goshen	Elkhart	Indiana	179,988
Elmira	Chemung	New York	101,706
Enid	Garfield	Oklahoma	64,850
Erie	Erie	Pennsylvania	286,310
Eugene-Springfield	Lane	Oregon	371,712
Evansville-Henderson	Henderson	Kentucky	48,549
	Posey	Indiana	26,813
	Vanderburgh	Indiana	190,138
	Warrick	Indiana	51,344
Fargo-Moorhead	Cass	North Dakota	121,915
	Clay	Minnesota	46,061
Fayetteville	Cumberland	North Carolina	356,984
Fayetteville-Springdale-Rogers	Benton	Arkansas	118,151
	Washington	Arkansas	132,935
Flagstaff	Coconino	Arizona	139,206
	Kane	Utah	8,606
Florence	Colbert	Alabama	57,661
	Lauderdale	Alabama	98,161
	Florence	South Carolina	141,037
Fort Collins-Loveland	Larimer	Colorado	260,092
Fort Lauderdale	Broward	Florida	1,555,266

Exhibit A-3 Population and Counties in Metropolitan Statistical Areas (cont.)

Metropolitan Statistical Area	County	State	Population 2007
Fort Myers-Cape Coral	Lee	Florida	447,165
Fort Pierce-Port St. Lucie	Martin	Florida	136,078
	St. Lucie	Florida	191,841
Fort Smith	Crawford	Arkansas	75,305
	Sebastian	Arkansas	104,320
	Sequoyah	Oklahoma	37,445
Fort Walton Beach	Okaloosa	Florida	184,439
Fort Wayne	Adams	Indiana	35,395
	Allen	Indiana	342,698
	De Kalb	Indiana	42,748
	Huntington	Indiana	40,419
	Wells	Indiana	23,700
	Whitley	Indiana	30,756
Fresno	Fresno	California	815,757
	Madera	California	106,610
Gadsden	Etowah	Alabama	118,516
Gainesville	Alachua	Florida	239,196
Glens Falls	Warren	New York	49,944
	Washington	New York	38,931
Goldsboro	Wayne	North Carolina	130,660
Grand Forks	Grand Forks	North Dakota	82,408
	Polk	Minnesota	30,925
Grand Junction	Mesa	Colorado	128,755
Grand Rapids-Muskegon-Holland	Allegan	Michigan	93,412
	Kent	Michigan	552,812
	Muskegon	Michigan	167,496
	Ottawa	Michigan	186,386
Great Falls	Cascade	Montana	99,816
Green Bay	Brown	Wisconsin	218,748
Greensboro--Winston-Salem--High Point	Alamance	North Carolina	133,111
	Davidson	North Carolina	171,258
	Davie	North Carolina	38,220
	Forsyth	North Carolina	328,689
	Guilford	North Carolina	449,442
	Randolph	North Carolina	143,574
	Stokes	North Carolina	35,783
	Yadkin	North Carolina	43,616
Greenville	Pitt	North Carolina	135,297

Exhibit A-3 Population and Counties in Metropolitan Statistical Areas (cont.)

Metropolitan Statistical Area	County	State	Population 2007
Greenville-Spartanburg-Anderson	Anderson	South Carolina	177,971
	Cherokee	South Carolina	53,831
	Greenville	South Carolina	394,213
	Pickens	South Carolina	98,847
	Spartanburg	South Carolina	260,792
Harrisburg-Lebanon-Carlisle	Cumberland	Pennsylvania	188,821
	Dauphin	Pennsylvania	241,090
	Lebanon	Pennsylvania	124,180
	Perry	Pennsylvania	43,514
Hartford	Hartford	Connecticut	940,275
	New London	Connecticut	257,140
	Tolland	Connecticut	129,274
Hattiesburg	Forrest	Mississippi	63,946
	Lamar	Mississippi	50,276
Hickory-Morganton-Lenoir	Alexander	North Carolina	33,648
	Burke	North Carolina	99,492
	Caldwell	North Carolina	93,516
	Catawba	North Carolina	143,181
Houma	LaFourche	Louisiana	94,575
	Terrebonne	Louisiana	101,320
Houston	Brazoria	Texas	279,348
	Chambers	Texas	22,216
Houston (cont.)	Fort Bend	Texas	279,224
	Galveston	Texas	307,232
	Harris	Texas	3,665,160
	Liberty	Texas	72,557
	Montgomery	Texas	251,257
	Waller	Texas	36,339
Huntington-Ashland	Boyd	Kentucky	61,982
	Cabell	West Virginia	103,921
	Carter	Kentucky	27,105
	Greenup	Kentucky	39,215
	Lawrence	Ohio	79,127
	Wayne	West Virginia	26,545
Huntsville	Limestone	Alabama	57,243
	Madison	Alabama	283,197
Indianapolis	Boone	Indiana	37,196
	Hamilton	Indiana	105,160

Exhibit A-3 Population and Counties in Metropolitan Statistical Areas (cont.)

Metropolitan Statistical Area	County	State	Population 2007
	Hancock	Indiana	44,288
	Hendricks	Indiana	73,305
	Johnson	Indiana	148,916
	Madison	Indiana	154,883
	Marion	Indiana	902,953
	Morgan	Indiana	64,073
	Shelby	Indiana	42,188
Iowa City	Johnson	Iowa	101,591
Jackson	Jackson	Michigan	155,830
	Hinds	Mississippi	283,988
	Madison	Mississippi	68,527
	Rankin	Mississippi	100,180
	Chester	Tennessee	15,727
	Madison	Tennessee	96,308
Jacksonville	Clay	Florida	106,644
	Duval	Florida	917,919
	Nassau	Florida	57,836
	St. Johns	Florida	97,808
	Onslow	North Carolina	190,295
Jamestown	Chautauqua	New York	144,849
Janesville-Beloit	Rock	Wisconsin	161,217
Johnson City-Kingsport-Bristol	Bristol	Virginia	39,828
	Carter	Tennessee	59,504
	Hawkins	Tennessee	44,941
	Scott	Virginia	30,795
Johnson City-Kingsport-Bristol (cont.)	Sullivan	Tennessee	160,736
	Unicoi	Tennessee	13,114
	Washington	Tennessee	125,723
	Washington	Virginia	62,837
Johnstown	Cambria	Pennsylvania	178,087
	Somerset	Pennsylvania	75,413
Jonesboro	Craighead	Arkansas	83,910
Joplin	Jasper	Missouri	88,183
	Newton	Missouri	66,924
Kalamazoo-Battle Creek	Calhoun	Michigan	142,742
	Kalamazoo	Michigan	228,777
	Van Buren	Michigan	69,545
Kansas City	Cass	Missouri	85,189

Exhibit A-3 Population and Counties in Metropolitan Statistical Areas (cont.)

Metropolitan Statistical Area	County	State	Population 2007
	Clay	Missouri	192,614
	Clinton	Missouri	15,214
	Jackson	Missouri	533,890
	Johnson	Kansas	422,469
	Lafayette	Missouri	35,420
	Leavenworth	Kansas	58,919
	Miami	Kansas	26,581
	Platte	Missouri	52,841
	Ray	Missouri	20,048
	Wyandotte	Kansas	348,779
Killeen-Temple	Bell	Texas	256,642
	Coryell	Texas	76,073
Knoxville	Anderson	Tennessee	88,295
	Blount	Tennessee	98,190
	Knox	Tennessee	413,282
	Loudon	Tennessee	50,643
	Sevier	Tennessee	65,931
	Union	Tennessee	21,446
Kokomo	Howard	Indiana	93,535
	Tipton	Indiana	15,822
La Crosse	Houston	Minnesota	16,786
	La Crosse	Wisconsin	114,245
Lafayette	Clinton	Indiana	36,559
	Tippecanoe	Indiana	147,866
	Acadia	Louisiana	62,501
	Lafayette	Louisiana	188,498
	St. Landry	Louisiana	85,693
	St. Martin	Louisiana	45,322
Lake Charles	Calcasieu	Louisiana	184,810
Lakeland-Winter Haven	Polk	Florida	526,755
Lancaster	Lancaster	Pennsylvania	439,469
Lansing-East Lansing	Clinton	Michigan	82,444
	Eaton	Michigan	145,296
	Ingham	Michigan	229,021
Laredo	Webb	Texas	174,981
Las Cruces	Dona Ana	New Mexico	180,761
Las Vegas	Clark	Nevada	1,294,955
	Mohave	Arizona	140,633

Exhibit A-3 Population and Counties in Metropolitan Statistical Areas (cont.)

Metropolitan Statistical Area	County	State	Population 2007
	Nye	Nevada	32,052
Lawrence	Douglas	Kansas	95,395
Lawton	Comanche	Oklahoma	125,946
Lewiston-Auburn	Androscoggin	Maine	112,945
Lexington	Bourbon	Kentucky	20,972
	Clark	Kentucky	31,105
	Fayette	Kentucky	245,718
	Jessamine	Kentucky	43,670
	Madison	Kentucky	65,485
	Scott	Kentucky	26,476
	Woodford	Kentucky	21,090
Lima	Allen	Ohio	120,173
	Auglaize	Ohio	46,691
Lincoln	Lancaster	Nebraska	241,281
Little Rock-North Little Rock	Faulkner	Arkansas	70,939
	Lonoke	Arkansas	47,016
	Pulaski	Arkansas	425,636
	Saline	Arkansas	67,021
Longview-Marshall	Gregg	Texas	101,586
	Harrison	Texas	98,027
	Upshur	Texas	46,015
Los Angeles-Long Beach	Los Angeles	California	10,787,273
	Orange	California	2,910,595
	Riverside	California	1,420,146
	San Bernardino	California	1,833,774
	Ventura	California	811,814
Louisville	Bullitt	Kentucky	48,330
	Clark	Indiana	105,393
	Floyd	Indiana	57,196
	Harrison	Indiana	34,065
	Jefferson	Kentucky	760,081
Louisville (cont.)	Oldham	Kentucky	43,849
	Scott	Indiana	24,023
Lubbock	Lubbock	Texas	294,525
Lynchburg	Amherst	Virginia	27,885
	Bedford	Virginia	43,616
	Bedford City	Virginia	7,861
	Campbell	Virginia	78,196

Exhibit A-3 Population and Counties in Metropolitan Statistical Areas (cont.)

Metropolitan Statistical Area	County	State	Population 2007
	Lynchburg	Virginia	76,125
Macon	Bibb	Georgia	204,380
	Houston	Georgia	123,369
	Jones	Georgia	29,287
	Peach	Georgia	24,453
	Twiggs	Georgia	10,006
Madison	Dane	Wisconsin	417,101
Mansfield	Crawford	Ohio	45,106
	Richland	Ohio	143,179
McAllen-Edinburg-Mission	Hidalgo	Texas	501,759
Medford-Ashland	Jackson	Oregon	191,802
Melbourne-Titusville-Palm Bay	Brevard	Florida	522,202
Memphis	Crittenden	Arkansas	61,523
	De Soto	Mississippi	72,816
	Fayette	Tennessee	28,868
	Shelby	Tennessee	1,047,856
	Tipton	Tennessee	42,437
Merced	Merced	California	216,576
Milwaukee-Waukesha	Milwaukee	Wisconsin	1,090,555
	Ozaukee	Wisconsin	88,427
	Racine	Wisconsin	188,974
	Washington	Wisconsin	109,707
	Waukesha	Wisconsin	342,632
Minneapolis-St. Paul	Anoka	Minnesota	298,159
	Carver	Minnesota	54,455
	Chisago	Minnesota	36,895
	Dakota	Minnesota	329,595
	Hennepin	Minnesota	1,190,378
	Isanti	Minnesota	29,977
	Pierce	Wisconsin	28,292
	Ramsey	Minnesota	516,682
	Scott	Minnesota	78,315
	Sherburne	Minnesota	45,391
	St. Croix	Wisconsin	64,084
Minneapolis-St. Paul (cont.)	Washington	Minnesota	194,467
	Wright	Minnesota	76,137
Missoula	Missoula	Montana	102,046
Mobile	Baldwin	Alabama	111,772

Exhibit A-3 Population and Counties in Metropolitan Statistical Areas (cont.)

Metropolitan Statistical Area	County	State	Population 2007
	Mobile	Alabama	445,806
Modesto	Stanislaus	California	458,480
Monroe	Ouachita	Louisiana	159,432
Montgomery	Autauga	Alabama	36,292
	Elmore	Alabama	57,067
	Montgomery	Alabama	247,357
Muncie	Delaware	Indiana	133,491
Myrtle Beach	Horry	South Carolina	172,374
Naples	Collier	Florida	194,829
Nashville	Cheatham	Tennessee	38,155
	Davidson	Tennessee	638,546
	Dickson	Tennessee	40,770
	Robertson	Tennessee	58,776
	Rutherford	Tennessee	149,125
	Sumner	Tennessee	128,562
	Williamson	Tennessee	77,941
	Wilson	Tennessee	96,514
New London-Norwich	Washington	Rhode Island	109,790
New Orleans	Jefferson	Louisiana	350,777
	Orleans	Louisiana	706,050
	Plaquemines	Louisiana	39,055
	St. Bernard	Louisiana	48,306
	St. Charles	Louisiana	38,750
	St. James	Louisiana	25,437
	St. John the Baptist	Louisiana	44,795
	St. Tammany	Louisiana	158,546
New York	Bergen	New Jersey	1,185,226
	Bronx	New York	1,084,664
	Dutchess	New York	242,003
	Essex	New Jersey	754,779
	Fairfield	Connecticut	828,663
	Hudson	New Jersey	695,167
	Hunterdon	New Jersey	118,768
	Kings	New York	2,605,842
	Litchfield	Connecticut	157,809
	Mercer	New Jersey	256,800
	Middlesex	Connecticut	142,704
New York (cont.)	Middlesex	New Jersey	736,316

Exhibit A-3 Population and Counties in Metropolitan Statistical Areas (cont.)

Metropolitan Statistical Area	County	State	Population 2007
	Monmouth	New Jersey	600,680
	Morris	New Jersey	480,555
	Nassau	New York	1,378,171
	New Haven	Connecticut	805,581
	New York	New York	949,251
	Ocean	New Jersey	477,040
	Orange	New York	307,181
	Passaic	New Jersey	568,487
	Pike	Pennsylvania	29,220
	Putnam	New York	122,472
	Queens	New York	2,040,186
	Richmond	New York	388,260
	Rockland	New York	231,030
	Somerset	New Jersey	328,435
	Suffolk	New York	1,438,434
	Sussex	New Jersey	158,618
	Union	New Jersey	391,026
	Warren	New Jersey	90,867
	Westchester	New York	984,082
Norfolk-Virginia Beach-Newport News	Chesapeake	Virginia	266,152
	Currituck	North Carolina	19,809
	Gloucester	Virginia	36,747
	Hampton	Virginia	287,472
	Isle of Wight	Virginia	24,302
	James City	Virginia	61,727
	Mathews	Virginia	8,460
	Newport News	Virginia	157,041
	Norfolk	Virginia	199,055
	Poquoson City	Virginia	37,277
	Portsmouth	Virginia	132,013
	Suffolk	Virginia	57,874
	Virginia Beach	Virginia	423,444
	Williamsburg	Virginia	4,070
	York	Virginia	34,874
Ocala	Marion	Florida	259,484
Odessa-Midland	Ector	Texas	155,113
	Midland	Texas	140,701
Oklahoma City	Canadian	Oklahoma	72,124

Exhibit A-3 Population and Counties in Metropolitan Statistical Areas (cont.)

Metropolitan Statistical Area	County	State	Population 2007
	Cleveland	Oklahoma	204,385
	Logan	Oklahoma	35,261
Oklahoma City (cont.)	McClain	Oklahoma	25,060
	Oklahoma	Oklahoma	679,527
	Pottawatomie	Oklahoma	74,669
Omaha	Cass	Nebraska	23,421
	Douglas	Nebraska	510,567
	Pottawattamie	Iowa	86,510
	Sarpy	Nebraska	64,335
	Washington	Nebraska	18,105
Orlando	Lake	Florida	185,909
	Orange	Florida	930,255
	Osceola	Florida	137,994
	Seminole	Florida	336,327
Owensboro	Daviess	Kentucky	97,223
Panama City	Bay	Florida	166,259
Parkersburg-Marietta	Washington	Ohio	58,776
	Wood	West Virginia	96,334
Pensacola	Escambia	Florida	359,439
	Santa Rosa	Florida	100,264
Peoria-Pekin	Peoria	Illinois	210,137
	Tazewell	Illinois	121,979
	Woodford	Illinois	34,644
Philadelphia	Atlantic	New Jersey	242,431
	Bucks	Pennsylvania	721,397
	Burlington	New Jersey	376,536
	Camden	New Jersey	559,251
	Cape May	New Jersey	105,143
	Cecil	Maryland	75,578
	Chester	Pennsylvania	381,366
	Cumberland	New Jersey	167,282
	Delaware	Pennsylvania	469,634
	Gloucester	New Jersey	267,647
	Montgomery	Pennsylvania	684,815
	New Castle	Delaware	572,829
	Philadelphia	Pennsylvania	1,736,353
	Salem	New Jersey	54,077
Phoenix-Mesa	Maricopa	Arizona	3,130,132

Exhibit A-3 Population and Counties in Metropolitan Statistical Areas (cont.)

Metropolitan Statistical Area	County	State	Population 2007
	Pinal	Arizona	168,280
Pine Bluff	Jefferson	Arkansas	102,116
Pittsburgh	Allegheny	Pennsylvania	1,374,661
	Beaver	Pennsylvania	189,945
	Butler	Pennsylvania	154,527
Pittsburgh (cont.)	Fayette	Pennsylvania	142,436
	Washington	Pennsylvania	232,167
	Westmoreland	Pennsylvania	365,691
Pittsfield	Berkshire	Massachusetts	149,519
Pocatello	Bannock	Idaho	103,235
Portland	Cumberland	Maine	257,111
Portland-Vancouver	Clackamas	Oregon	429,973
	Clark	Washington	290,215
	Columbia	Oregon	63,212
	Marion	Oregon	290,392
	Multnomah	Oregon	759,590
	Polk	Oregon	73,595
	Washington	Oregon	376,312
	Yamhill	Oregon	87,736
Providence-Fall River-Warwick	Bristol	Rhode Island	48,176
	Kent	Rhode Island	180,970
	Newport	Rhode Island	88,118
	Providence	Rhode Island	613,284
Provo-Orem	Utah	Utah	379,915
Pueblo	Pueblo	Colorado	170,854
Punta Gorda	Charlotte	Florida	129,773
Raleigh-Durham-Chapel Hill	Chatham	North Carolina	44,710
	Durham	North Carolina	224,052
	Franklin	North Carolina	44,865
	Johnston	North Carolina	104,399
	Orange	North Carolina	128,203
	Wake	North Carolina	542,236
Rapid City	Pennington	South Dakota	90,759
Reading	Berks	Pennsylvania	330,183
Redding	Shasta	California	178,718
Reno	Washoe	Nevada	444,290
Richland-Kennewick-Pasco	Benton	Washington	154,021
	Franklin	Washington	47,994

Exhibit A-3 Population and Counties in Metropolitan Statistical Areas (cont.)

Metropolitan Statistical Area	County	State	Population 2007
Richmond-Petersburg	Charles City	Virginia	6,974
	Chesterfield	Virginia	209,960
	Colonial Heights	Virginia	47,407
	Dinwiddie	Virginia	24,039
	Goochland	Virginia	16,157
	Hanover	Virginia	96,238
	Henrico	Virginia	245,338
	Hopewell	Virginia	27,470
Richmond-Petersburg (cont.)	New Kent	Virginia	15,339
	Petersburg	Virginia	27,981
	Powhatan	Virginia	18,842
	Prince George	Virginia	17,064
	Richmond City	Virginia	300,492
Roanoke	Botetourt	Virginia	30,628
	Roanoke	Virginia	130,741
	Roanoke City	Virginia	114,940
Rochester	Olmsted	Minnesota	125,308
	Genesee	New York	58,676
	Livingston	New York	59,683
	Monroe	New York	740,592
	Ontario	New York	88,153
	Orleans	New York	43,564
	Wayne	New York	84,356
Rockford	Boone	Illinois	34,334
	Ogle	Illinois	49,483
	Winnebago	Illinois	268,755
Rocky Mount	Edgecombe	North Carolina	74,439
	Nash	North Carolina	93,155
Sacramento	El Dorado	California	153,396
	Placer	California	201,223
	Sacramento	California	1,265,658
	Yolo	California	188,554
Saginaw-Bay City-Midland	Bay	Michigan	122,673
	Midland	Michigan	75,394
	Saginaw	Michigan	229,942
Salinas	Monterey	California	463,926
Salt Lake City-Ogden	Davis	Utah	320,150
	Salt Lake	Utah	1,055,128

Exhibit A-3 Population and Counties in Metropolitan Statistical Areas (cont.)

Metropolitan Statistical Area	County	State	Population 2007
	Weber	Utah	183,366
San Angelo	Tom Green	Texas	129,131
San Antonio	Bexar	Texas	1,570,715
	Comal	Texas	70,552
	Guadalupe	Texas	67,097
	Wilson	Texas	26,960
San Diego	San Diego	California	3,040,458
San Francisco	Alameda	California	1,564,111
	Contra Costa	California	1,015,368
	Marin	California	274,898
	Napa	California	141,650
San Francisco (cont.)	San Francisco	California	810,176
	San Mateo	California	885,538
	Santa Clara	California	1,795,115
	Santa Cruz	California	238,573
	Solano	California	412,068
	Sonoma	California	476,488
San Luis Obispo-Atascadero-Paso Robles	San Luis Obispo	California	265,215
Santa Barbara-Santa Maria-Lompoc	Santa Barbara	California	452,536
Santa Fe	Los Alamos	New Mexico	25,113
	Santa Fe	New Mexico	138,043
Sarasota-Bradenton	Manatee	Florida	294,140
	Sarasota	Florida	361,022
Savannah	Bryan	Georgia	18,217
	Chatham	Georgia	296,255
	Effingham	Georgia	29,253
Scranton--Wilkes-Barre--Hazleton	Columbia	Pennsylvania	52,642
	Lackawanna	Pennsylvania	218,704
	Luzerne	Pennsylvania	369,260
	Wyoming	Pennsylvania	33,871
Seattle-Bellevue-Everett	Island	Washington	80,699
	King	Washington	2,045,339
	Kitsap	Washington	245,851
	Pierce	Washington	807,478
	Snohomish	Washington	572,104
	Thurston	Washington	214,010
Sharon	Mercer	Pennsylvania	121,878
Sheboygan	Sheboygan	Wisconsin	116,523

Exhibit A-3 Population and Counties in Metropolitan Statistical Areas (cont.)

Metropolitan Statistical Area	County	State	Population 2007
Sherman-Denison	Grayson	Texas	127,379
Shreveport-Bossier City	Bossier	Louisiana	117,840
	Caddo	Louisiana	248,616
	Webster	Louisiana	46,967
Sioux City	Dakota	Nebraska	17,544
	Woodbury	Iowa	109,316
Sioux Falls	Lincoln	South Dakota	15,079
	Minnehaha	South Dakota	148,638
South Bend	St. Joseph	Indiana	262,727
Spokane	Spokane	Washington	482,077
Springfield	Menard	Illinois	11,633
	Sangamon	Illinois	195,339
Springfield	Franklin	Massachusetts	71,145
	Hampshire	Massachusetts	154,330
Springfield	Christian	Missouri	33,376
	Greene	Missouri	241,667
	Webster	Missouri	26,684
St. Cloud	Benton	Minnesota	48,890
	Stearns	Minnesota	131,430
St. Joseph	Andrew	Missouri	21,069
	Buchanan	Missouri	93,770
St. Louis	Clinton	Illinois	32,067
	Crawford	Missouri	27,043
	Franklin	Missouri	88,665
	Jefferson	Missouri	194,966
	Jersey	Illinois	21,023
	Lincoln	Missouri	33,989
	Madison	Illinois	318,284
	Monroe	Illinois	16,837
	St. Charles	Missouri	218,093
	St. Clair	Illinois	279,975
	St. Louis	Missouri	1,116,347
	St. Louis City	Missouri	449,668
	Warren	Missouri	22,537
State College	Centre	Pennsylvania	129,802
Steubenville-Weirton	Brooke	West Virginia	17,605
	Hancock	West Virginia	28,798
	Jefferson	Ohio	95,970

Exhibit A-3 Population and Counties in Metropolitan Statistical Areas (cont.)

Metropolitan Statistical Area	County	State	Population 2007
Stockton-Lodi	San Joaquin	California	583,401
Sumter	Sumter	South Carolina	122,049
Syracuse	Cayuga	New York	85,333
	Madison	New York	69,991
	Onondaga	New York	481,586
	Oswego	New York	122,913
Tallahassee	Gadsden	Florida	58,007
	Leon	Florida	253,789
Tampa-St. Petersburg-Clearwater	Hernando	Florida	126,608
	Hillsborough	Florida	1,094,652
	Pasco	Florida	389,024
	Pinellas	Florida	1,103,119
Terre Haute	Clay	Indiana	29,170
	Vermillion	Indiana	19,012
	Vigo	Indiana	119,050
Texarkana	Bowie	Texas	131,124
	Miller	Arkansas	23,865
Toledo	Fulton	Ohio	39,403
	Lucas	Ohio	481,114
	Wood	Ohio	146,861
Topeka	Shawnee	Kansas	189,989
Tucson	Pima	Arizona	982,093
Tulsa	Creek	Oklahoma	53,551
	Osage	Oklahoma	32,933
	Rogers	Oklahoma	70,927
	Tulsa	Oklahoma	609,293
	Wagoner	Oklahoma	39,859
Tuscaloosa	Tuscaloosa	Alabama	172,189
Tyler	Smith	Texas	197,408
Utica-Rome	Herkimer	New York	67,934
	Oneida	New York	253,991
Victoria	Victoria	Texas	98,674
Visalia-Tulare-Porterville	Tulare	California	379,467
Waco	McLennan	Texas	251,395
Washington	Anne Arundel	Maryland	500,770
	Arlington	Virginia	149,832
	Baltimore	Maryland	772,026
	Baltimore City	Maryland	906,517

Exhibit A-3 Population and Counties in Metropolitan Statistical Areas (cont.)

Metropolitan Statistical Area	County	State	Population 2007
	Berkeley	West Virginia	60,633
	Calvert	Maryland	58,573
	Carroll	Maryland	134,450
	Charles	Maryland	120,372
	Clarke	Virginia	15,462
	Culpeper	Virginia	34,840
	Fairfax	Virginia	1,117,645
	Fauquier	Virginia	63,098
	Frederick	Maryland	178,138
	Harford	Maryland	216,604
	Howard	Maryland	239,050
	Jefferson	West Virginia	36,843
	King George	Virginia	15,959
	Loudoun	Virginia	81,723
	Manassas City	Virginia	79,273
	Montgomery	Maryland	983,677
	Prince Georges	Maryland	905,612
	Prince William	Virginia	218,832
	Queen Annes	Maryland	44,005
	Spotsylvania	Virginia	75,538
Washington (cont.)	Stafford	Virginia	89,200
	Warren	Virginia	32,897
	Washington	District of Columbia	506,501
	Washington	Maryland	150,756
Waterloo-Cedar Falls	Black Hawk	Iowa	131,508
Wausau	Marathon	Wisconsin	131,430
West Palm Beach-Boca Raton	Palm Beach	Florida	1,133,763
Wheeling	Belmont	Ohio	64,075
	Marshall	West Virginia	44,354
	Ohio	West Virginia	59,646
Wichita	Butler	Kansas	61,032
	Harvey	Kansas	35,072
	Sedgwick	Kansas	457,080
Wichita Falls	Archer	Texas	10,976
	Wichita	Texas	160,680
Williamsport	Lycoming	Pennsylvania	122,232
Wilmington	Brunswick	North Carolina	70,478
	New Hanover	North Carolina	150,535

Exhibit A-3 Population and Counties in Metropolitan Statistical Areas (cont.)

Metropolitan Statistical Area	County	State	Population 2007
Yakima	Yakima	Washington	253,518
Youngstown-Warren	Columbiana	Ohio	119,602
	Mahoning	Ohio	310,534
	Trumbull	Ohio	224,191
Yuba City	Sutter	California	84,508
	Yuba	California	63,227
Yuma	Yuma	Arizona	160,239

APPENDIX B: IPM™ MODEL DESCRIPTION AND POWER PLANT EMISSION SUMMARY

ICF Consulting (2000) analyzed the impacts to the U.S. electric power sector of two alternative emission control scenarios, using ICF Consulting's Integrated Planning Model™ (IPM™). This study focuses on the impacts to the electric power generating units in the District of Columbia and the 48 contiguous states in the U.S. ICF used those modeling assumptions developed and used by the EPA in its regulatory and policy analyses. These assumptions are described briefly in this report and in greater detail in by EPA (1998b).

IPM™ is a multi-region linear programming model that determines the least-cost capacity expansion and dispatch strategy for operating the power system over specified future periods, under specified operational, market, and regulatory constraints. Constraints include emissions caps, transmission constraints, regional reserve margins, and meeting regional electric demand. Given a specified set of parameters and constraints, IPM™ develops an optimal capacity expansion plan, dispatch order, and air emissions compliance plan for the power generation system based on factors such as fuel prices, capital costs and operation and maintenance (O&M) costs of power generation, etc.

The model is dynamic: it makes decisions based on expectations of future conditions, such as fuel prices, and technology costs. Decisions are made on the basis of minimizing the net present value of capital plus operating costs over the full planning horizon. The model draws on a database containing information on the characteristics of each power generating unit (such as unit ID, unit type, unit location, fuel used, heat rate, emission rate, existing emission control technology, etc.) in the U.S.

The results of this study indicate that in the policy case, the national annual SO_2 emissions decline by about 70 percent and the national annual NO_x emissions decline by over 50 percent relative to the base case in 2007, consistent with the national emissions limitations imposed. Compliance options in the model include with the emissions limits are achieved through installation of emission control technologies, dispatch changes, and fuel switching.

B.1 BASELINE SCENARIO

Under the baseline scenario we made the following assumptions for each pollutant:

• SO_2: The baseline includes the requirements of Title IV of the CAAA. Under this regulation, all affected sources are subject to a national annual SO_2 cap of 9.47 million tons during 2000-2009 and 8.95 million tons from 2010 onwards. A national cap and trade program is modeled, consistent with the Acid Rain Trading Program. At the beginning of the year 2000, the bank of SO2 allowances was estimated to be approximately 11.4 million tons.[24]

•**NO_x**: The baseline includes the requirements of Title IV of the CAAA, Reasonably Achievable Control Technology (RACT) under Title I of the CAAA, state regulations, and the NO_x SIP Call policy. The

[24] This is the most recent SO_2 allowance bank estimate, based on ICF's research.

NO_x SIP Call policy is modeled consistent with the original proposed rule, which included 22 Eastern States and DC (hereafter referred to as the "SIP Call area") beginning May, 2003.[25]

The baseline is consistent with the EPA's NO_x SIP Call policy analysis (EPA 1998a) and has a cap and trade program that requires all fossil-fired power plants in the SIP Call area to reduce their total summer NO_x emissions to 543.8 thousand tons or below from May 2003 onwards. In modeling, all the regulated sources in the SIP Call area are allowed to trade NO_x allowances among them without any restriction, but banking of allowances is not permitted.[26]

For those fossil-fired units that are located outside the SIP Call area, NO_x emission limits were determined based on the applicable requirements of Title IV of the CAAA, Reasonably Achievable Control Technology (RACT) under Title I of the CAAA, and State regulations.[27]

B.2 "75 Percent Reduction" SCENARIO

In the "75 Percent Reduction" scenario, ICF modeled the Title IV SO_2 regulations for the years 2000 through 2004. However, in 2005, a more stringent policy that restricts annual national SO_2 emissions to about one-third of the Phase II SO_2 limit is assumed to come into effect. This new SO_2 policy requires all fossil-fired power plants with capacities greater than 15 MW to reduce their total annual SO_2 emissions to 3.1 million tons.[28] This scenario also allows trading of SO_2 emission allowances among regulated sources. However, banking of SO_2 allowances is not permitted. Also, the SO_2 bank remaining at the end of 2004 from Title IV regulation is not available for use under the new policy that begins in 2005.

Regarding NO_x emisisons, ICF assumed a nation-wide annual NO_x policy beginning in 2005. Under this policy, all fossil-fired power plants with capacities greater than 15 MW are required to reduce their total annual NO_x emissions to 1.8 million tons. This "75 Percent Reduction" scenario allows trading of NO_x emission allowances among regulated sources, but does not permit banking.

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[25] The 22 SIP Call States include: Alabama, Connecticut, Delaware, Georgia, Indiana, Illinois, Kentucky, Maryland, Massachusetts, Michigan, Missouri, New Jersey, New York, North Carolina, Ohio, Pennsylvania, Rhode Island, South Carolina, Tennessee, Virginia, West Virginia, and Wisconsin, which has been exempted from SIP Call by a recent court ruling.

[26] For more information on the EPA's NO_x SIP Call policy analysis, refer to the EPA website at: http://www.epa.gov/ttn/rto/sip/index.html and http://www.epa.gov/capi/.

[27] For more details on EPA's modeling of NO_x emission policies, refer to Appendix 4 (EPA 1998b).

[28] In modeling the policy scenario, only those fossil-fired "model" plants—each of which is an aggregation of EGUs with similar characteristics, such as capacity, heat rate, and unit type, generated for modeling purposes—that constitute majority of the EGUs with capacities greater than 15 MW were modeled as regulated units both for SO_2 and NO_x.

B.3 STUDY METHODS

IPM™ is a multi-region linear programming model that determines the least-cost capacity expansion and dispatch strategy for operating the power system over specified future periods, under specified operational, market, and regulatory constraints. Constraints include emissions caps, transmission constraints, regional reserve margins, and meeting regional electric demand. Given a specified set of parameters and constraints, IPM™ develops an optimal capacity expansion plan, dispatch order, and air emissions compliance plan for the power generation system based on factors such as fuel prices, capital costs and operation and maintenance (O&M) costs of power generation, etc. EPA (1998b) provides additional details about the IPM™ model.

The model is dynamic: it makes decisions based on expectations of future conditions, such as fuel prices, and technology costs. Decisions are made on the basis of minimizing the net present value of capital plus operating costs over the full planning horizon. The model draws on a database containing information on the characteristics of each power generating unit at a power plant (such as unit ID, unit type, unit location, fuel used, heat rate, emission rate, existing emission control technology, etc.) in the U.S. For modeling purposes, these power plants are aggregated into model plants of similar characteristics.

B.3.1 Modeling Assumptions

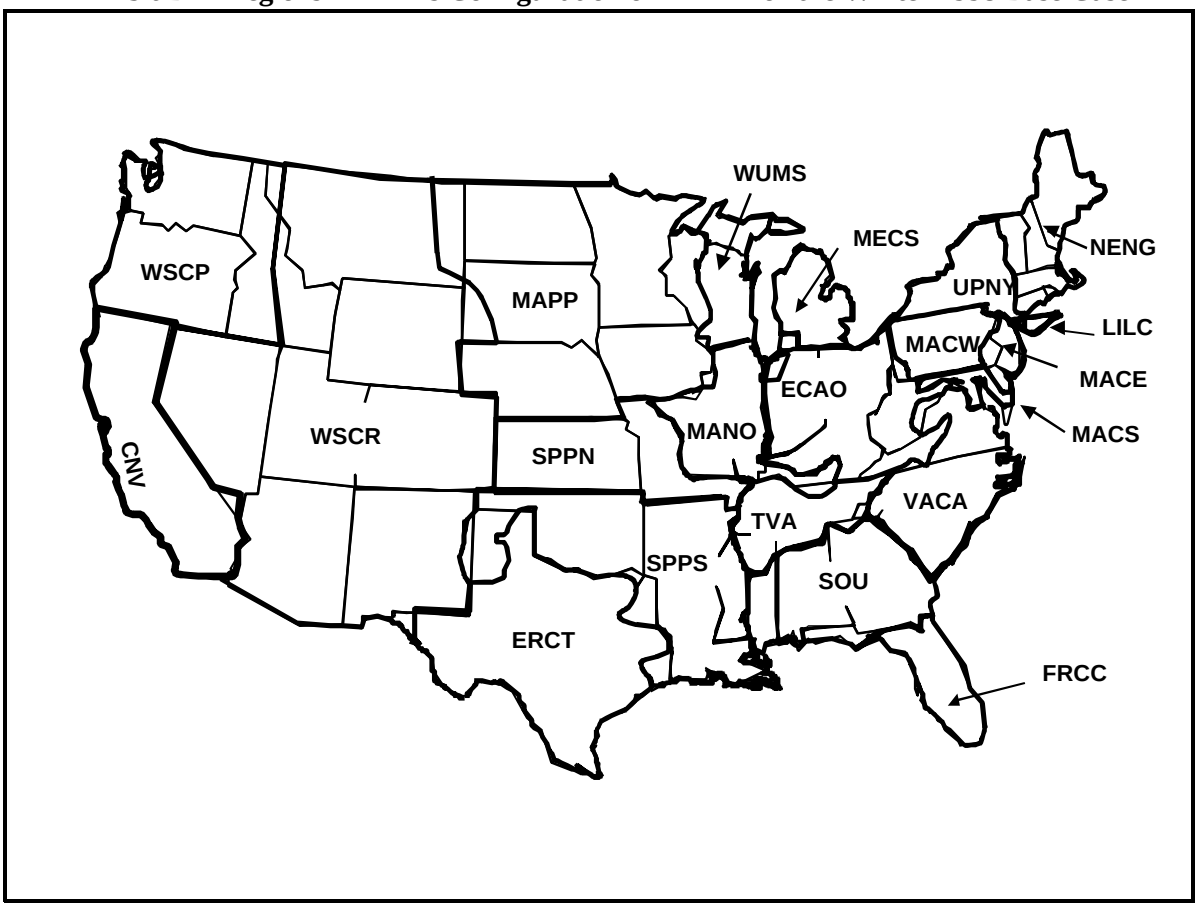
Study Area

This study includes all the power plants in the DC and the 48 contiguous states in the U.S. This study area is divided into 21 regions (Exhibit B-1). While some of these model regions correspond to North American Reliability Council (NERC) regions or sub-regions, other regions are finer divisions of NERC regions or sub-regions.

Modeling Time Period

In this study, the modeling period is 2000 through 2025. Because it would not be feasible to model each calendar year, consistent with the EPA's Winter 1998 Base Case only the following six runs years were modeled: 2001, 2003, 2005, 2007, 2010, and 2020. The model accounts for all years in the planing period by “mapping” multiple years to the model run years. Further, for each model run year, two seasons are modeled. The summer season is assumed to be from May 1 through September 30 and winter season includes the remainder of the year. However, for further analysis, and for a discussion of the results in this report, the model results for the year 2007 have been used.

Exhibit B-1 Regions in EPA's Configuration of IPM™ for the Winter 1998 Base Case



Source: EPA (1998b)

Electric Power System Operating Conditions

• **Electricity Demand**: Under its 1998 Winter Base Case, EPA assumed that the electricity demand would grow at the following rates: (a) 1.6 % per year from 1996 to 2000, (b) 1.8% per year from 2000 through 2010, and (c) 1.3% per year from 2011 onwards. These demand projections for 2000 and beyond were then reduced to reflect EPA's estimate of the the electric demand reductions due to the implementation of the President's Climate Change Action Plan (CCAP). These same assumptions are used in this study. Consistent with EPA's (1998b) modeling methodology, we have not modeled electricity demand responses to changes in electricity prices.

• **Reserve Margins**: Reserve margins are region-specific and they are in the range of about 10 percent to 18.7 percent, with the national weighted average being approximately 15 percent.

• **Power Plant Lifetimes**: Scheduled plant retirements are assumed to occur at 65 years for coal-, oil/gas-, biomass-, and waste fuel-fired steam turbine generating units that are at least 50 MW, and 45 years for steam turbine generating units less than 50 MW. The lifetime for combustion turbines is assumed to be 30 years. The model may choose to retire fossil steam units prior to planned retirement dates for economic reasons.

For nuclear power plants, the lifetime is assumed to be 40 years from their dates of license. In addition, some of the early nuclear plant retirement decisions made in the AEO 1998 are also incorporated in this analysis.

• **Fossil Power Plant Capacity**: For utility generating units, fossil power plant capacity data were obtained from EIA and NERC Electricity Supply and Demand (ES&D) databases. For non-utility generating units, the capacity data were obtained from UDI and NERC ES&D databases.

• **Fossil Power Plant Availability**: The power plant availability, which is defined as the percentage of time that a generating unit is available to provide electricity to the grid, for all coal and oil and gas steam units is assumed to be 83.5 percent during 2000 through 2004, and 85 percent from 2005 onwards.

• **Power Plant Heat Rates**: EPA assumes that the power plant heat rates will remain constant over time.

• **Nuclear Generation**: Nuclear capacity is assumed to decline gradually throughout the modeling period, from 93 GW in 2001 to 81 GW in 2010 to 50 GW in 2020. The capacity factor projections for the nuclear power plants are also based on AEO projections. The national weighted average capacity factors are in the range of 80 to 82 percent during the modeling period.

• **Hydroelectric Generation**: Seasonal averages of historic hydroelectric generation, calculated for each model region using EIA's Form 759 database. The national hydroelectric generation is assumed to remain constant at approximately 277 billion kWh from 2001 through the entire modeling period.

• **Transmission**: For the EPA Base Case, transmission capacity limits between IPM model regions are based on NERC estimates.

• **Net International Imports**: International imports and exports of electricity to and from the U.S. are explicitly modeled in IPM™. Data on imports and exports of electricity were obtained from EIA and NERC databases.

Economic Assumptions

The two major economic assumptions used in this study relate to the discount rate and capital charge rate for investments in new generation capacity and pollution control technology.

•**Discount Rate**: A real discount rate of six percent is used.

•**Capital Charge Rate**: A real capital charge rate of 10.4 percent is used to amortize the capital costs through the lifetime of the investments.

Fuel Prices

In IPM™, fuel prices could be modeled either endogenously (i.e., determined within the model through demand and supply curves for fuels) or exogenously (i.e., provided as input to the model). EPA's 1998 Winter Base Case assumptions (EPA, 1998b) were adopted in modeling fuel prices. These assumptions are briefly described below.

• **Natural Gas Prices**: In IPM™, gas markets are represented by gas supply curves and fuel transportation costs. Well head gas prices are determined within the model by the level of natural gas demand from the electric power sector, as simulated by IPM™, and gas demand from other sectors, as represented by a gas demand curve. The price level consistent with this level of gas supply is determined from gas supply curves. The natural gas supply curves were developed by ICF Consulting using its North American Natural Gas Analysis System (NANGAS).

• **Coal Prices**: In IPM™, coal markets are modeled endogenously through coal supply curves and transportation information. While coal demand by type of coal is simulated through the model using ICF's coal supply curves by type of coal are provided as input to the model. The coal supply curves in IPM are ICF projections based on the coal resource base, current mining production and transportation costs, and expected future increases in mining and transportation productivity.

• **Oil Prices**: Residual fuel oil prices are exogenous inputs to IPM™ for the entire modeling time period and are based on EIA's AEO 1998 forecasts

• **Biomass Fuel Prices**: In IPM™, biomass fuel prices are determined within the model using regional biomass supply curves, based on EIA data.

Costs for Existing Power Plants

The costs for existing power plants vary by the type and the age of the units, and the projected retrofit types for those units. The cost (which include capital, variable operation and maintenance (O&M), and fixed O&M costs) characteristics modeled for existing power plants were developed and used by EPA in its regulatory and policy analyses (EPA 1998b).

Existing steam fossil power plants included in the model have several retrofit choices including: (a) early retirement due to economic reasons, (b) repowering to combined cycle operation, and (c) pollution control technology.

Repowering refers to retrofitting existing fossil-steam power plants with new combined cycle (CC) or integrated coal gasification combined cycle (IGCC) equipment. EPA has assumed for its 1998 Winter Base Case that repowering will become economical only from 2010 and that only those power plants with 500 MW capacities or less could be repowered. Further, it is assumed that repowering will double the capacity of the retrofitted power plant.

Repowering options available for power plants differ by the type of fuel used. While coal steam plants could choose to repower either to CCs or IGCCs, oil and gas steam plants are allowed to repower only to CC operation. Repowering requires a capital investment and increases O&M costs. CC repowering costs and the thermal efficiency of the repowered units are the same for both coal and oil and gas steam units. The IGCC repowering costs are much higher. For example, the capital cost for an IGCC is over five times higher than the capital cost for a CC. The cost and performance characteristics of alternative repowering options are briefly summarized below in Exhibit B-2.

Exhibit B-2 Cost and Performance Characteristics of Repowering Options

[1]

2010 - 2030 Period	Repower Coal to Coal IGCC	Repower Coal to Gas Combined Cycle	Repower Oil/Gas to Gas Combined Cycle
Typical Size (MW)	500	600	600
Heat Rate (Btu/kWh)	8,825	6,498	6,498
Capital (1997$/kW)	1,566	279	279
Fixed O&M ($/kW/yr)	25.44	19.5	19.5
Variable O&M ($/MWh)	2.42	1.1	1.1

Source: EPA (1998b, Table A3-8).

[1]Repowering options are modeled for the years, 2010 through 2025.

Cost and Performance Characteristics for New Power Plants

EPA's assumptions about the costs and performance characteristics for new power plants differ by type of power plant technology, which include fossil, nuclear, and renewable technologies (EPA, 1998b). While for some technologies (such as IGCC and combustion turbines) the costs and the performance characteristics are expected to remain unchanged during the modeling period, for other technologies (such as CC), the costs are assumed to decline and the performance characteristics are assumed to improve over time during the modeling period.

For example, as Exhibit B-3 shows, EPA has assumed that three vintage models (i.e., 2000-2004, 2005-2009, and 2010 and after) of CCs would become available during the modeling period, with each successive model being more efficient and less costly than its predecessor. Accordingly, the capital costs of new CCs are assumed to decline by about 30% in 2005, and by about 40% in 2010, below the 2000 level. Similarly, the thermal efficiency of new CCs are assumed to increase by about 3 percent in 2005, and by an additional 3 percent in 2010, over the 2000 level.

Exhibit B-3 Cost and Performance Characteristics for Selected New Fossil Technologies

Year		Combined Cycle (400 MW)	Combustion Turbine (80 MW)	IGCC (380 MW)
2000 - 2004	Heat Rate (Btu/kWh)	6,773	11,075	--
	Capital (1997$/kW)	617	379	--
	Fixed O&M (1997$/kW/yr)	19.5 [a]	1.74	--
	Variable O&M (1997$/MWh)	1.1	1.0	--
2005-2009	Heat Rate (Btu/kWh)	6,562	11,075	8,470
	Capital (1997$/kW)	431	379	2,136
	Fixed O&M (1997$/kW/yr)	19.5	1.74	25.44
	Variable O&M (1997$/MWh)	1.1	1.0	2.02
2010 and after	Heat Rate (Btu/kWh)	6,350	11,075	8,470
	Capital (1997$/kW)	367	379	2,136
	Fixed O&M (1997$/kW/yr)	19.5	1.74	25.44
	Variable O&M (1997$/MWh)	1.1	1.0	2.02

Source: EPA (1998b, Table A3-2).

[a] We add to the fixed O&M for new combined-cycle units a charge for acquiring a non-interruptible gas contract. This cost varies across regions and over time.

Emission Rates and Pollution Control Technology

ICF (2000)used emission rates for SO_2 and NO_x for power generating units based on the EPA report (1998b). Further, SO_2 and NO_x emission control options are provided to power generating units. The model endogenously assigns emission control technlogies to power generating units, such scrubbers for SO_2 and three post-combustion control technologies (i.e., SCR, SNCR, and gas reburn) for NO_x. In addition, NO_x combustion control technologies are exogenously assigned to all coal-fired generating units as described in the EPA report (1998b). The characteristics of these pollution control technologies used in this study are briefly summarized below.

Sulfur Dioxide

All coal fired steam plants with capacities greater than 500 MW are given the options to be retrofitted with wet scrubber technology. In addition, plants could comply with the SO_2 emission limits through fuel switching (such as switching from high sulfur to low sulfur coal), dispatch changes (to alter fuel consumption), or repowering.

The SO_2 removal efficiency of scrubbers is assumed to be 95 percent (EPA, 1998b) and invariant to sulfur content of coal. Installation of scrubbers is assumed to entail both capacity and energy penalties of 2.1 percent each.

Nitrogen Oxides

For the baseline, consistent with EPA assumptions (EPA 1998b), it was assumed that all coal-fired generating units with greater than 25 MW will be retrofitted with NO_x combustion control technology, such as low NO_x burners. The combustion control technology was exogenously assigned to the coal-fired units. The NO_x control efficiency of the combustion control technology was assumed to vary by the coal-fired boiler type. The NO_x removal rates of these technologies are in the range of about 31 percent to 68 percent (EPA 1998b).

In addition to combustion control technology, in the model, coal and oil and gas steam plants were assigned the option to take on one of the following three post-combustion control technologies: SCR, SNCR, or Gas Reburn. EPA assumes that all new combined cycle (CC) units are built with SCR and combustion controls, resulting in a NO_X rate of 0.02 lb/MMBtu and that all combustion turbines (CT) are built with combustion controls, resulting in a NO_X rate of 0.08 lb/MMBtu (EPA 1998b). NO_x removal efficiency of post-combustion NO_x control technology may vary depending on the type and the existing NO_x emission rate of the unit, as shown in Exhibit B-4.

The cost characteristics of the post-combustion NO_x control technologies also vary by the existing NO_x emission rate, the type, and the capacity of the unit. In the Base Case, it was assumed that these technologies would be operated only during the summer (avoiding variable O&M costs during the rest of the year). However, in the policy case, the plants were given the option to run the units during summer only, during winter only, or all year long. The decision to retrofit plants with the appropriate post-combustion control technologies is made endogenously on a least-cost basis.

Exhibit B-4 NO_x Removal Rates of Post Combustion NO_x Control Technologies

Post-Combustion NO_x Control Technology		NO_x Removal Rate (%)
SCR for Coal Units		
	Low NO_x Rate[a]	70%
	High NO_x Rate[a]	80%
SNCR for Coal Units		
Low NO_x Rate		40%
	High NO_x Rate	35%
Gas Reburn for Coal Units		
Low NO_x Rate		40%
	High NO_x Rate	50%
SCR for Oil/Gas Steam Units & New CCs		80%
SNCR for Oil/Gas Steam Units & New CCs		50%
Gas Reburn for Oil/Gas Steam Units & New CCs		50%

Source: EPA (1998b, Tables A5-5 and A5-6).

[a] Low NO_x rate corresponds to NO_x rate of less than 0.5 lb per MMBtu and High NO_x rate corresponds to NO_x rate of 0.5 lb per MMBtu or higher.

B.4 Emissions Summary

Exhibit B-5 shows that there are significant reductions in both SO_2 and NO_x emissions from the baseline to the 75 percent control scenario. Exhibit B-6 shows regional changes in NO_x and SO_2 in 2007 in the "75 Percent Reduction" scenario relative to the baseline. The results indicate that emissions of all pollutants decline in 2007, with the exception of summer NO_x emissions in the MAIN NERC region, which increase by about 15 percent in the policy case. As expected, in general, the percentage reductions in the summer and the annual NO_x emissions are the largest in the non-SIP Call regions in 2007 in the policy case. In the case of SO_2 emissions, the emission reductions (in terms of percentage change in emissions relative to the base case) are the highest in the coal-intensive regions, such as ECAR, MAAC, and SERC, and lowest in WSCC which has a significant share of hydro generation.

Exhibit B-5 Change in Annual Emissions in 2007 in the Policy Case

Pollutant	Emission Reductions in the Policy Case	Percentage Change in Emissions in the Policy Case over the Base Case
SO_2 (million tons)	7.1	-70%
NO_x (million tons)	2.4	-57%

Exhibit B-6 Change in Regional Emissions of NO_x and SO_2 in 2007 in the Policy Case over the Base Case[a]

NERC Regions	IPM™ Regions	Change in Summer NO_x Emissions	Change in Annual NO_x Emissions	Change in Annual SO_2 Emissions
ECAR	ECAO, MECS	-11%	-55%	-71%
ERCOT	ERCOT	-68%	-72%	-68%
FRCC	FRCC	-64%	-65%	-65%
MAAC	MACE, MACW, MACS	-7%	-47%	-87%
MAIN	MANO, WUMS	15%	-37%	-70%
MAPP	MAPP	-73%	-73%	-63%
NPCC	LILC, NENG, UPNY	-9%	-17%	-64%
SERC	SOU, TVA, VACA	-14%	-53%	-77%
SPP	SPPN, SPPS [b]	-59%	-64%	-52%
WSCC	CNV, WSCR, WSCP	-63%	-62%	-30%
Total		-42%	-57%	-70%

[a] Includes emissions from all power plant sources.

[b] Includes Entergy NERC sub-region, which is currently a part of SERC, but used to be a part of SPP when the EPA's 1998 Winter Base Case was developed.

APPENDIX C: DETAILS OF THE EMISSIONS INVENTORY

This chapter documents the development of the emission inventories and modeling input files used in this analysis. Pechan (2000) developed the emissions inventories for the business-as-usual (baseline) scenario and for three scenarios: a 75% reduction two-pollutant policy scenario, an All Power Plant scenario, and a scenario eliminating on-road and off-road diesel-powered vehicle emissions

To estimate total emissions for each scenario, Pechan (2000) summed the emissions of five major emission sectors: power plant, non-power plant point, stationary area, non-road, and on-road mobile source sectors. To estimate power plant emissions, Pechan used the results of the Integrated Planning Model™ (IPM™). Except for the power plants, Pechan developed the emissions used in this analysis under an EPA contract in support of EPA's Tier 2 rulemaking analysis (Pechan 1999). These non-power plant emission inventories contain 2007 emission estimates for on-road mobile, non-power plant point, stationary area, and non-road sources. We refer to these non-power plant estimates as the "2007 Tier 2 emission inventories."

The 2007 Tier 2 emission inventories contain annual and summer season daily emissions of NO_x, VOC, CO, SO_2, PM_{10}, $PM_{2.5}$, and NH_3. Non-Power plant point source emissions are provided at state-county-plant-point-stack-SCC level detail. Stationary area, on-road, and non-road sources are provided at the state-county-SCC level detail. In general, Pechan (1999) developed the non-power plant emission inventories by projecting 1996 National Emission Trends (NET) emission estimates to 2007. They provide further details of this projection methodology in their report. In general, Pechan (1999) developed the non-power plant emission inventories by projecting 1996 National Emission Trends (NET) emission estimates to 2007. They provide further details of this projection methodology in their report.

C.1 POWER PLANT EMISSIONS

ICF Consulting (2000) used the IPM™ to forecast SO_2 and NO_x emissions at power plants. For the baseline, ICF assumed a continuation of current EPA policies until the year 2007: full implementation of the NO_x State Implementation Plan (SIP) Call by 2003, full implementation of Phase II of Title IV of the Clean Air Act (CAA) Amendments of 1990, and no explicit adoption of a global warming climate treaty. Using these results and data on plant and fuel types, Pechan (2000) complemented the estimates of SO_2 and NO_x by estimating emissions of carbon monoxide (CO), volatile organic carbon (VOC), ammonia (NH_3), secondary organic aerosols (SOA) and direct particulates for 2007 baseline and control scenario inventories.

ICF Consulting (2000) prepared data files on forecasted heat input, SO_2 emissions, NO_x emissions, and characteristics of the plant and fuel. To supplement these emission estimates and build a complete emission inventory, Pechan (1999) used plant and fuel types to estimate emissions of VOC, CO, PM_{10}, $PM_{2.5}$, NH_3, and secondary organic aerosol (SOA). In addition, Pechan latitude, longitude and stack parameters, which Pechan and ICF used in the air quality modeling.

Pechan developed an emission inventory that included unit-level information for all existing or known planned units. For new units (additional capacity needed to meet future generation demands), Pechan developed state-level estimates by plant type (prime mover) and fuel type are provided.

County Identifiers

For those units with no county identifiers, counties available in cross-reference files developed for the NO_x SIP Call power plant file and other prior analyses performed by Pechan were utilized to incorporate the county code. In some cases, plants were matched to other inventories by state and plant name. Others were matched to Energy Information Administration (EIA)-860 planned unit files or to North American Electric Reliability Council (NERC North American Electric Reliability Council (NERC) reports to identify the county.

Latitude and Longitude

Latitude and longitude coordinates from other inventories, including the NET inventory and the Ozone Transport Assessment Group (OTAG) inventory, were used where units were matched to these inventories at the boiler or plant level. For all other units, county centroids were assigned.

Source Classification Code (SCC)

The source classification code (SCC) is needed to determine the appropriate emission rates of the additional pollutants and to incorporate stack parameters for units that do not match to existing inventories. SCCs were assigned by first matching to existing inventories and then by assigning SCCs based on the unit, fuel, firing, and bottom types. In cases where SCCs taken from other inventories indicate a fuel other than that specified in the unit-level file, SCCs were updated based on the indicated fuel, unit, bottom, and firing types.

Exhibit C-1 Data Elements Provided to Pechan for All Power Plant Scenarios

Data Elements	Description
Plant Name	Plant name
Plant Type	Combined cycle, coal steam, oil/gas steam, turbine, other
State Name	State name
State Code	Federal Information Processing Standard (FIPS) State code
County Name	County name (sometimes missing)
County Code	FIPS county code (sometimes missing)
ORIS Code	ORIS plant code for those units assigned codes, IPM plant code otherwise
Blr	ORIS boiler or unit code where available, otherwise IPM unit code
Capacity	Boiler/unit capacity (MW)
July Day Heat	July day heat input (BBtu/day)
Fuel Type	Primary fuel burned: coal, gas, natural gas, none, refuse, waste coal, wood waste
Bottom	Boiler bottom type: dry, wet, other, unknown, or blank
Firing	Firing type: cell, cyclone, tangential, vertical, well, wet, other, or unknown
Existing SO_2/NO_x Controls	Existing control for SO_2 and/or NO_x - scrubbed, unscrubbed, or blank
Retrofit SO_2/NO_x Controls	Coal to combined cycle, gas reburn, oil/gas selective noncatalytic reduction (SNCR), oil/gas to combined cycle, retirement, coal selective catalytic reduction (SCR), coal scrubber, coal SNCR, or blank
Typical July Day NO_x	Typical July day NO_x emissions (tons/day)
Ash Content	Coal ash content (for fuel type - coal only)
Fuel Sum	5 month summer fuel use or heat input (TBtu)
Fuel Tot	Annual fuel use or heat input (TBtu)
NO_x Sum	5 month NO_x emissions (MTon)
NO_x Tot	Annual NO_x emissions (MTon)
SO_2 Tot	Annual SO_2 emissions (MTon)

Stack Parameters

Stack parameters are added to the power plant file by matching to other inventories. For units where matches to other inventories could not be made, default parameters by SCC were assigned. These default parameters are shown in Exhibit C-2. Stack flow rate and velocity were quality assured to ensure consistency between the two data elements and that the velocities were within acceptable modeling ranges (below 650 feet per second).

Emissions

Emissions of VOC, CO, PM_{10}, $PM_{2.5}$, NH_3, and SOA were added to the inventory. AP-42 (or updated) rates were applied to the reported heat input for each unit to calculate these emissions. For PM_{10} and $PM_{2.5}$, the reported ash content was also utilized along with control efficiency data obtained from other inventories.

A default PM efficiency of 80 percent was applied to all coal-fired units that did not match to other inventories. The emission rates used in this analysis are shown in Exhibit C-2.

New Units

The unit-level data sets provide projected heat input from new units, by prime mover and fuel type. This projected heat input is divided into individual new units based on the model plant parameters shown in Exhibit C-3. New units are then allocated to existing unit sites based on a hierarchy that avoids ozone nonattainment areas (Pechan, 1997b). After assigning location parameters to units, SCCs were assigned based on prime mover and fuel type. Default stack parameters and emissions were added using the same methods applied for existing units.

Mass Emission Inventories and Emission Preprocessor System (EPS) Files

After adding the additional parameters described above to the unit-level file, the final mass and modeling inventories were prepared. June and August daily heat input and emissions were added to the file. This was based on monthly percentage profiles by State, prime mover, and fuel provided by EPA (Stella, 1999). The 5-month (May through September) heat input was allocated to the month and then divided by the number of days in the month. Summer season day emissions were allocated using the same procedure, assuming that the emission rate remained the same across these five months. The June and August daily heat input and emissions were incorporated into the mass files. The EPS 2.5 input files were derived directly from the prepared mass emission files, utilizing the annual emissions.

Exhibit C-2 Default Parameters for Utility Boilers

Unit Type	Primary Fuel	Bottom Type	Firing Type	Ash Content (%)	SCC	PM10 Rate (lbs/MMBtu)	CO Rate (lbs/MMBtu)	VOC Rate (lbs/MMBtu)	Stack Temp. (degrees F)	Stack Height (feet)	Stack Diameter (feet)	Stack Flow (ft3/sec)
AB	Coal			all	10100217	0.3000	0.6923	0.0019	175	570	24	16,286
CC	Gas			---	20100201	0.0133	0.1095	0.0120	300	280	12	2,601
CT	Gas			---	20100201	0.0133	0.1095	0.0120	300	280	12	2,601
ST	Gas			---	10100601	0.0029	0.0381	0.0013	300	280	12	2,601
ST	Coal			5.46	10100202	0.4830	0.0192	0.0023	175	570	24	16,286
ST	Coal	DRY	FRONT	5.92	10100202	0.5237	0.0192	0.0023	175	570	24	16,286
ST	Coal	DRY	FRONT	6.22	10100202	0.5502	0.0192	0.0023	175	570	24	16,286
ST	Coal	DRY	FRONT	9.58	10100202	0.8475	0.0192	0.0023	175	570	24	16,286
ST	Coal	DRY	OPPOSED	9.85	10100202	0.8713	0.0192	0.0023	175	570	24	16,286
ST	Coal	DRY	OPPOSED/CELL	9.32	10100202	0.8245	0.0192	0.0023	175	570	24	16,286
ST	Coal	WET	CYCLONE	7.03	10100203	0.0703	0.0192	0.0042	175	570	24	16,286
ST	Coal	WET	CYCLONE	10.21	10100203	0.1021	0.0192	0.0042	175	570	24	16,286
ST	Coal	DRY	TANGENTIAL	9.92	10100212	0.8775	0.0192	0.0023	175	570	24	16,286
ST	Coal	DRY	TANGENTIAL	16.63	10100212	1.4711	0.0192	0.0023	175	570	24	16,286
ST	Coal	DRY	TANGENTIAL	21.18	10100212	1.8736	0.0192	0.0023	175	570	24	16,286
ST	Oil			---	10100401	0.0190	0.0333	0.0051	300	290	12	3,619
ST	Gas			---	10100601	0.0029	0.0381	0.0013	300	280	12	2,601

Exhibit C-3 Model Plant Parameters for Projected New Utility Units

Plant Parameters	Combined Cycle	Gas Turbine	Coal
Fuel Type	Natural Gas	Natural Gas	Coal
Unit Capacity (megawatts)	225	80	500
SCC	20100201	20100201	10100201
Stack Height [feet (ft)]	280	280	570
Stack Diameter (ft)	12	12	24
Stack Temperature (F)	300	300	175
Exhaust Gas Flow Rate (ft^3/sec)	2,601	2,601	16,286
Stack Gas Velocity (ft/sec)	23	23	36

C.2 POINT SOURCES OTHER THAN POWER PLANTS

Pechan (2000) extrapolated the 2007 non-power plant point source inventory from the 1996 national emission inventory using Bureau of Economic Analysis (BEA) Gross State Product (GSP) growth factors at the State level by 2-digit Standard Industrial Classification (SIC) Code. This inventory includes both annual and summer season daily emissions. Pechan excluded units with SCCs of 101xxx or 201xxx from the non-power plant point inventory because they included them in the power plant inventory. Pechan added SOA emissions by using fractional aerosol coefficients (FACs) based on speciation of the VOC emissions.

Control measures reflecting CAA requirements in addition to NO_x SIP Call control requirements (22 States plus the District of Columbia) were incorporated. The NO_x SIP Call controls applied annual NO_x emission reductions for point sources for controls expected to operate for 12 months/year. Five month reductions were applied to source types with controls expected to operate only during the ozone season. This was necessary to estimate accurate annual emissions since controls such as low NO_x burners cannot be turned off in the winter.

C.3 STATIONARY AREA SOURCES

Pechan (2000) estimated 2007 stationary area source inventory by projecting growths and declines in activity as well as changes in control levels from the 1996 emission inventory. Pechan (1999) provide the growth and control assumptions utilized for this analysis.

C.4 NON-ROAD SOURCES

The 2007 non-road source inventory is based on projected changes (growth or decline) in activity as well as changes in control levels from the 1996 county-level non-road emissions derived from EPA's April 1999 draft version of the "NON-ROAD" model. Emission estimates for VOC, NO_x, CO, SO_2, PM_{10}, and $PM_{2.5}$ are available from the model. NON-ROAD does not estimate NH_3 and SOA emissions; therefore, these emissions were calculated outside the model. Aircraft, commercial marine, and locomotives are not presently included in the NON-ROAD model and were developed separately.

The NON-ROAD model estimates pollutant emissions for the following general equipment categories: (1) agricultural; (2) airport service; (3) light commercial; (4) construction and mining; (5) industrial; (6) lawn and garden; (7) logging; (8) pleasure craft; (9) railway maintenance; and (10) recreational equipment. These applications are further classified according to fuel and engine type [diesel, gasoline 2-stroke, gasoline 4-stroke, compressed natural gas (CNG), and liquid petroleum gas (LPG)].

Base year aircraft emissions were taken from the existing 1996 NET inventory. Locomotive emissions for 1996 were also based on existing NET estimates. Revised VOC, NO_x, CO, and total PM national emission estimates for commercial marine diesel engines were provided by EPA's Office of Transportation and Air Quality (OTAQ). PM_{10} was assumed to be equivalent to PM, and $PM_{2.5}$ was estimated by multiplying PM_{10} emissions by a factor of 0.92. These new national estimates were distributed to counties using the geographic distribution in the existing 1996 NET data base.

2007 Non-road Emissions – No Diesel Scenario

For the No Diesel sensitivity analysis scenario, Pechan (1999) dropped the portion of the emissions inventory associated with diesel combustion from the following non-road sources:

- Recreational Equipment
- Construction and Mining Equipment
- Industrial Equipment
- Lawn and Garden Equipment
- Agricultural Equipment
- Commercial Equipment
- Logging Equipment
- Airport Ground Support Equipment
- Underground Mining Equipment
- Commercial Marine Vessels
- Pleasure Craft
- Military Marine Vessels
- Railroad Equipment.

C.5 ON-ROAD VEHICLE SOURCES

Pechan (1999) based the 2007 on-road vehicle emission inventory on the 1996 emission inventory. They calculated VOC, NO_x, and CO on-road vehicle emission factors using the inputs from the national emission inventory and EPA's MOBILE5b emission factor model. Pechan calculated emission factors for on-road SO_2, PM_{10}, and $PM_{2.5}$ using EPA's PART5 model, and calculated NH_3 emission factors for on-road vehicles using national vehicle-specific emission factors. Pechan then applied various correction factors (VOC and NO_x exhaust, air conditioning usage, and heavy-duty diesel vehicle (HDDV) NO_x defeat device) to the MOBILE5b VOC and NO_x emission factors to simulate emission factors that would result from using MOBILE6, as well as accounting for issues not included in MOBILE5b. The correction factors were provided by OTAQ.

Pechan (1999) projected vehicle miles traveled (VMT) used in 2007 from 1996, using data supplied by OTAQ on the fraction of VMT by vehicle type. The data provided by OTAQ included the VMT fraction for light-duty gasoline vehicles (LDGVs), light-duty gasoline trucks 1 and 2 (LDGT1s, LDGT2s), light-duty

diesel vehicles (LDDVs), and light-duty diesel trucks (LDDTs). The VMT fraction for the remaining vehicle types was calculated to be in the same relative distribution as in the 1996 VMT file. The 1996 VMT at the county/vehicle type/roadway type level of detail was then projected to 2007 by allocating the VMT for each vehicle type according to population growth factors by metropolitan statistical areas and rest-of-State areas.

To simulate the effects of on-board diagnostic (OBD) devices in the projection year, Pechan (1999) made adjustments to the MOBILE5b input files for areas modeled with an inspection and maintenance (I/M) program. They modelled this by adding or modifying pressure and purge test input lines, such that 1996 and later model year LDGVs and LDGTs would receive the full benefits of a test-only pressure test and purge test.

C.5.1 2007 No Diesel On-road Vehicle Emissions

For the no diesel scenario, Pechan (1999) deleted all diesel emissions from the on-road inventory: Light Duty Diesel Vehicles (LDDV); Light Duty Diesel Trucks (LDDT); and Heavy Duty Diesel Vehicles (HDDV).

APPENDIX D: DETAILS OF THE REMSAD AIR QUALITY MODELING

The Regulatory Modeling System for Aerosols and Deposition (REMSAD) was used to simulate estimates of particulate matter concentration for three future-year scenarios. ICF Consulting/Systems Applications International, Inc. (ICF/SAI) performed the REMSAD modeling. The modeling results were subsequently used to estimate the health- and welfare- related costs for each of the scenarios.

The REMSAD model is designed to simulate the effects of changes in emissions on PM concentrations and deposition. REMSAD calculates concentrations of pollutants by simulating the physical and chemical processes in the atmosphere. The basis for REMSAD is the atmospheric diffusion or species continuity equation. This equation represents a mass balance that includes all of the relevant emissions, transport, diffusion, chemical reactions, and removal processes in mathematical terms.
Because it accounts for spatial and temporal variations as well as differences in the reactivity of emissions, REMSAD is ideal for evaluating the air-quality effects of emission control scenarios. Model inputs are prepared from observed meteorological, emissions, and air quality data for selected episode days using various input preparation techniques. The model is then applied with these inputs, and the results are evaluated to determine model performance. Once the model results have been evaluated and determined to perform within prescribed levels, the same base-case meteorological inputs are combined with *modified* or *projected* emission inventories to simulate possible alternative/future emission scenarios.

The meteorological fields for this application of the REMSAD modeling system represent a base year of 1990. These inputs were tested and evaluated by EPA (1999b) and thus no additional modeling of the 1990 base year was done for this study. The modeling domain encompasses the contiguous 48 state, as well as portions of Canada and Mexico. The REMSAD model was applied using a horizontal grid resolution of approximately 56 km. The model was run for an entire year to enable the calculation of annual average values of particulate concentrations.

Three REMSAD simulations were run: 1) a future-year baseline with emissions representing the year 2007, 2) a simulation in which the emissions were reduced in accordance with the "75 Percent Reduction" scenario (with emission limits for NO_x and sulfur dioxide SO_2), and 3) a simulation without emissions from all electric generating units ("power plant"). Gridded, model-ready emission inventories were prepared by ICF/SAI.

Differences between the simulated concentration values for the two emission reduction scenarios and the baseline simulation were used to quantify the effects of the measures on seasonal and annual air quality. The spatial distribution of the differences/effects was also examined.

The remainder of this section contains an overview of the REMSAD modeling system, a summary of the procedures used for this application, and a brief presentation of the simulation results.

D.1 OVERVIEW OF THE REMSAD MODELING SYSTEM

The REMSAD programs have been developed to support a better understanding of the distributions, sources, and removal processes relevant to fine particles and other airborne pollutants, including soluble acidic components and toxics. Consideration of the different processes that affect primary and secondary (i.e., formed by atmospheric processes) particulate matter at the regional scale in different places is fundamental to

advancing this understanding and to assessing the effects of proposed pollution control measures. These same control measures will, in most cases, affect ozone, particulate matter and deposition of pollutants to the surface.

The REMSAD system is built on the foundation of the variable grid Urban Airshed Model (UAM-V) regional air quality model. The aerosol and toxics deposition module (ATDM) is capable of "nesting" a finer-scale subgrid within a coarser overall grid, which permits high resolution over receptor regions. The modeling system may thus be applied at scales ranging from a single metropolitan region to a continent containing multiple urban areas.

The REMSAD system consists of a meteorological data preprocessor (METPROC), the core aerosol and toxic deposition model (ATDM), and postprocessing programs (EXTRACT and REPORT). The ATDM is a three-dimensional grid 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 pollutant concentrations. The basis for the model is the atmospheric diffusion or species continuity equation. This equation represents a mass balance in which all of the relevant emissions, transport, diffusion, chemical reactions, and removal processes are expressed in mathematical terms. The model is typically exercised for a full year.

ATDM input data can be classified into six categories: (1) simulation control, (2) emissions, (3) initial and boundary concentrations, (4) meteorological, (5) surface characteristics, and (6) chemical rates (Exhibit D-1). Each category of inputs contains two or more input files. Each category of inputs contains two or more input files. Some of the input files are optional so that necessary input files may vary between model applications.

The REMSAD predictions of pollutant concentrations are calculated from the emissions, advection, and dispersion of precursors and the formation and deposition of pollutants within every grid cell of the modeling domain. The model is capable of simulating transport and deposition of particulates, toxics, or both. To adequately replicate the full three-dimensional structure of the atmosphere during an episode, the REMSAD program requires an hourly and day-specific database for input preparation. These data require preprocessing steps to translate raw emissions, meteorological, air quality, and grid-specific data to develop final input files.

Exhibit D-1 ATDM Input Data Files.

Data Type	Files	Description
Control	CONTROL	Simulation control information
Emissions	PTSOURCE	Elevated source emissions
	EMISSIONS	Surface emissions
Initial and boundary concentrations	AIRQUALITY	Initial concentrations
	BOUNDARY	Lateral boundary concentrations
	O3CONC/	Ozone concentrations
Meteorological	WIND	X,Y-components of winds
	TEMPERATURE	3D array of temperature
	PSURF	2D array of surface pressure
	H2O	3D array of water vapor
	VDIFFUSION	3D array of vertical turbulent diffusivity coefficients
	RAIN	2D array of rainfall rates
Surface characteristics	SURFACE	Gridded land use
	TERRAIN	Terrain heights
Chemical rates	CHEMPARAM	Chemical reaction rates
	OHLOWR	Hydroxyl radical concentration for lower layer(s)
	OHUPPR	Hydroxyl radical concentration for upper layer(s)
	RATES	Photolysis rates file

Fine particles (or aerosols) are currently thought to pose one of the greatest problems for human health impacts from air pollution. The major factors that affect aerosol air quality include:

- spatial and temporal distribution of toxic and particulate emissions including SO_2, NO_x, VOCs, and NH_3 (both anthropogenic and nonanthropogenic),
- size composition of the emitted PM,
- spatial and temporal variations in the wind fields,
- dynamics of the boundary layer, including stability and the level of mixing,
- chemical reactions involving PM, SO_2, NO_x and other important precursor species,
- diurnal variations of solar insulation and temperature,
- loss of primary and secondary aerosols and toxics by dry and wet deposition, and
- ambient air quality immediately upwind and above the region of study.

The ATDM module simulates these processes when it is used to simulate aerosol distribution and deposition. The model solves the species continuity equation using the method of fractional steps, in which the individual terms in the equation are solved separately in the following order: emissions are injected; horizontal advection/diffusion is solved; vertical advection/diffusion and deposition is solved; and chemical transformations are performed for reactive pollutants. The model performs this four-step solution procedure during one half of each advective (driving) time step, and then reverses the order for the following half time step. The maximum advective time step for stability is a function of the grid size and the maximum wind velocity or horizontal diffusion coefficient. Vertical diffusion is solved on fractions of the advective time step to keep their individual numerical schemes stable. A typical advective time step for coarse (50–80 km) grid spacing is 10–15 minutes, whereas time steps for fine grid spacing (10–30 km) are on the order of a few minutes.

Model inputs are prepared for meteorological and emissions data for the simulation days. Once the model results have been evaluated and determined to perform within prescribed levels, a *projected* emission inventory can be used to simulate possible policy-driven emission scenarios.
REMSAD provides gridded, averaged surface and multi-layer instantaneous concentrations, and surface deposition output for all species and grids simulated. The averaged surface concentrations and depositions are intended for comparison with measurements and ambient standards. The instantaneous concentration output is primarily used to restart the model, and to examine model results in the upper levels.

The particulate matter species modeled by REMSAD include a primary coarse fraction (corresponding to particulates in the 2.5 to 10 micron size range), a primary fine fraction (corresponding to particulates less than 2.5 microns in diameter), and several secondary particulates (e.g., sulfates, nitrates, and organics). The sum of the primary fine fraction and all of the secondary species is assumed to be representative of $PM_{2.5}$. Exhibit D-2 lists the simulated species written to the REMSAD output files.

A number of issues are particularly important to a successful application of REMSAD for evaluating the atmospheric transport and deposition of pollutants. These include the meteorology, accuracy and representativeness of the emission inventory, resolution, structure and extent of the modeling grid, and the treatment of urban areas in both the source and receptor areas of the computational grid. Accurate representation of the input meteorological fields (both spatially and temporally) is necessary in order to adequately capture the transport and deposition of pollutants. The meteorology must be sufficiently resolved in order for the model to accurately simulate the effects of terrain and to diagnose the appropriate cloud characteristics required by the various parameterizations of the cloud processes in the model. The required input fields include temporally varying three dimensional gridded wind, temperature, humidity and vertical exchange coefficient fields, and surface pressure and precipitation rates.

Exhibit D-2 REMSAD output file species.

REMSAD Species[1]	Gas/Aerosol	Description
NO	G	Nitric oxide
NO_2	G	Nitrogen dioxide
SO_2	G	Sulfur dioxide
CO	G	Carbon monoxide
NH_3	G	Ammonia
VOC	G	Volatile organic compounds
HNO_3	G	Nitric acid
PNO_3	A	Particulate nitrate
GSO4	A	Particulate sulfate (gas phase production
ASO4	A	Particulate sulfate (aqueous phase production)
NH4N	A	Ammonium nitrate
NH4S	A	Ammonium sulfate
SOA	A	Secondary organic aerosols
POA	A	Primary organic aerosols
PEC	A	Primary elemental carbon
PMfine	A	Primary fine PM (<2.5 microns)
PMcoarse	A	Primary coarse PM[2] (2.5 to 10 microns)

Sulfate=GSO4+ASO4+NH4S; Nitrate=PNO3+NH4N; Total PM2.5 surrogate=sulfate+nitrate+SOA+POA+Pmfine

These are names used in the model and, for the aerosols, are not necessarily the correct molecular formula (the integers are subscripted only when the formula correctly reflects the species).

Note that (for consistency with the REMSAD User's Guide) we are using the terminology "coarse PM" to mean PM in the size range of 2.5 to 10 microns, which is not in agreement with general use, which defines coarse PM to be particles with size greater than 2.5 microns.

Version 5.0 of the REMSAD modeling system (with simplified ozone chemistry) was employed for this study. All submodules correspond to this version number.

D.2 PARAMETERIZATION OF REACTIONS

The main purpose of the core chemistry module is to provide the necessary fields of atmospheric oxidants (ozone and hydroxyl radical) for calculation of atmospheric particulates. Since the chemistry is parameterized it is computationally efficient, but it is also non-linear and provides a physically reasonable representation of atmospheric chemistry.

The model utilizes a parameterization scheme for hydroxyl, which should provide an adequate first approximation to the photochemistry of importance for PM calculations. The desirable characteristics for such a parameterization are that it respond to changes in ozone, NO_x and light levels, that it capture the diurnal cycle properly, and that it not carry the computational burden associated with standard photochemical model codes.

Hydroxyl is also the initiator for most of the chemical reactions of importance for transformation of the species of interest for applications of REMSAD to transport of toxics. The parameterization accounts for loss of hydroxyl by reactions with methane and carbon monoxide and includes the effects of reactions of hydroxyl with NO_2, SO_2, and a single generic VOC species.

The seven key variables that influence OH concentrations are ozone, NO_x, SO_x, VOC, H_2O, temperature, and solar radiation. All of these are important to accurate prediction of OH; however, ozone, NO_x, and VOC are of particular importance since the other three parameters are invariant under any control scenario. It is important for the model to capture any nonlinearities that occur when NO_x, VOC, and ozone are reduced. For example, under some conditions reductions in NO_x can lead to increases in OH (and hence in the rates of secondary PM production) whereas under other conditions reductions in NO_x emissions can lead to reductions in OH levels.

The photochemical mechanism module used in REMSAD is a reduced-form version of the Carbon Bond Mechanism - version 4 (CBM4) (Gery et al., 1989) as enhanced to include radical-radical termination reactions. This reduced-form version is termed "micro-CB4" (mCB4) and is based on a drastic reduction in the speciation of the organic compounds; the inorganic and radical parts of the mechanism are identical to CBM4. In the original version of mCB4 the organic portion was based on one primary species (VOC) and one primary and secondary carbonyl species (CARB). The original VOC species was incorporated with kinetics representing an average anthropogenic hydrocarbon species. A second primary VOC species representing biogenic emissions has since been added, with kinetic characteristics representing isoprene.

D.2.1 Parameterization of Cloud Chemistry

Chemical processes that occur in the aqueous phase of clouds, rain, and fogs can be important in the formation of secondary particulate matters and in the transformation of toxic pollutants. The process of primary importance for PM applications in REMSAD is sulfate formation. In-cloud processes can account for the majority of atmospheric sulfate formation, especially in the wintertime when gas-phase chemistry is slow. The two most important pathways for in-cloud sulfate formation are the reactions of aqueous SO_2 with ozone and hydrogen peroxide (H_2O_2). At cloud pH below 4-5 (the most common situation in the eastern U.S), the ozone reaction is slow and the H_2O_2 reaction dominates. Since the H_2O_2 is often present at the ambient concentrations below those of SO_2, formation of sulfate can be limited by the availability of H_2O_2, thus can be quite nonlinear. The formation of H_2O_2 is tied to the overall atmospheric photochemical system, and responds to changes in ambient levels of VOC and NO_x. Because of this link, emission changes for VOC and NO_x may have effects on ambient sulfate levels that are equal to or greater than effects due to changes in SO_2 emissions.

When the parameterized chemistry is specified, a parameterized in-cloud sulfate formation algorithm is used. The parameterized in-cloud sulfate formation algorithm is adopted from RTM-II. In this algorithm, relative humidity is used as surrogate for clouds. The humidity-dependent heterogeneous SO_2 conversion rate (R_{SO2}) is calculated from the following formula:

$$R_{SO2} = \exp [0.072 (RH - 70)] - 1 \quad (\%/hr)$$

where RH is relative humidity. This equation was developed based upon measured SO_2 conversion rates for power plant plume in the literature [Dittenhoefer, 1980 #1871]. The equation is only applied when RH exceeds 70 percent. The highest RH value is capped at 95 percent, resulting in a maximum SO_2 conversion rate of 5 percent per hour. (The typical gas-phase conversion rate is 1 percent per hour).

D.3 APPLICATION OF REMSAD FOR THE CONTINENTAL U.S.

The REMSAD modeling procedures used for this application are consistent with those used for the EPA-sponsored Section 812 prospective analysis (EPA, 1999b). All of the inputs, with the exception of the emissions inventories, were adapted from the EPA prospective analysis modeling study.

D.3.1 Modeling Domain

The modeling domain encompasses the contiguous 48 states. The domain extends from 126 degrees west longitude to 66 degrees west longitude, and from 24 degrees north latitude to 52 degrees north latitude. A grid cell size of 2/3 degree longitude by 1/2 degree latitude (approximately 56 by 56 km) was used across the grid, resulting in a 90 by 55 grid (4,950 cells) for each vertical layer. Eight vertical layers were used for the PM modeling.

D.3.2 Simulation Periods

The simulation period includes the entire year of 1990. The output consists of daily average files for the species concentrations. The daily averages were consolidated to calculate the yearly averages as well as the seasonal averages. To be consistent with the emissions files provided by Pechan-Avanti, summer is defined as May through September, and winter the rest of the months (January through April and October through December).

Input Preparation

The REMSAD modeling system also 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.

Separate emission inventories were prepared for the 2007 baseline simulation and each of the scenarios. All other inputs were specified for the base-year model application (1990) and remained unchanged for each modeling scenario.

D.3.3 Modeling Emission Inventories

Emissions for each scenario were provided by Pechan-Avanti and were transformed into to gridded, model-ready inventories using version 2.5e of the Emissions Preprocessing System (EPS 2.5e). The emissions scenarios for this study included the baseline, “75 Percent Reduction”, and All Power Plant Scenarios.

D.3.4 Air Quality, Meteorological, and Land-Use Inputs

Initial species concentrations and lateral boundary conditions were specified to approximate background concentrations of the species; for the lateral boundaries the concentrations varied (decreased parabolically) with height. The background concentrations are listed in Exhibit D-3.

Exhibit D-3 Background Species Concentration Used for REMSAD Initial and Boundary Conditions.

Species	Concentration (ppb)
NO	0.0
NO2	0.1
SO2	0.7
NH3	0.5
VOC	20.0
NHO3	0.01
PNO3	0.01
GSO4	0.1
ASO4	0.0
NH4N	0.01
NH4S	0.1
SOA	1
POA	1
PEC	5
PMFINE	1
PMCOARSE	1

Meteorological inputs were derived based on output from the Pennsylvania State University/ National Center for Atmospheric Research (PSU/NCAR) mesoscale model (MM4). Gridded fields of horizontal wind components, temperature, water-vapor concentration, vertical exchange coefficient, precipitation, and pressure were prepared for input to REMSAD. Land-use information was obtained from the U.S. Geological Survey (USGS) database (at 18 km resolution).

D.3.5 Preparation of REMSAD Output for Health-Effects Calculations

For this study, the following REMSAD-derived species and averages were calculated for each surface-layer grid cell in the modeling domain and provided in electronic format for use in the health-effects calculations:

- daily average values of PM_{10} and $PM_{2.5}$
- annual average ammonium sulfate (NH4S)
- annual average ammonium nitrate (NH4N)
- annual average primary organic aerosols (POA)
- annual average secondary organic aerosols (SOA)
- annual average elemental carbon (PEC)
- annual average ammonia (NH3)

Note that PM_{10} in this case is defined as the sum of the REMSAD species PMcoarse and PMfine from Table 2. $PM_{2.5}$ is equivalent to the PMfine variable.

APPENDIX E: S-R MATRIX-BASED RESULTS

In addition to developing estimates based on the REMSAD model, we estimated the health benefits of emissions reductions based on particulate matter forecasts developed by Pechan (2000). Pechan used the S-R matrix to estimate annual and peak particulate levels for each county in the U.S., and we then used these county-level mean and peak values to estimate the daily average, annual mean, and annual median PM concentrations, which we use in a number of C-R functions.[29] Annual mean PM concentrations are used directly from the air quality data provided by Pechan-Avanti. However, as we discuss below, to estimate annual median and daily average PM concentrations. Below we summarize the S-R model, discuss how we used the S-R results, and we then present estimates of the reduction in adverse health effects for three scenarios. We consider the change in adverse health effects when reducing emissions from the 2007 baseline levels to the "75 Percent Reduction," the "All Power Plant," and the "All Diesel" scenarios.

E.1 DEVELOPMENT OF THE U.S. PM S-R MATRIX

A regional dispersion model was applied to a 1990 U.S. national emission inventory to estimate ambient concentrations throughout North America. Version 3 of the National Particulates Inventory (Pechan, 1995; Barnard, 1996)was selected as the base year inventory since it covers the 48 contiguous States and provides a consistent data set for all of the precursors leading to the formation of ozone and PM. A S-R matrix, relating emissions from a source to a concentration at a receptor county, was then developed based on this air quality modeling. This section describes the development of the regional dispersion model and summarizes a comparison of the modeled concentrations to monitored values. This dispersion-modeling was conducted by Latimer & Associates (Latimer) and is described below.

Latimer applied a regional dispersion model to estimate ambient PM concentrations in the 48-contiguous States. This dispersion model, the Lagrangian Regional Model (LRM), was applied to single emission sources. Because of the extensive computer requirements, it was not possible within the timeframe of the air quality modeling project to apply the LRM to all of the nearly 6,000 sources in the United States. Thus, the limited LRM results were used to guide the adjustment of the CRDM that was developed during the first phase of the work. The adjusted CRDM was applied to calculate a transfer matrix of S-R relationships for all relevant emissions and chemical species and to calculate cumulative regional ambient concentrations of $PM_{2.5}$ and PM_{10} as well as important chemical constituents including sulfate, nitrate, and secondary organics. The modified CRDM, when used with greatly scaled down primary PM emissions, provides comparable estimates of the spatial distribution of annual concentrations in the United States.

E.1.1 Lagrangian Regional Model (LRM)

A LRM approach was developed that calculates the transport, diffusion, deposition, and chemical conversion of emissions using a spatially and temporally varying wind field. The North American wind field was provided by EPA based on mesoscale model calculations carried out in 1994 for the meteorology of 1990. These data were reduced by Latimer to a smaller input file by calculating mixing height and average winds and relative humidities in the mixed layer.

[29] In Appendix F, we describe the C-R functions in detail.

The LRM was tested for a single point source using a few days of data. LRM is based on simple dispersion, deposition, and chemical conversion concepts used in HAZEPUFF (Latimer, 1993). Puffs are released hourly and transported by the averaged winds appropriate for the time and location of the puff. A single uniform concentration for each hourly puff is calculated by expanding the puff box using standard Pasquill-Gifford D_z values, limited by the mixed layer height, and mesoscale D_y values from(Gifford, 1982). Deposition is handled using deposition velocities applied to the ground-level concentrations. Sulfur oxidation is calculated at a rate that depends on relative humidity (rh) ranging from 0.5 percent/hour for rh<40 percent to 1.5 percent/hour for rh>70 percent. Nitrogen oxidation was assumed to take place at 2 percent/hour.

The LRM was successfully applied to a single source; however, the computer memory and run times were excessive to be able to set up LRM for the entire country with 6,000 sources and 3,000 receptors.

E.1.2 Climatological Regional Dispersion Model (CRDM)

CRDM uses assumptions similar to those in an EPA-recommended model, version 2 of the Industrial Source Complex Long Term model (ISC2LT), but incorporates terms for wet and dry deposition of gases and particles and chemical conversion of SO_2 and NO_x. CRDM employs as input climatological summaries (annual average mixing heights and joint frequency distributions of wind speed and direction) for 100 upper-air meteorological monitoring sites throughout North America.

The model uses Turner's sector-average approach, which is recommended for long-term average concentrations. Turner uses a probabilistic approach in which the frequency of occurrence of various wind and atmospheric stability conditions are used to calculate the frequency of transport in various sectors. Winds are divided into 16 cardinal wind directions (e.g., north, north-northeast, northeast, etc.). The area of each area source is determined from the area of the given county. The width of the area source is calculated as the square root of the county area.

The impact of a county on its own receptor was handled in a somewhat different manner. It was assumed that all emissions (area and point source aggregations) from the county are evenly distributed over a square with the same area as the county. The county centroid is the center of the square. The concentrations were calculated at the downwind edge of this square. It was assumed that emissions from the county are always impacting the county. A simple box model was used for each wind speed and stability category. Actual measured concentrations would be expected to be higher than those modeled with these assumptions if the monitor location was in, or generally downwind from, a portion of the county with emission densities much higher than the county average. On the other hand, concentrations would be expected to be lower if the monitor is located at the prevailing upwind edge of the county, or in an area of relatively low emission density. In addition, it should be noted that the most intensely urbanized portion of a county might be only a fraction of the county area; for example, this is the case in Los Angeles County.

The mass flux of a directly emitted primary species is dependent upon the amount of material initially emitted, as well as the amount chemically converted to a secondary pollutant, and the amount deposited by wet and dry processes during the transport time from the emission point to the downwind distance of the receptor. The mass flux of secondary pollutants is dependent upon the fraction of the primary species that is chemically converted in the atmosphere to the secondary species and the amount of the secondary species that is deposited by wet and dry deposition processes during the transport time from the stack to the downwind receptor. Dry deposition rates were selected as follows: 0.1 centimeters per second (cm/s) for all particles (including sulfates and nitrates), 0.5 cm/s for SO_2 and 1 cm/s for NO_x, gaseous nitrate, and NH_3.

Wet deposition rates were parameterized using wet deposition velocities from Yamartino (1985). These velocities are referenced to the annual precipitation rate (P; in inches) at the given location: 0.08P for particles, 0.008P for SO_2, 0.014P for NH_3, and 0.025P for $_{Nox}$.

The pseudo-first-order rate constant for deposition was calculated from these dry and wet deposition velocities by dividing by the mixing height (mh). The deposition rates of primary and secondary species are calculated by multiplying the concentration by the applicable deposition velocity.

The vertical diffusion parameter was calculated using the subroutine from EPA's ISC2 and SCREEN2 models. Atmospheric stabilities were assumed to be C class (slightly unstable) during the day and E class (slightly stable) at night. However, if winds were greater than 6 meters per second (m/s), stability was assumed to be neutral (class D). If the selected atmospheric stabilities are more stable than actual conditions, dispersion will be under-estimated and concentrations over-predicted.

Meteorological variables were calculated from NAMER-WINDTEMP rawinsonde data obtained from the National Climatic Data Center (NCDC). Winds for each of 100 sites throughout North America were averaged for the following layers: the surface to 250 meters above ground level (m agl), 250-500 m agl, 500-1,000 m agl, 1,000-2,000 m agl, and 2,000-4,000 m agl. For each of these levels and for each of the 100 meteorological sites, a joint frequency distribution of wind direction (16 cardinal directions) and wind speeds (11 speeds in 1 m/s increments) was calculated for 1990. These distributions were calculated separately for the twice-daily soundings. The early morning soundings were assumed to be associated with the E stability category, and the late afternoon soundings were assumed to be associated with the C stability category. The appropriate wind layer for concentration calculations was determined using the centroid of the diffusing plume.

Mixing heights were determined from each sounding by calculating the virtual potential temperature. The annual average afternoon mixing heights were calculated for each of the 100 meteorological sites and were used to calculate the upper limit of vertical diffusion (h_m).

E.2 EMISSION INPUTS USED FOR CRDM AIR QUALITY MODELING

NPI Version 3.0 emissions inputs to the CRDM were primarily at the county level, with four source type groupings: (1) area sources and point sources with (2) low (3) medium and (4) high effective stack heights. There are 3,080 counties in the 48 contiguous United States. Ground-level area source emissions were estimated for each of these counties. The NPI includes a total of 61,619 point sources - too many sources to model individually. Therefore, a scheme was developed to aggregate elevated point source emissions to the county level. The effective stack height of each of these sources was calculated for an average wind speed (5 m/s). Two aggregated elevated point source groupings were made: one for sources with effective stack heights less than 250 meters, and another for sources with effective stack heights between 250 and 500 meters. There were 1,887 counties with aggregated point source emissions in the first category, and 373 counties in the second category. Sources with effective stack heights greater than 500 meters were modeled individually. There were 565 such sources. Therefore, including the ground-level area sources, there were 5,905 sources modeled in the contiguous United States (3,080 + 1,887 + 373 + 565). The S-R matrix contains a source index number that corresponds to each of the aggregate sources.

In addition to U.S. emissions, Canadian and Mexican emissions were modeled. Canadian emissions were specified by province. It was assumed that the emissions for a given province were released from an area around the largest urban area (e.g., Montreal, Quebec, and Toronto). There were 10 Canadian provinces

modeled. There were 29 Mexican sources, including specific cities and states in northern Mexico. Thus, 5,944 North American sources were modeled.

For each source, primary (directly emitted) $PM_{2.5}$ and PM_{10} emissions were modeled; approximately 90 percent of primary PM_{10} and 70 percent of primary $PM_{2.5}$ emissions are estimated to result from natural and man-made fugitive dust sources. In addition to primary emissions, secondary components of $PM_{2.5}$ were estimated from the gaseous precursors. Secondary organics formed from anthropogenic and biogenic emissions were modeled using fractional aerosol coefficients; since these reactions occur within a few hours, these species were modeled similarly to primary PM. Emissions of SO_2, NO_x, and NH_3 were included in order to compute ammonium sulfate and ammonium nitrate concentrations.

The CRDM is used to develop a matrix of S-R transfer coefficients that link emissions from every county and major elevated point source in the United States, emissions from major Canadian urban areas, and emissions from the largest sources in northern Mexico, to PM air quality within every U.S. county, State centroid, Canadian province, and northern Mexican receptor. Each coefficient represents the incremental ambient air quality impact of a certain species at a given receptor from a particular area or point source. The natural source-apportionment capability of the CRDM allows for the entire matrix of air quality impacts to be expressed in terms of "normalized" increments, or more specifically, the mg/m^3 increment that occurs given each unit of emissions in mg/s. In this way, a multitude of emission scenarios by year and/or control strategy can be analyzed for their air quality impacts without requiring repetitive runs of CRDM itself. It simply requires the multiplication of an emission inventory with each S-R matrix, which yields the estimated air quality increments.

Four separate S-R matrices were developed using CRDM: (1) primary PM, appropriate for inert primary emissions of PM_{10} and $PM_{2.5}$ as well as anthropogenic and biogenic SOA (which are treated as primary inert species); (2) sulfate; (3) nitrate; and (4) NH_3. The specific size of each S-R matrix is 5,944 area and elevated points sources by 3,315 receptors (3,081 counties, 10 Canadian provinces, 29 Mexican areas, 147 Class I Areas, and 48 State centroids). To develop these matrices, CRDM was run with each source emitting at 1 mg/s, resulting in transfer coefficients with units of s/m^3.

E.3 ADJUSTMENTS TO S-R MATRIX

The S-R matrix was applied to a 1996 inventory to determine model-estimated 1996 air quality for each county in the 48 contiguous States. These results were used as the basis for the normalization adjustments described below. The same types of adjustments as were made in the PM NAAQS analysis were then applied:

- A fugitive dust adjustment factor of 0.25 was applied to primary $PM_{2.5}$ and PM_{10} emissions from fugitive dust sources, so that the contribution of this pollutant to total $PM_{2.5}$ concentrations better matched monitoring data. In addition, emissions from natural sources were removed from the inventory prior to normalization. This adjustment has little effect on the current analysis, since the current analysis models changes in motor vehicle emissions.

- The annual average modeled concentrations were compared with 1993-1995 monitoring data and normalization factors were applied so that the modeled concentrations would be equivalent to the monitored values. Normalization factors were applied equivalently to all pollutant species, so that the relative contributions of the individual pollutants to total PM mass do not change. All modeled results are normalized, regardless of over-prediction or under-prediction relative to monitored values.

Monitored county normalization factors are calculated from ambient concentrations supplied by EPA for counties where data exist (Tier 1, Tier 2, and Tier 3 where the tiers are based on completeness criteria, with Tier 1 being the most complete). Because of the lack of ambient $PM_{2.5}$ monitoring data, the ambient $PM_{2.5}$ data used for this analysis is statistically developed from the 1993-1995 ambient PM_{10} data set (Pechan, 1997a). The ambient concentrations are based on 1993 to 1995 PM_{10} monitoring data. The normalization factors for nonmonitored counties (Tier 4) are calculated as the average of factors determined for the 504 (Tier 1) monitored counties based on modeling region and county type (i.e., urban or nonurban). Outliers, identified as values not within two standard deviations of the average, were removed prior to the calculation of the average regional normalization factors.

E.4 Estimating the Parameters of a Gamma Distribution, Given the Mean and a Peak Value

We develop daily average and the median exposure estimates by first assuming that a gamma distribution is reasonably representative of the PM distribution, and then by using a maximum likelihood estimation procedure to estimate the gamma distribution parameters for each county most consistent with the mean and peak values.[30] A distribution of daily PM values is then estimated for both the baseline and the control scenario in each county, and then the estimated change in PM. This analysis assumes that the order of PM concentrations across days does not change from the baseline to any control scenario, so the change in PM on the n^{th} percentile day equals baseline PM on the n^{th} percentile day minus control scenario PM on the n^{th} percentile day.

Note that for PM_{10}, the peak value is defined as the value corresponding to the 99.7^{th} percentile value of the distribution of actual daily 24-hour average PM_{10} values. For $PM_{2.5}$, the peak value is defined as the value corresponding to the 98^{th} percentile value of the distribution of estimated daily 24-hour average $PM_{2.5}$ values. Also note that daily PM_{10} and $PM_{2.5}$ values derived from the gamma distribution generation procedure are adjusted to reflect the natural occurrence of background concentrations of PM_{10} and $PM_{2.5}$ (the level at which a given PM constituent exists naturally in the environment). Prior to the distribution estimation, an assumed background concentration is subtracted from the mean and peak PM concentrations used to predict the gamma distribution. Once the distribution of daily PM values is predicted, the background concentration is added back to the representative air quality value that has been estimated. In instances where the initial mean value is below a given background concentration assumption, estimates of daily air quality are generated directly from the mean and peak PM values without any background adjustment. Background concentrations are assumed to be $8ug/m^3$ for PM_{10} and $3.5ug/m^3$ for $PM_{2.5}$.

The gamma distribution has two parameters, which will be denoted as λ and r, that must be estimated for each county in order for the distribution of daily average PM concentrations to be completely specified. The parameters of a distribution are usually estimated from a random sample drawn from the distribution. Given a sample from the distribution, one of several possible standard methods (for example, maximum likelihood estimation or the method of moments) could be used to estimate the parameters, λ and r. Even given only the sample mean and the sample variance, λ and r could be estimated by the method of moments.

[30]We compared a number of different distributions with the distribution of actual PM observations and found the gamma distribution to be most representative.

However, neither the whole sample nor the sample variance are available. Instead, the only available information about the distribution is the sample mean and a peak statistic (e.g.,the eighth largest daily average is the 98th percentile point of 365 daily values). The following method, which combines aspects of both the method of moments and maximum likelihood estimation, was therefore used to estimate the two parameters of the gamma distribution from the available statistics.

As in the method of moments, equate the sample mean with the population mean, E(x). The population mean of a gamma distribution is:

$$E(X) = \frac{r}{l} .$$

Therefore, denoting the sample mean as x_s , set:

$$X_S = E(X) = \frac{r}{l} .$$

Solving for λ as a function of x_s and r yields:

$$l = \frac{r}{X_S} .$$

The first piece of information, the sample mean, has been used to reduce the problem from one of estimating two parameters to one of estimating only one parameter. An estimate of r will yield an estimate of λ, given the sample mean.

In the second step, the peak statistic (e.g., the eighth largest daily average PM concentration) is used to estimate r. The distribution of the peak can be derived from the distribution of the daily average PM concentrations.

The peak PM concentration has a probability density function (pdf) that is itself a function of the pdf of the daily PM concentration and the corresponding cumulative distribution function (cdf) of the daily PM concentration. (The cumulative distribution function describes the probability of being less than any given value.) In particular, if the daily average PM concentration is distributed according to a pdf denoted as f(x; λ, r), and the corresponding cumulative distribution function (cdf) is denoted as F(x; λ, r), then the probability density function of the peak, denoted as $f_{n-\alpha+1}(x;\lambda, r)$, can be shown to be:

$$f_{n-a+1}(x; l, r) = \frac{n!}{(a-1)!(n-a)!}\left[F(x; l, r)\right]^{a-1}\left[1 - F(x; l, r)\right]^{n-a} f(x; l, r) \ ,$$

where n=365 (because there are 365 days in a year) and α represents the peak (e.g., α=358 for the eighth highest $PM_{2.5}$ value out of 365 days)[31]. (Note that the pdf of any order statistic can be derived analogously.) Because λ is a function of r, there is only one unknown parameter that requires estimation.

Maximum likelihood estimation is used to estimate r in the pdf of the peak PM concentration, using the one observation from that pdf -- the peak PM concentration.

The method described above for estimating λ and r has two features that guarantee reasonable estimates. First, the method constrains the estimation of the two parameters so that the estimated population mean, which is a function of both parameters, equals the sample mean. This is reasonable, since the sample mean is the best guess at what the population mean is. Second, this method produces the "most likely" estimate of r, given this constraint. That is, it produces the value of r that maximizes the chance of having gotten the particular second daily maximum PM concentration.

To generate 365 daily PM concentrations from the distribution whose parameters are estimated, we could use Monte Carlo techniques. If the number of iterations in a Monte Carlo exercise is large enough, the frequency distribution of generated observations will approximate the distribution from which the observations were generated. The smaller the number of iterations, however, the rougher the approximation. Instead of generating observations by Monte Carlo techniques, values corresponding to evenly-spaced percentile points of the estimated distribution are used. This guarantees that the sample distribution will correspond to the assumed distribution. First, the percentile of the eighth highest concentration (given) is calculated from the estimated distribution. The percentiles of the 364 other concentrations are evenly spaced around this percentile. The percentile of the highest observation was set midway between the percentile of the second highest observation and the 100th percentile.

[31]The probability density function of the peak is from Mood et al. (1974, p. 254).

E.5 Interpolation of Air Quality Data to the CAPMS Grid Cell Centers

The annual mean and constructed median and daily average results are extrapolated from the county-centers to unmonitored locations to estimate PM levels at each CAPMS grid-cell based on Voronoi Neighbor Averaging (VNA). The value for a given CAPMS gridcell is calculated as follows:

$$CAPMS\ cell_{i,2018} = \sum_{h=1}^{N} County_{h,2018} \cdot d_{h,i}$$

where:

$CAPMS\ cell_{i,2018}$ = predicted PM concentration at CAPMS cell *i*

N = number of neighboring county centers for CAPMS gridcell i

$County_{h,2018}$ = 2018 PM level at county center h

$d_{h,i}$ = inverse-distance weight for cell *i* to county h .

Once we have estimates for both the baseline and control scenarios at each CAPMS grid cell, we take the difference between the baseline and control to estimate the impact of the policy. This is a straightforward calculation for an annual statistic like annual mean or median PM. Calculating changes in daily average PM at each CAPMS grid cell, however, requires additional processing. Recall that for the purposes of computational efficiency, we create 20 bins of PM data to represent a year's worth of PM data. We subtract the baseline value in the first bin from the control value in the first bin, and so on for each of the 20 bins. For each CAPMS gridcell, we then get 20 values representing the difference between the baseline and control, and we use these to estimate the change in adverse effects associated with the implementation of the policy. Note that since we are interested in PM values for the whole year, each binned value represents 18.25 days (365/20). We then multiply each of the 20 incidence change estimates by 18.25 to reconstruct an entire year's worth of incidence changes in the CAPMS grid cell.

E.6 RESULTS

Exhibits E-1 to E-3 present the reduction in health effects and the estimated value of these health effects for the "75 Percent Reduction", "All Power Plant", and "All Diesel" scenarios. Exhibits E-4 through E-11 present maps of ambient air quality for these scenarios and for the baseline.

Exhibit E-1 Estimated PM-Related Health Benefits Associated with Air Quality Changes Resulting from the S-R Matrix-Based "75 Percent Reduction" Scenario

Endpoint	Reference	Avoided Incidence (cases/year)			Monetary Benefits (millions 1999$)		
		5th %ile	Mean	95th %ile	5th %ile	Mean	95th %ile
MORTALITY							
Ages 30+, Mean, All Cause	Krewski et al. (2000)	6,870	12,300	17,400	9,760	75,200	170,000
CHRONIC ILLNESS							
Chronic Bronchitis	Pooled Analysis	2,560	7,450	12,900	232	2,450	8,040
HOSPITALIZATION							
COPD-Related	Samet et al. (2000)	295	1,290	2,300	4	16	28
Pneumonia-Related	Samet et al. (2000)	870	1,580	2,290	13	23	34
Asthma-Related	Sheppard et al. (1999)	481	1,190	1,860	3	8	13
Cardiovascular-Related	Samet et al. (2000)	3,250	3,810	4,410	60	70	81
Asthma-Related ER Visits	Schwartz et al. (1993)	1,140	2,740	4,260	0	1	1
MINOR ILLNESS							
Acute Bronchitis	Dockery et al. (1996)	-123	24,400	49,400	0	1	3
Upper Respiratory Symptoms	Pope et al. (1991)	88,500	265,000	441,000	2	6	14
Lower Respiratory Symptoms	Schwartz et al. (1994)	123,000	267,000	408,000	1	4	8
Asthma Attacks	Whittemore and Korn (1980)	81,400	236,000	390,000	3	10	20
Work Loss Days	Ostro (1987)	1,800,000	2,080,000	2,340,000	191	220	247
MRAD	Ostro and Rothschild (1989)	9,180,000	10,800,000	12,300,000	314	533	755
TOTAL PRIMARY PM-RELATED BENEFITS					-	78,500	-

Exhibit E-2 Estimated PM-Related Health and Benefits Associated with Air Quality Changes Resulting from the S-R Matrix-Based All Power Plant Scenario

Endpoint	Reference	Avoided Incidence (cases/year)			Monetary Benefits (millions 1999$)		
		5th %ile	Mean	95th %ile	5th %ile	Mean	95th %ile
MORTALITY							
Ages 30+, Mean, All Cause	Krewski et al. (2000)	10,600	18,900	26,800	15,000	116,000	261,000
CHRONIC ILLNESS							
Chronic Bronchitis	Pooled Analysis	4,400	12,500	21,200	384	4,110	13,500
HOSPITALIZATION							
COPD-Related	Samet et al. (2000)	507	2,230	3,960	6	28	49
Pneumonia-Related	Samet et al. (2000)	1,500	2,720	3,940	22	40	58
Asthma-Related	Sheppard et al. (1999)	746	1,850	2,890	5	13	20
Cardiovascular-Related	Samet et al. (2000)	5,590	6,560	7,590	103	121	140
Asthma-Related ER Visits	Schwartz et al. (1993)	1,980	4,750	7,380	1	1	2
MINOR ILLNESS							
Acute Bronchitis	Dockery et al. (1996)	-192	37,600	75,400	0	2	5
Upper Respiratory Symptoms	Pope et al. (1991)	153,000	458,000	763,000	3	11	24
Lower Respiratory Symptoms	Schwartz et al. (1994)	190,000	411,000	627,000	2	6	12
Asthma Attacks	Whittemore and Korn (1980)	141,000	407,000	672,000	4	17	35
Work Loss Days	Ostro (1987)	2,790,000	3,210,000	3,610,000	295	340	382
MRAD	Ostro and Rothschild (1989)	14,200,000	16,600,000	18,900,000	484	804	1,160
TOTAL PRIMARY PM-RELATED BENEFITS					-	121,000	-

Exhibit E-3 Estimated PM-Related Health and Benefits Associated with Air Quality Changes Resulting from the S-R Matrix-Based No-Diesel Scenario

Endpoint	Reference	Avoided Incidence (cases/year)			Monetary Benefits (millions 1999$)		
		5th %ile	Mean	95th %ile	5th %ile	Mean	95th %ile
MORTALITY							
Ages 30+, Mean, All Cause	Krewski et al. (2000)	8,640	15,400	21,800	12,300	94,500	213,000
CHRONIC ILLNESS							
Chronic Bronchitis	Pooled Analysis	3,900	11,100	18,800	341	3,660	12,000
HOSPITALIZATION							
COPD-Related	Samet et al. (2000)	422	1,850	3,300	5	23	41
Pneumonia-Related	Samet et al. (2000)	1,250	2,260	3,280	18	33	48
Asthma-Related	Sheppard et al. (1999)	683	1,690	2,650	5	12	18
Cardiovascular-Related	Samet et al. (2000)	4,650	5,460	6,320	86	100	116
Asthma-Related ER Visits	Schwartz et al. (1993)	1,800	4,330	6,730	1	1	2
MINOR ILLNESS							
Acute Bronchitis	Dockery et al. (1996)	-173	33,900	68,200	0	2	5
Upper Respiratory Symptoms	Pope et al. (1991)	137,000	411,000	683,000	3	10	22
Lower Respiratory Symptoms	Schwartz et al. (1994)	170,000	368,000	560,000	2	6	11
Asthma Attacks	Whittemore and Korn (1980)	127,000	367,000	606,000	4	15	32
Work Loss Days	Ostro (1987)	2,550,000	2,940,000	3,300,000	270	311	349
MRAD	Ostro and Rothschild (1989)	12,900,000	15,200,000	17,200,000	442	735	1,060
TOTAL PRIMARY PM-RELATED BENEFITS					-	99,400	-

Exhibit E-4 Annual Mean $PM_{2.5}$ Level in 2007: S-R Matrix Baseline Scenario

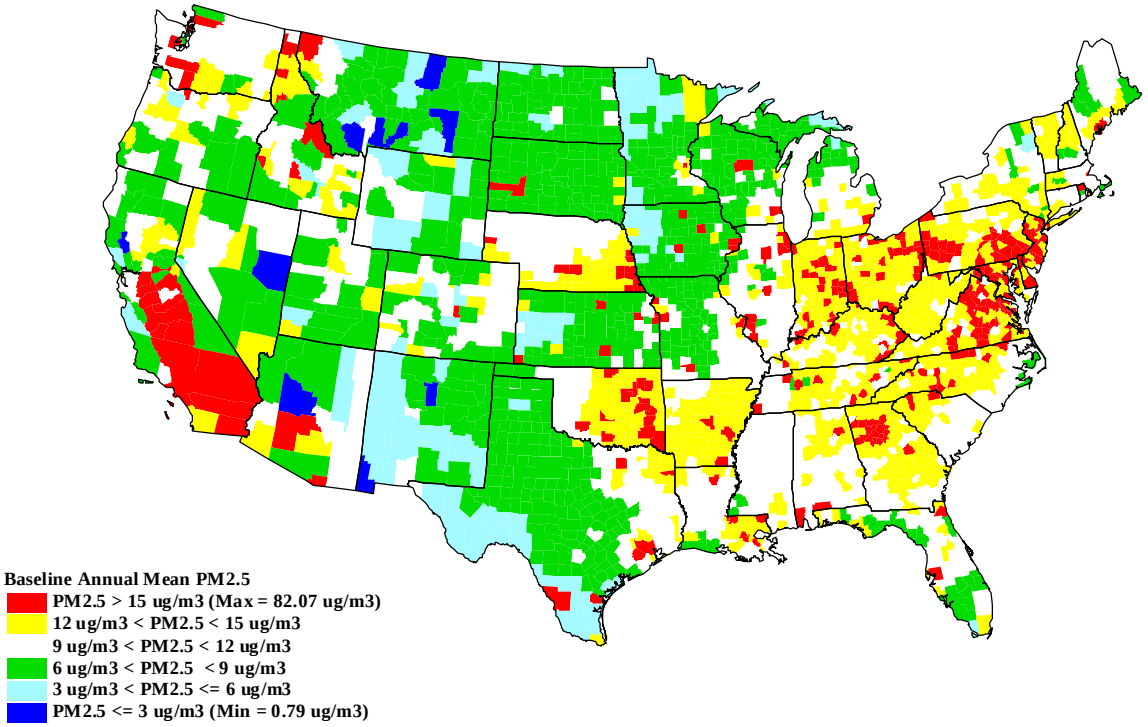


Exhibit E-5 Change in Annual Mean $PM_{2.5}$ Levels in 2007: S-R Matrix "75 Percent Reduction" Scenario

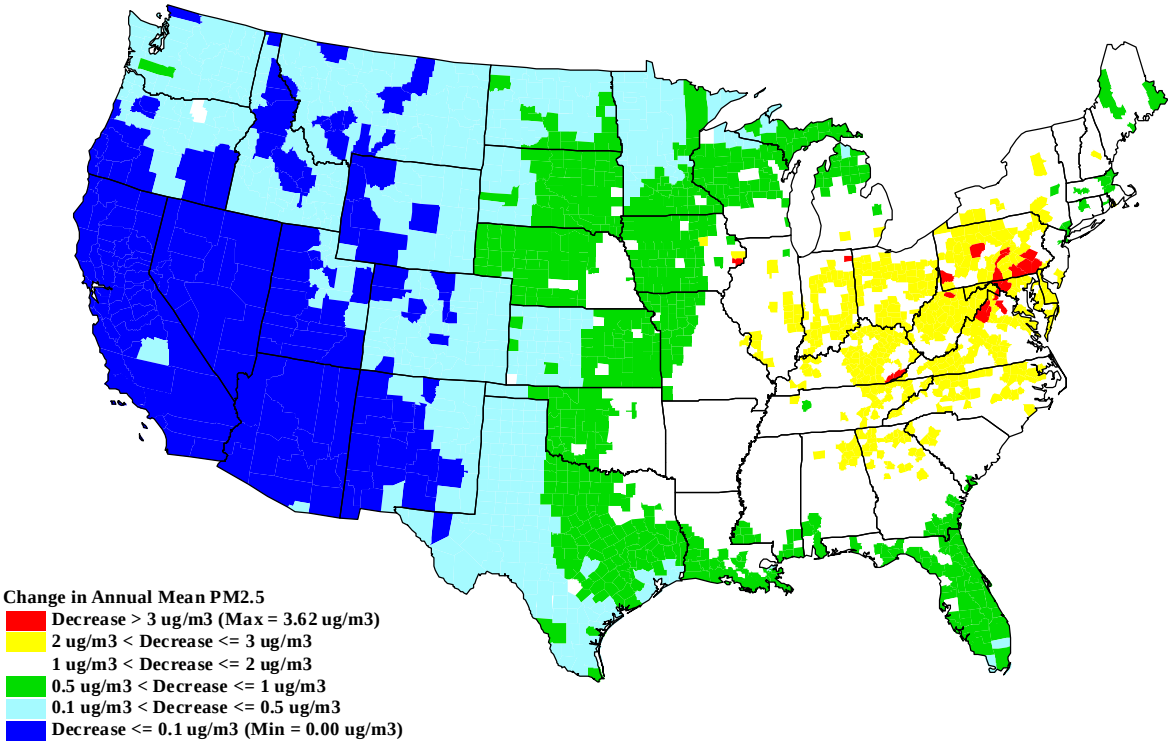


Exhibit E-6 Change in Annual Mean $PM_{2.5}$ Levels in 2007: S-R Matrix "All Power Plant" Scenario

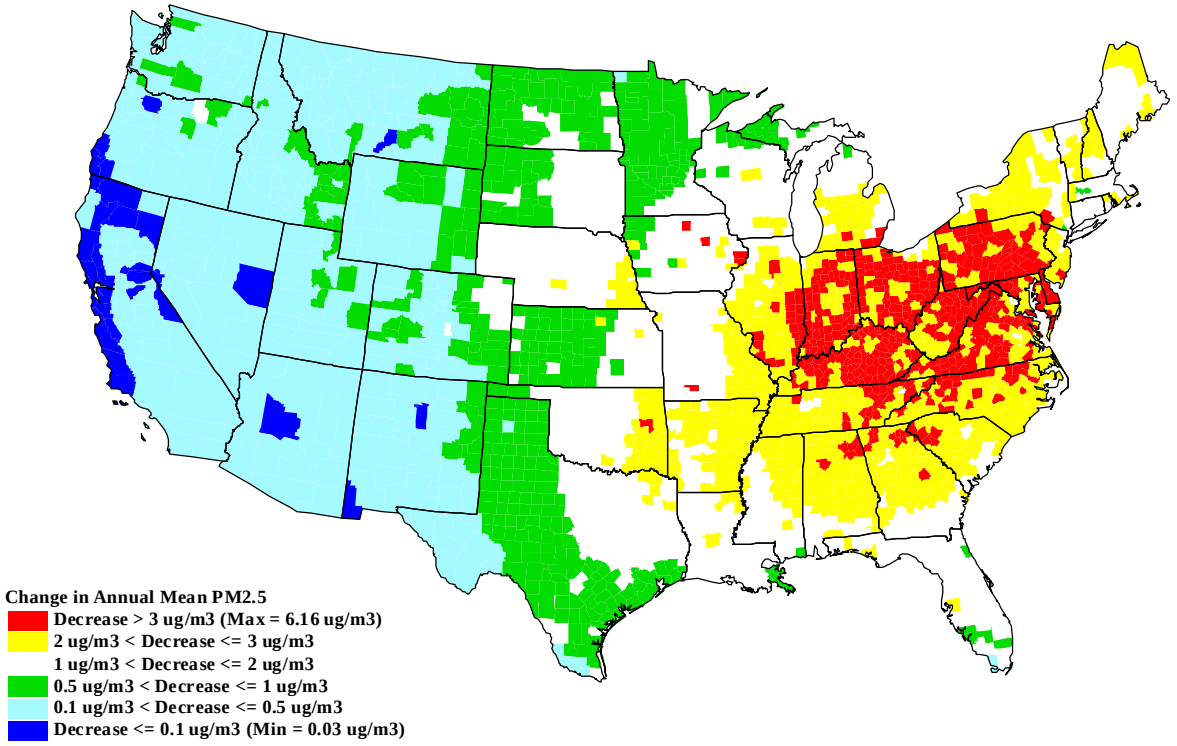


Exhibit E-7 Change in Annual Mean $PM_{2.5}$ Levels in 2007: S-R Matrix "No-Diesel" Scenario

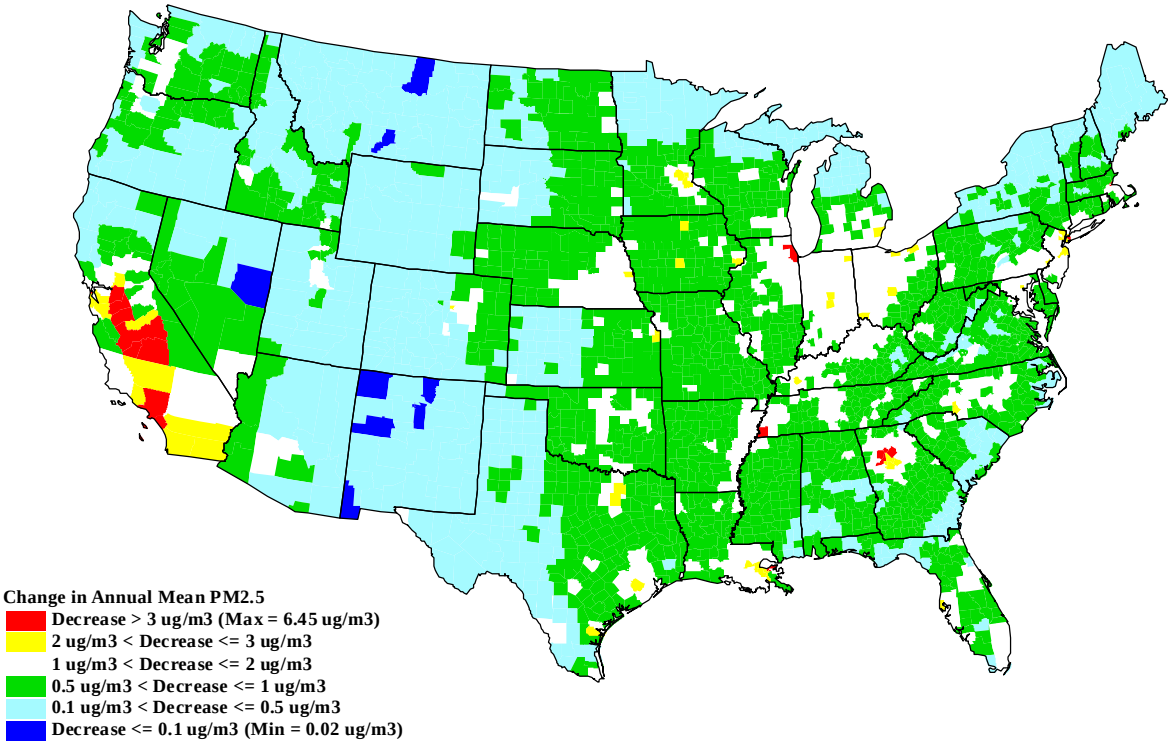


Exhibit E-8 Annual Mean PM_{10} Level in 2007: S-R Matrix Baseline Scenario

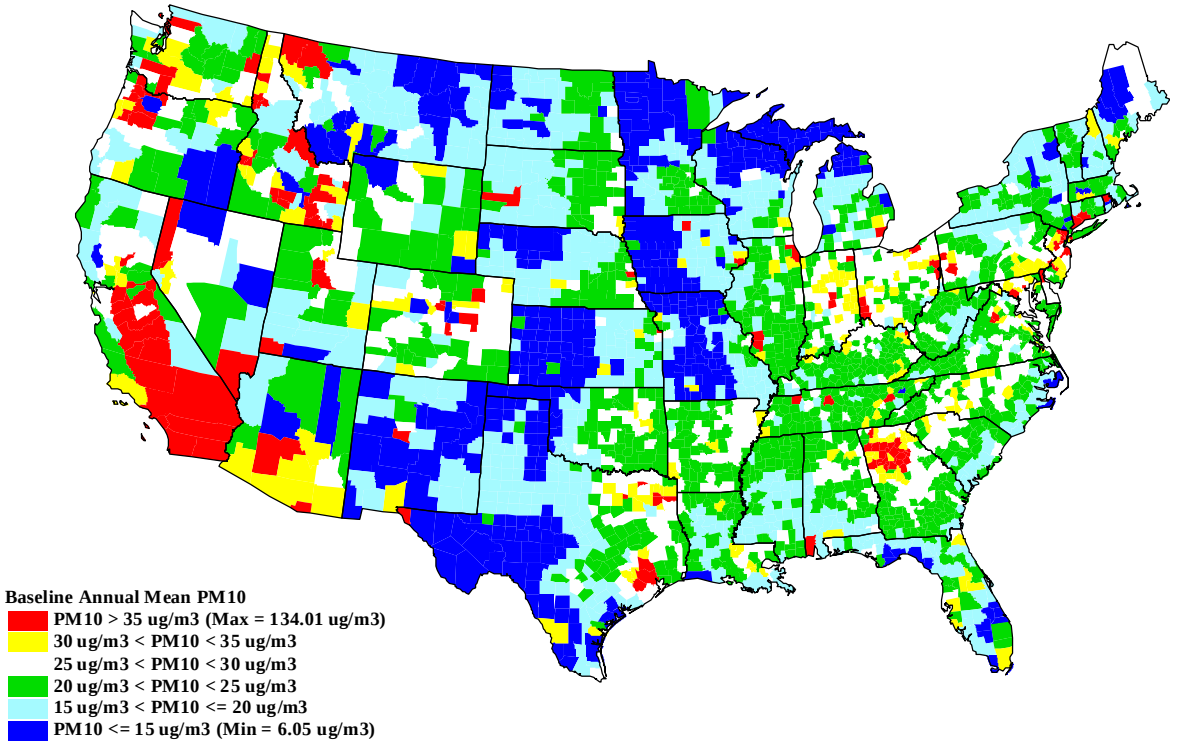


Exhibit E-9 Change in Annual Mean PM_{10} Levels in 2007: S-R Matrix "75 Percent Reduction" Scenario

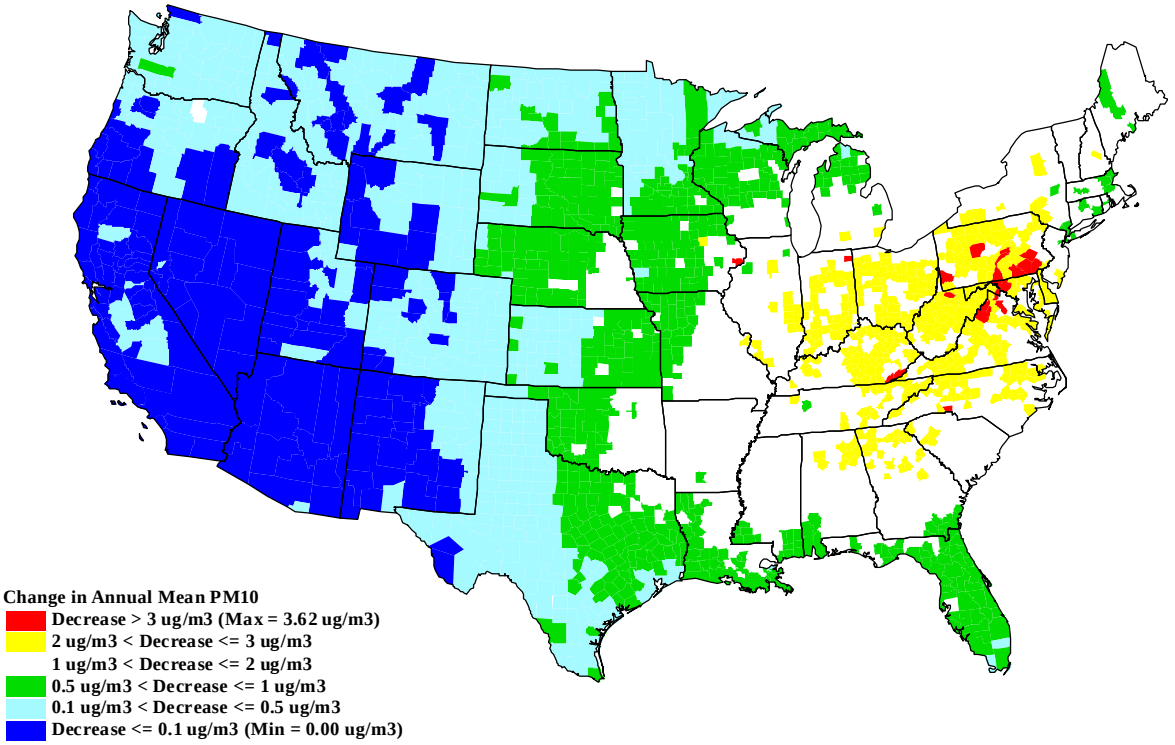


Exhibit E-10 Change in Annual Mean PM_{10} Levels in 2007: S-R Matrix "All Power Plant" Scenario

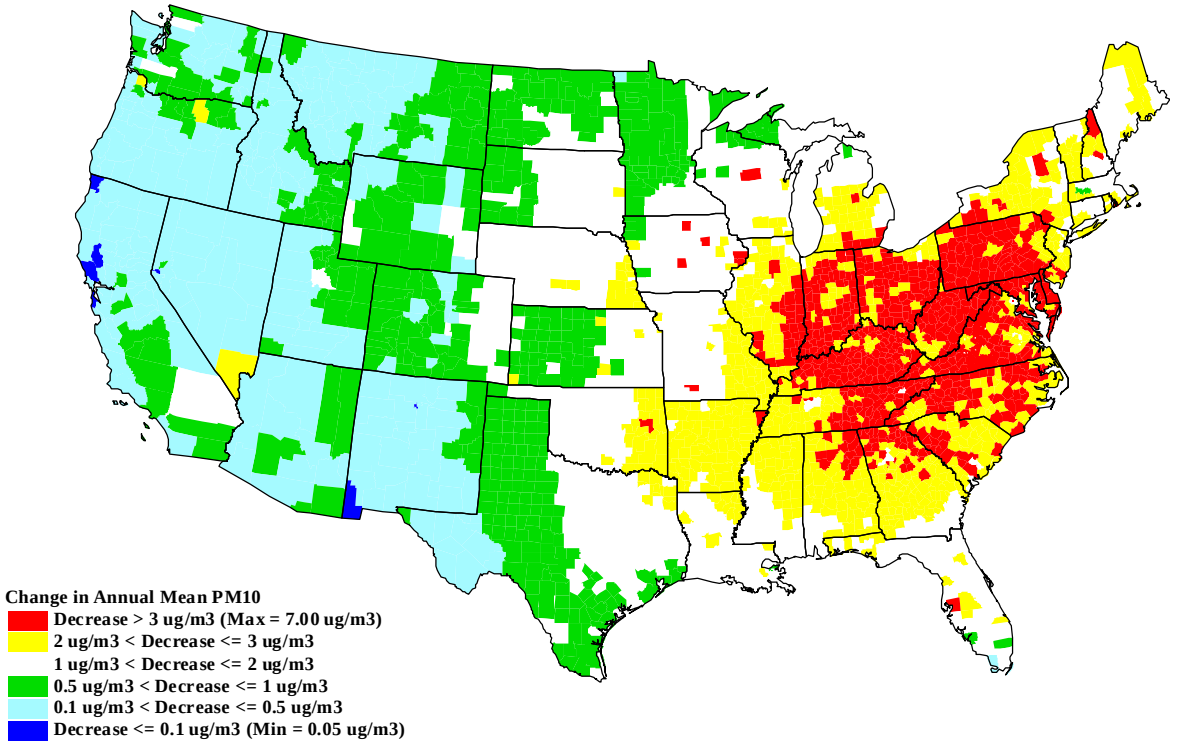
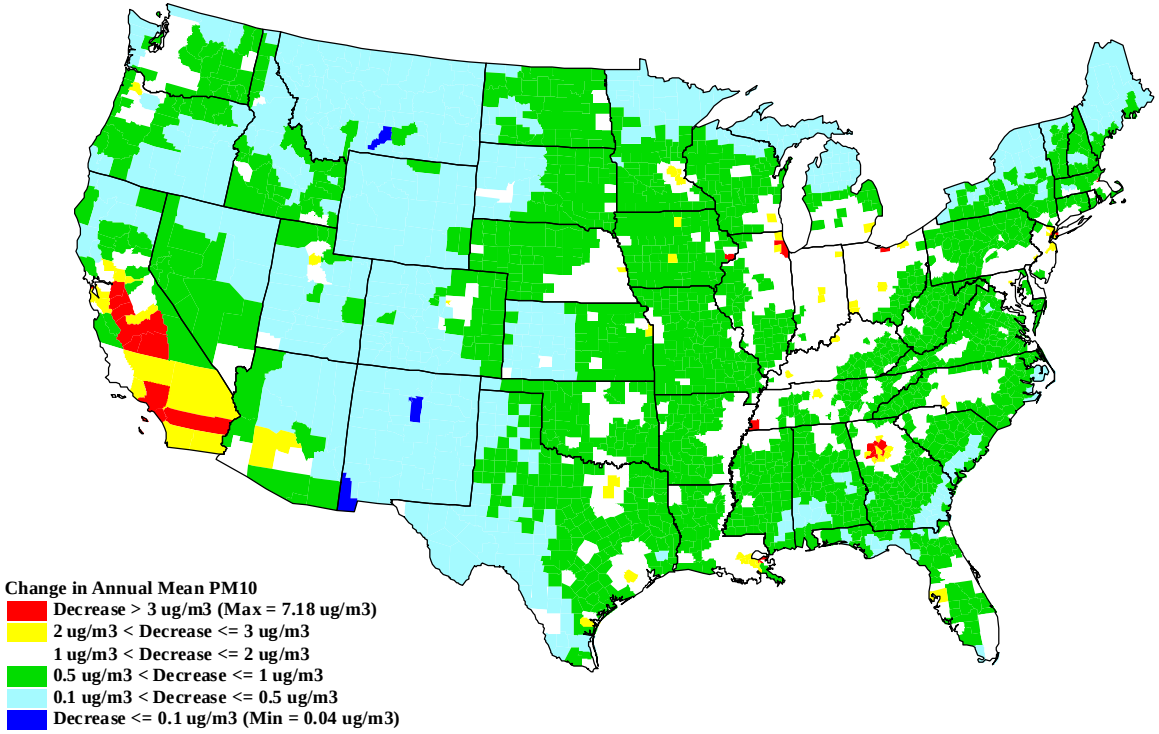


Exhibit E-11 Change in Annual Mean PM_{10} Levels in 2007: S-R Matrix "No-Diesel" Scenario



APPENDIX F: PARTICULATE MATTER C-R FUNCTIONS

Appendix F describes the concentration-response functions that we use in this analysis. Note that for all of the concentration-response functions we define ΔPM as $PM_{baseline}$ - $PM_{control}$, and we define the change in incidence as: - ($incidence_{control}$ - $incidence_{baseline}$).

F.1 MORTALITY

There are two types of exposure to PM that may result in premature mortality. Short-term exposure may result in excess mortality on the same day or within a few days of exposure. Long-term exposure over, say, a year or more, may result in mortality in excess of what it would be if PM levels were generally lower, although the excess mortality that occurs will not necessarily be associated with any particular episode of elevated air pollution levels. In other words, long-term exposure may capture a facet of the association between PM and mortality that is not captured by short-term exposure.

F.1.1 Mortality (Krewski et al., 2000) Based on ACS Cohort: Mean $PM_{2.5}$

The C-R function to estimate the change in long-term mortality is:

$$\Delta Mortality = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{2.5}} - 1)\right] \cdot pop,$$

where:

y_0 = county-level all-cause annual death rate per person ages 30 and older
β = $PM_{2.5}$ coefficient = 0.0046257
$\Delta PM_{2.5}$ = change in annual <u>mean</u> $PM_{2.5}$ concentration
pop = population of ages 30 and older
σ_β = standard error of β = 0.0012046

Incidence Rate. To estimate county-specific baseline mortality incidence among individuals ages 30 and over, this analysis used the average annual all-cause county mortality rate from 1994 through 1996 (U.S. Centers for Disease Control, 1999). Note that the Krewski et al. (2000) replication of Pope et al. (1995) used the same all-cause mortality when estimating the impact of PM.

Coefficient Estimate (β). The coefficient (β) is estimated from the relative risk (1.12) associated with a change in mean exposure of 24.5 μg/m^3 (based on the range from the original ACS study) (Krewski et al., 2000, Part II - Table 31, 63 city Dichotomous sampler).

$$b = \frac{\ln(1.12)}{(24.5)} = 0.0046257.$$

Standard Error (σ_β). The standard error (σ_β) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Krewski et al., 2000, Part II - Table 31).

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.19)}{24.5} - \frac{\ln(1.12)}{24.5}\right)}{1.96} = 0.0012625$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.12)}{24.5} - \frac{\ln(1.06)}{24.5}\right)}{1.96} = 0.0011466$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.0012046$$

F.1.2 Mortality (Krewski et al., 2000), Based on ACS Cohort: Median $PM_{2.5}$

The C-R function to estimate the change in long-term mortality is:

$$\Delta Mortality = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{2.5}} - 1)\right] \cdot pop,$$

where:
y_0 = county-level all-cause annual death rate per person ages 30 and older
β = $PM_{2.5}$ coefficient = 0.0053481
$\Delta PM_{2.5}$ = change in annual median $PM_{2.5}$ concentration
pop = population of ages 30 and older
σ_β = standard error of β = 0.0014638

Incidence Rate. To estimate county-specific baseline mortality incidence among individuals ages 30 and over, this analysis used the average annual county mortality rate from 1994 through 1996 (U.S. Centers for Disease Control, 1999). Note that the Krewski et al. (2000) replication of Pope et al. (1995) used the same all-cause mortality when estimating the impact of PM.

Coefficient Estimate (β). The coefficient (β) is estimated from the relative risk (1.14) associated with a change in median exposure of 24.5 $\mu g/m^3$ (based on original ACS study) (Krewski et al., 2000, Part II - Table 31):

$$b = \frac{\ln(1.14)}{(24.5)} = 0.0053481.$$

Standard Error (σ_β). The standard error (σ_β) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Krewski et al., 2000, Part II - Table 31):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.22)}{24.5} - \frac{\ln(1.14)}{24.5}\right)}{1.96} = 0.0014124$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.14)}{24.5} - \frac{\ln(1.06)}{24.5}\right)}{1.96} = 0.0015152$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.0014638$$

F.1.3 Mortality (Krewski et al., 2000), Based on ACS Cohort, Random Effects with Regional Adjustment: Median $PM_{2.5}$

The C-R function to estimate the change in long-term mortality is:

$$\Delta Mortality = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{2.5}} - 1)\right] \cdot pop,$$

where:

y_0 = county-level all-cause annual death rate per person ages 30 and older
β = $PM_{2.5}$ coefficient = 0.00605796
$\Delta PM_{2.5}$ = change in annual median $PM_{2.5}$ concentration
pop = population of ages 30 and older
σ_β = standard error of β = 0.0033826

Incidence Rate. To estimate county-specific baseline mortality incidence among individuals ages 30 and over, this analysis used the average annual county mortality rate from 1994 through 1996 (U.S. Centers for Disease Control, 1999). Note that the Krewski et al. (2000) replication of Pope et al. (1995) used the same all cause mortality when estimating the impact of PM.

Coefficient Estimate (β). The coefficient (β) is estimated from the relative risk (1.16) associated with a change in median exposure of 24.5 $\mu g/m^3$ (based on original ACS study) (Krewski et al., 2000, Part II - Table 46):

$$b = \frac{ln(1.16)}{(24.5)} = 0.00605796 .$$

Standard Error (σ_β). The standard error (σ_β) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Krewski et al., 2000, Part II - Table 46):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{ln(1.37)}{24.5} - \frac{ln(1.16)}{24.5}\right)}{1.96} = 0.0034650$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{ln(1.16)}{24.5} - \frac{ln(0.99)}{24.5}\right)}{1.96} = 0.0033001$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.0033826$$

F.1.4 Mortality (Krewski et al., 2000), Based on ACS Cohort, Random Effects with Independent Cities: Median $PM_{2.5}$

The C-R function to estimate the change in long-term mortality is:

$$\Delta Mortality = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{2.5}} - 1)\right] \cdot pop,$$

where:

y_0 = county-level all-cause annual death rate per person ages 30 and older
β = $PM_{2.5}$ coefficient = 0.0103936
$\Delta PM_{2.5}$ = change in annual median $PM_{2.5}$ concentration
pop = population of ages 30 and older
σ_β = standard error of β = 0.0029021

Incidence Rate. To estimate county-specific baseline mortality incidence among individuals ages 30 and over, this analysis used the average annual county mortality rate from 1994 through 1996 (U.S. Centers for Disease Control, 1999). Note that the Krewski et al. (2000) replication of Pope et al. (1995) used the same all cause mortality when estimating the impact of PM.

Coefficient Estimate (β). The coefficient (β) is estimated from the relative risk (1.29) associated with a change in median exposure of 24.5 $\mu g/m^3$ (based on original ACS study) (Krewski et al., 2000, Part II - Table 46):

$$b = \frac{ln(1.29)}{(24.5)} = 0.0103936.$$

Standard Error (σ_β). The standard error (σ_β) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Krewski et al., 2000, Part II - Table 46):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{ln(1.48)}{24.5} - \frac{ln(1.29)}{24.5}\right)}{1.96} = 0.0028613$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{ln(1.29)}{24.5} - \frac{ln(1.12)}{24.5}\right)}{1.96} = 0.0029428$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.0029021$$

F.1.5 Mortality (Pope et al., 1995), Based on ACS Cohort: Median $PM_{2.5}$

The C-R function to estimate the change in long-term mortality is:

$$\Delta Mortality = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{2.5}} - 1)\right] \cdot pop,$$

where:
y_0 = county-level all-cause annual death rate per person ages 30 and older
β = $PM_{2.5}$ coefficient = 0.006408
$\Delta PM_{2.5}$ = change in annual median $PM_{2.5}$ concentration
pop = population of ages 30 and older
σ_β = standard error of β = 0.001509

Incidence Rate. To estimate county-specific baseline mortality incidence among individuals ages 30 and over, this analysis used the average annual county mortality rate from 1994 through 1996 (U.S. Centers for Disease Control, 1999). Note that Pope et al. (1995) used all cause mortality when estimating the impact of PM.

Coefficient Estimate (β). The coefficient (β) is estimated from the relative risk (1.17) associated with a change in median exposure going from 9 $\mu g/m^3$ to 33.5 $\mu g/m^3$ (Pope et al., 1995, Table 2).

$$b = \frac{\ln(1.17)}{(33.5 - 9)} = 0.006408.$$

Standard Error (σ_β). The standard error (σ_β) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Pope et al., 1995, Table 2).

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.26)}{24.5} - \frac{\ln(1.17)}{24.5}\right)}{1.96} = 0.001543$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.17)}{24.5} - \frac{\ln(1.09)}{24.5}\right)}{1.96} = 0.001475$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.001509.$$

F.1.6 Mortality (Krewski et al., 2000), Based on Six-City Cohort: Mean $PM_{2.5}$

The C-R function to estimate the change in long-term mortality is:

$$\Delta Mortality = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{2.5}} - 1)\right] \cdot pop,$$

where:
y_0 = county-level all-cause annual death rate per person ages 25 and older
β = $PM_{2.5}$ coefficient = 0.013272
$\Delta PM_{2.5}$ = change in annual mean $PM_{2.5}$ concentration
pop = population of ages 25 and older
σ_β = standard error of β = 0.004070

Incidence Rate. To estimate county-specific baseline mortality incidence among individuals ages 25 and over, this analysis used the average annual county mortality rate from 1994 through 1996 (U.S. Centers for Disease Control, 1999). The Krewski et al. (2000) reanalysis of Dockery et al. (1993, p. 1754) appears to have used all-cause mortality when estimating the impact of PM.

Coefficient Estimate (β). The coefficient (β) is estimated from the relative risk (1.28) associated with a change in mean exposure going from 11.0 $\mu g/m^3$ to 29.6 $\mu g/m^3$ (Krewski et al., 2000, Part I - Table 19c):

$$b = \frac{\ln(1.28)}{(29.6 - 11)} = 0.013272 .$$

Standard Error (σ_β). The standard error (σ_β) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Krewski et al., 2000, Part I - Table 19c):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.48)}{18.6} - \frac{\ln(1.28)}{18.6}\right)}{1.96} = 0.003982$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.28)}{18.6} - \frac{\ln(1.10)}{18.6}\right)}{1.96} = 0.004157$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.004070$$

F.1.7 Mortality (Dockery et al., 1993), Based on Six-City Cohort: Mean $PM_{2.5}$

Dockery et al. (1993) examined the relationship between PM exposure and mortality in a cohort of 8,111 individuals aged 25 and older, living in six U.S. cities. They surveyed these individuals in 1974-1977 and followed their health status until 1991. While they used a smaller sample of individuals from fewer cities than the study by Pope et al., they used improved exposure estimates, a slightly broader study population (adults aged 25 and older), and a follow-up period nearly twice as long as that of Pope et al. (1995). Perhaps because of these differences, Dockery et al. study found a larger effect of PM on premature mortality than that found by Pope et al.

The C-R function to estimate the change in long-term mortality is:

$$\Delta\, Mortality = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{2.5}} - 1)\right] \cdot pop,$$

where:

y_0 = county-level all-cause annual death rate per person ages 25 and older
β = $PM_{2.5}$ coefficient = 0.0124
$\Delta PM_{2.5}$ = change in annual mean $PM_{2.5}$ concentration
pop = population of ages 25 and older
σ_β = standard error of β = 0.00423

Incidence Rate. Dockery et al. (1993, p. 1754) appear to have used all-cause mortality when estimating the impact of PM. To estimate county-specific baseline mortality incidence among individuals ages 25 and over, this analysis used the average all-cause annual county mortality rate from 1994 through 1996 (U.S. Centers for Disease Control, 1999).

Coefficient Estimate (β). The coefficient (β) is estimated from the relative risk (1.26) associated with a change in mean exposure going from 11.0 $\mu g/m^3$ to 29.6 $\mu g/m^3$ (Dockery et al., 1993, Tables 1 and 5):

$$b = \frac{\ln(1.26)}{(29.6 - 11)} = 0.0124.$$

Standard Error (σ_β). The standard error (σ_β) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Dockery et al., 1993, Table 5):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.47)}{18.6} - \frac{\ln(1.26)}{18.6}\right)}{1.96} = 0.00423$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.26)}{18.6} - \frac{\ln(1.08)}{18.6}\right)}{1.96} = 0.00423$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.00423.$$

F.2 CHRONIC MORBIDITY

Schwartz (1993) and Abbey et al. (1993; 1995b) provide evidence that PM exposure over a number of years gives rise to the development of chronic bronchitis in the U.S., and a recent study by McDonnell et al. (1999) provides evidence that ozone exposure is linked to the development of asthma in adults. These results are consistent with research that has found chronic exposure to pollutants leads to declining pulmonary functioning (Detels et al., 1991; Ackermann-Liebrich et al., 1997; Abbey et al., 1998).[32]

We estimate the changes in the new cases of chronic bronchitis by pooling the estimates from the studies by Schwartz (1993) and Abbey et al. (1995b). The Schwartz study is somewhat older and uses a cross-sectional design, however, it is based on a national sample, unlike the Abbey et al. study which is based on a sample of California residents.

F.2.1 Chronic Bronchitis (Schwartz, 1993)

Schwartz (1993) examined survey data collected from 3,874 adults ranging in age from 30 to 74, and living in 53 urban areas in the U.S. The survey was conducted between 1974 and 1975, as part of the National Health and Nutrition Examination Survey, and is representative of the non-institutionalized U.S. population. Schwartz (1993, Table 3) reported chronic bronchitis prevalence rates in the study population by age, race, and gender. Non-white males under 52 years old had the lowest rate (1.7%) and white males 52 years and older had the highest rate (9.3%). The study examined the relationship between the prevalence of reported chronic bronchitis, asthma, shortness of breath (dyspnea) and respiratory illness[33], and the annual levels of TSP, collected in the year prior to the survey (TSP was the only pollutant examined in this study). TSP was significantly related to the prevalence of chronic bronchitis, and marginally significant for respiratory illness. No effect was found for asthma or dyspnea.

Schwartz (1993) examined the *prevalence* of chronic bronchitis, not its *incidence*. To use Schwartz's study and still estimate the change in incidence, there are at least two possible approaches. The first is to simply assume that it is appropriate to use the baseline *incidence* of chronic bronchitis in a C-R function with the estimated coefficient from Schwartz's study, to directly estimate the change in incidence. The second is to estimate the percentage change in the prevalence rate for chronic bronchitis using the estimated coefficient from Schwartz's study in a C-R function, and then to assume that this percentage change applies to a baseline incidence rate obtained from another source. (That is, if the prevalence declines by 25 percent with a drop in PM, then baseline incidence drops by 25 percent with the same drop in PM.) This analysis is using the latter approach, and estimates a percentage change in prevalence which is then applied to a baseline incidence rate.

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[32] There are a limited number of studies that have estimated the impact of air pollution on chronic bronchitis. An important hindrance is the lack of health data and the associated air pollution levels over a number of years.

[33] Respiratory illness defined as a significant condition, coded by an examining physician as ICD-8 code 460-519.

The C-R function to estimate the change in chronic bronchitis is:

$$\Delta Chronic\ Bronchitis = -\left[\frac{y_0}{(1-y_0)\cdot e^{\Delta PM_{10}\cdot b} + y_0} - y_0\right]\cdot\left[\frac{z_0}{y_0}\right]\cdot pop\ ,$$

where:

y_0 = national chronic bronchitis prevalence rate for individuals 18 and older (Adams and Marano, 1995, Table 62 and 78) = 0.0535

z_0 = annual bronchitis incidence rate per person (Abbey et al., 1993, Table 3) = 0.00378

β = estimated PM_{10} logistic regression coefficient = 0.0123

ΔPM_{10} = change in annual average PM_{10} concentration

pop = population of ages 30 and older without chronic bronchitis = 0.9465*population 30+

σ_β = standard error of β = 0.00434 .

Prevalence Rate. The national chronic bronchitis prevalence rate was not available for individuals 30 and older. Instead, we used the prevalence rate for individuals 18 and older (Adams and Marano, 1995, Table 62 and 78). The 1994 national figures are the latest available, and are suggested here.

Incidence Rate. The annual incidence rate is derived by taking the number of new cases (234), dividing by the number of individuals in the sample (3,310), as reported by Abbey et al.(1993, Table 3), dividing by the ten years covered in the sample, and then multiplying by one minus the reversal rate (the percentage of reversals is estimated to be 46.6% based on Abbey et al. (1995a, Table 1)). Using the same data base, Abbey et al. (1995a, Table 1) reported the incidences by three age groups (25-54, 55-74, and 75+) for "cough type" and "sputum type" bronchitis, but they did not report an overall incidence rate for bronchitis.

Coefficient Estimate (β). The estimated logistic coefficient (β) is based on the odds ratio (= 1.07) associated with 10 $\mu g/m^3$ change in TSP (Schwartz, 1993, p. 9). Assuming that PM_{10} is 55 percent of TSP[34] and that particulates greater than ten micrometers are harmless, the coefficient is calculated as follows:

$$b_{PM_{10}} = \frac{ln(1.07)}{0.55\cdot 10} = 0.0123.$$

[34]The conversion of TSP to PM_{10} is from ESEERCO (1994, p. V-5), who cited studies by EPA (1986) and the California Air Resources Board (1982).

Standard Error (σ_β) The standard error for the coefficient (σ_β) is calculated from the reported lower and upper bounds of the odds ratio (1.02 to 1.12) (Schwartz, 1993, p. 9):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.12)}{0.55 \cdot 10} - \frac{\ln(1.07)}{0.55 \cdot 10}\right)}{1.96} = 0.00424$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.07)}{0.55 \cdot 10} - \frac{\ln(1.02)}{0.55 \cdot 10}\right)}{1.96} = 0.00444$$

$$s_b = \frac{s_{b,high} + s_{b,low}}{2} = 0.00434 .$$

Population. The study population in Schwartz (1993) includes 3,874 individuals over the age of 30, living in 57 urban areas in the United States. To what extent the study should be applied to individuals under the age of 30 is unclear, and no effect is assumed for these individuals.

F.2.2 Chronic Bronchitis (Abbey et al., 1995b, California)

Abbey et al. (1995b) examined the relationship between estimated $PM_{2.5}$ (annual mean from 1966 to 1977), PM_{10} (annual mean from 1973 to 1977) and TSP (annual mean from 1973 to 1977) and the same chronic respiratory symptoms in a sample population of 1,868 Californian Seventh Day Adventists. The initial survey was conducted in 1977 and the final survey in 1987. To ensure a better estimate of exposure, the study participants had to have been living in the same area for an extended period of time. In single-pollutant models, there was a statistically significant $PM_{2.5}$ relationship with development of chronic bronchitis, but not for AOD or asthma; PM_{10} was significantly associated with chronic bronchitis and AOD; and TSP was significantly associated with all cases of all three chronic symptoms. Other pollutants were not examined.

The C-R function to estimate the change in chronic bronchitis is:

$$\Delta Chronic\ Bronchitis = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{2.5}} - 1)\right] \cdot pop,$$

where:

y_0 = annual bronchitis incidence rate per person (Abbey et al., 1993, Table 3) = 0.00378
β = estimated $PM_{2.5}$ logistic regression coefficient = 0.0132
$\Delta PM_{2.5}$ = change in annual average $PM_{2.5}$ concentration
pop = population of ages 27 and older without chronic bronchitis[35] = 0.9465*population 27+

[35]Using the same data set, Abbey et al. (1995a, p. 140) reported that the respondents in 1977 ranged in age from 27 to 95. Chronic bronchitis prevalence from Adams and Marano (1995, Tables 62 and 78).

σ_β = standard error of β = 0.00680

Incidence Rate. The annual incidence rate is derived by taking the number of new cases (234), dividing by the number of individuals in the sample (3,310), as reported by Abbey et al.(1993, Table 3), dividing by the ten years covered in the sample, and then multiplying by one minus the reversal rate (estimated to be 46.6% based on Abbey et al. (1995a, Table 1)). Using the same data base, Abbey et al. (1995a, Table 1) reported the incidences by three age groups (25-54, 55-74, and 75+) for "cough type" and "sputum type" bronchitis, but they did not report an overall incidence rate for bronchitis.

Coefficient Estimate (β). The estimated coefficient (β) is based on the relative risk (= 1.81) associated with 45 μg/m^3 change in $PM_{2.5}$ (Abbey et al., 1995b, Table 2). The coefficient is calculated as follows:

$$b = \frac{\ln(1.81)}{45} = 0.0132 .$$

Standard Error (σ_β). The standard error for the coefficient (σ_β) is calculated from the reported lower and upper bounds of the relative risk (0.98 to 3.25) (Abbey et al., 1995b, Table 2):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(3.25)}{45} - \frac{\ln(1.81)}{45}\right)}{1.96} = 0.00664$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.81)}{45} - \frac{\ln(0.98)}{45}\right)}{1.96} = 0.00696$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.00680 .$$

F.3 HOSPITAL ADMISSIONS

There is a wealth of epidemiological information on the relationship between air pollution and hospital admissions for various respiratory and cardiovascular diseases; in addition, some studies have examined the relationship between air pollution and emergency room (ER) visits. Because most emergency room 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 do get admitted to the hospital, as discussed below.

Hospital admissions require the patient to be examined by a physician, and on average may represent more serious incidents than ER visits (Lipfert, 1993, p. 230). The two main groups of hospital admissions estimated in this analysis are respiratory admissions and cardiovascular admissions. There is not much evidence linking air pollution with other types of hospital admissions. The only types of ER visits that have been linked to air pollution in the U.S. or Canada are asthma-related visits.

F.3.1 Hospital Admissions for COPD (Samet et al., 2000, 14 Cities)

The C-R function to estimate the change in hospital admissions for COPD[36] associated with daily changes in PM_{10} is:

$$\Delta COPD\,Admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{10}} - 1)\right] \cdot pop,$$

where:

y_0	= daily hospital admission rate for COPD per person 65 and older = 3.12 E-5
β	= PM_{10} coefficient = 0.00288
ΔPM_{10}	= change in daily average PM_{10} concentration
pop	= population age 65 and older
σ_β	= standard error of β = 0.00139

Incidence Rate. COPD hospital admissions (ICD-9 codes: 490-492, 494-496) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (0.378 million) divided by the 1994 population of individuals 65 years and older (33.162 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (Graves and Gillum, 1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

[36] ICD-9 codes 490-492 and 494-496.

Coefficient Estimate (β). The estimated coefficient (β) is based on a 2.88 percent increase in admissions due to a PM_{10} change of 10.0 μg/m^3 (Samet et al., 2000, Part II - Table 14)[37]. This translates to a relative risk of 1.029. The coefficient is calculated as follows:

$$b = \frac{\ln(1.029)}{10.0} = 0.00288.$$

Standard Error (σ_β). The standard error (σ_β) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the percent increase (Samet et al., 2000, Part II - Table 14):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{0.0564}{10} - \frac{0.0288}{10}\right)}{1.96} = 0.00141$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{0.0288}{10} - \frac{0.0019}{10}\right)}{1.96} = 0.00137$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.00139.$$

F.3.2 Hospital Admissions for Pneumonia (Samet et al., 2000, 14 Cities)

The C-R function to estimate the change in hospital admissions for pneumonia[38] associated with daily changes in PM_{10} is:

$$\Delta pneumonia\ admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{10}} - 1)\right] \cdot pop,$$

where:

y_0	= daily hospital admission rate for pneumonia per person 65 and older = 5.30 E-5
β	= PM_{10} coefficient = 0.00207
ΔPM_{10}	= change in daily average PM_{10} concentration
pop	= population age 65 and older
σ_β	= standard error of β = 0.00058

[37] The random effects estimate of the unconstrained distributed lag model was chosen for COPD admissions since the chi-square test of heterogeneity was significant (see Samet et al., 2000, Part II - Table 15).

[38] ICD-9 codes 480-487.

Incidence Rate. Congestive heart failure hospital admissions (ICD-9 codes: 480-487) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (0.642 million) divided by the 1994 population of individuals 65 years and older (33.162 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (Graves and Gillum, 1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). The estimated coefficient (β) is based on a 2.07 percent increase in admissions due to a PM_{10} change of 10.0 μg/m^3 (Samet et al., 2000, Part II - Table 14)[39]. This translates to a relative risk of 1.021. The coefficient is calculated as follows:

$$b = \frac{\ln(1.021)}{10.0} = 0.00207 .$$

Standard Error (σ_β). The standard error (σ_β) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the percent increase (Samet et al., 2000, Part II - Table 14):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{0.0322}{10} - \frac{0.0207}{10}\right)}{1.96} = 0.00059$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{0.0207}{10} - \frac{0.0094}{10}\right)}{1.96} = 0.00058$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.00058 .$$

F.3.3 Hospital Admissions for Asthma (Sheppard et al., 1999, Seattle)

Sheppard et al. (1999) studied the relation between air pollution in Seattle and nonelderly hospital admissions for asthma from 1987 to 1994. They used air quality data for PM_{10}, $PM_{2.5}$, coarse $PM_{2.5\text{-}10}$, SO_2, ozone, and CO in a Poisson regression model with control for time trends, seasonal variations, and temperature-related weather effects. They found asthma hospital admissions associated with PM_{10}, $PM_{2.5}$, coarse $PM_{2.5\text{-}10}$, CO, and ozone. They did not observe an association for SO_2. They found PM and CO to be jointly associated with asthma admissions. The best fitting model was found using ozone. However, ozone data was only available April through October, so they did not consider ozone further. The C-R function in this analysis is based on a two-pollutant model with CO and $PM_{2.5}$.

[39] The random effects estimate of the unconstrained distributed lag model was chosen for pneumonia admissions since the chi-square test of heterogeneity was significant (see Samet et al., 2000, Part II - Table 15).

The C-R function to estimate the change in hospital admissions for asthma associated with daily changes in $PM_{2.5}$ is:

$$\Delta Asthma\ Admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{2.5}} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admission rate for asthma per person = 4.52 E-6
β = $PM_{2.5}$ coefficient = 0.00227
$\Delta PM_{2.5}$ = change in daily average $PM_{2.5}$ concentration
pop = population of ages less than 65
σ_β = standard error of β = 0.000948

Incidence Rate. Hospital admissions for asthma (ICD-9 code: 493) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (0.375 million) divided by the 1994 population (227.210 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). Based on a model with CO, the daily average coefficient (β) is estimated from the relative risk (1.03) associated with a change in $PM_{2.5}$ exposure over the interquartile range of 8 to 21 $\mu g/m^3$ (Sheppard et al., 1999, Table 3 and p. 28):

$$b = \frac{\ln(1.03)}{13} = 0.00227.$$

Standard Error (σ_β). The standard error (σ_β) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Sheppard et al., 1999, p. 28):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.06)}{13} - \frac{\ln(1.03)}{13}\right)}{1.96} = 0.00113$$

$$s_{b,low} = \frac{b - b_{low} -}{1.96} = \frac{\left(\frac{\ln(1.03)}{13} - \frac{\ln(1.01)}{13}\right)}{1.96} = 0.000770$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.000948.$$

F.4 EMERGENCY ROOM VISITS

There is a wealth of epidemiological information on the relationship between air pollution and hospital admissions for various respiratory and cardiovascular diseases; in addition, some studies have examined the relationship between air pollution and 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 do get admitted to the hospital, as discussed below.

The only types of ER visit that have been explicitly linked to ozone in U.S. and Canadian epidemiological studies are asthma visits. However, it seems likely that ozone may be linked to other types of respiratory-related ER visits.

F.4.1 Emergency Room Visits for Asthma (Schwartz et al., 1993, Seattle)

Schwartz et al. (1993) examined the relationship between air quality and emergency room visits for asthma in persons under 65 and 65 and over, living in Seattle from September 1989 to September 1990. Using single-pollutant models they found daily levels of PM_{10} linked to ER visits in individuals ages under 65, and they found no effect in individuals ages 65 and over. They did not find a significant effect for SO_2 and ozone in either age group. The results of the single pollutant model for PM_{10} are used in this analysis.

The C-R function to estimate the change in daily emergency room visits for asthma associated with daily changes in PM_{10} is:

$$\Delta \ Asthma\ ER visits = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{10}} - 1)\right] \cdot pop,$$

where:

y_0 = daily ER visits for asthma per person under 65 years old = 7.69 E-6
β = PM_{10} coefficient (Schwartz et al., 1993, p. 829) = 0.00367
ΔPM_{10} = change in daily average PM_{10} concentration
pop = population of ages 0-64
σ_β = standard error of β (Schwartz et al., 1993, p. 829) = 0.00126

Incidence Rate. Smith et al. (1997, p. 789) reported that in 1987 there were 445,000 asthma admissions and 1.2 million asthma ER visits. Assuming that all asthma hospital admissions pass through the ER room, then 37% of ER visits end up as hospital admissions. As described below, the 1994 asthma admission rate for people less than 65 is 4.522 E-6. So one might assume, ER visits = (1/0.37)*asthma admission rate = 2.7*asthma admission rate = 1.22 E-5. Now, ER visits (subtracting out those visits that end up as admissions)= 1.7*asthma admission rate = 7.69 E-6.

Asthma hospital admissions (ICD-9 code: 493) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (0.375 million) divided by the 1994 population of individuals under 65 years old (227.21 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (Graves and Gillum, 1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

F.5 ACUTE MORBIDITY

In addition to chronic illnesses and hospital admissions, there is a considerable body of scientific research that has estimated significant relationships between elevated air pollution levels and other morbidity health effects. Chamber study research has established relationships between specific air pollution chemicals and symptoms such as coughing, pain on deep inspiration, wheezing, eye irritation and headaches. In addition, epidemiological research has found air pollution relationships with acute infectious diseases (e.g., bronchitis, sinusitis) and a variety of "symptom-day" categories. Some "symptom-day" studies examine excess incidences of days with identified symptoms such as wheezing, coughing, or other specific upper or lower respiratory symptoms. Other studies estimate relationships for days with a more general description of days with adverse health impacts, such as "respiratory restricted activity days" or work loss days.

A challenge in preparing an analysis of the minor morbidity effects is identifying a set of effect estimates that reflects the full range of identified adverse health effects but avoids double counting. From the definitions of the specific health effects examined in each research project, it is possible to identify a set of effects that are non-overlapping, and can be ultimately treated as additive in a benefits analysis.

F.5.1 Acute Bronchitis C-R Function (Dockery et al., 1996)

Dockery et al. (1996) examined the relationship between PM and other pollutants on the reported rates of asthma, persistent wheeze, chronic cough, and bronchitis, in a study of 13,369 children ages 8-12 living in 24 communities in U.S. and Canada. Health data were collected in 1988-1991, and single-pollutant models were used in the analysis to test a number of measures of particulate air pollution. Dockery et al. found that annual level of sulfates and particle acidity were significantly related to bronchitis, and $PM_{2.1}$ and PM_{10} were marginally significantly related to bronchitis.[40] They also found nitrates were linked to asthma, and sulfates linked to chronic phlegm. It is important to note that the study examined annual pollution exposures, and the authors did not rule out that acute (daily) exposures could be related to asthma attacks and other acute episodes.

Earlier work, by Dockery et al. (1989), based on six U.S. cities, found acute bronchitis and chronic cough significantly related to PM_{15}. Because it is based on a larger sample, the Dockery et al. (1996) study is the better study to develop a C-R function linking $PM_{2.5}$ with bronchitis. The C-R function to estimate the change in acute bronchitis is:

$$\Delta Acute\ Bronchitis = -\left[\frac{y_0}{(1-y_0)\cdot e^{\Delta PM_{2.5}\cdot b} + y_0} - y_0\right]\cdot pop\ ,$$

where:

y_0 = annual bronchitis incidence rate per person = 0.044
β = estimated $PM_{2.5}$ logistic regression coefficient = 0.0272
$\Delta PM_{2.5}$ = change in annual average $PM_{2.5}$ concentration
pop = population of ages 8-12
σ_β = standard error of β = 0.0171

[40] The original study measured $PM_{2.1}$, however when using the study's results we use $PM_{2.5}$. This makes only a negligible difference, assuming that the adverse effects of $PM_{2.1}$ and $PM_{2.5}$ are comparable.

Incidence Rate. Bronchitis was counted in the study only if there were "reports of symptoms in the past 12 months" (Dockery et al., 1996, p. 501). It is unclear, however, if the cases of bronchitis are acute and temporary, or if the bronchitis is a chronic condition. Dockery et al. found no relationship between PM and chronic cough and chronic phlegm, which are important indicators of chronic bronchitis. For this analysis, we assumed that the C-R function based on Dockery et al. is measuring acute bronchitis.

In 1994, 2,115,000 children ages 5-17 experienced acute conditions (Adams and Marano, 1995, Table 6) out of population of 48.110 million children ages 5-17 (U.S. Bureau of the Census, 1998, Table 14), or 4.4 percent of this population. This figure is somewhat lower than the 5.34 percent of children under the age of 18 reported to have chronic bronchitis in 1990-1992 (Collins, 1997, Table 8). Dockery et al. (1996, p. 503) reported that in the 24 study cities the bronchitis rate varied from three to ten percent. Finally a weighted average of the incidence rates in the six cities in the Dockery et al. (1989) study is 6.34 percent , where the sample size from each city is used to weight the respective incidence rate (Dockery et al., 1989, Tables 1 and 4).[41] This analysis assumes a 4.4 percent prevalence rate is the most representative of the national population. Note that this measure reflects the fraction of children that have a chest ailment diagnosed as bronchitis in the past year, not the number of days that children are adversely affected by acute bronchitis.[42]

Coefficient Estimate (β). The estimated logistic coefficient (β) is based on the odds ratio (= 1.50) associated with being in the most polluted city ($PM_{2.1}$ = 20.7 $\mu g/m^3$) versus the least polluted city ($PM_{2.1}$ = 5.8 $\mu g/m^3$) (Dockery et al., 1996, Tables 1 and 4). The original study used $PM_{2.1}$, however, we use the $PM_{2.1}$ coefficient and apply it to $PM_{2.5}$ data.

$$b_{PM_{2.5}} = \frac{\ln(1.50)}{(20.7 - 5.8)} = 0.0272 .$$

Standard Error (σ_β). The standard error of the coefficient (σ_β) is calculated from the reported lower and upper bounds of the odds ratio (Dockery et al., 1996, Table 4):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(2.47)}{14.9} - \frac{\ln(1.50)}{14.9}\right)}{1.96} = 0.0171$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.50)}{14.9} - \frac{\ln(0.91)}{14.9}\right)}{1.96} = 0.0171$$

$$s_b = \frac{s_{b,high} + s_{b,low}}{2} = 0.0171 .$$

[41]The unweighted average of the six city rates is 0.0647.

[42]In 1994, there were 13,707,000 restricted activity days associated with acute bronchitis, and 2,115,000 children (ages 5-17) experienced acute conditions (Adams and Marano, 1995, Tables 6 and 21). On average, then, each child with acute bronchitis suffered 6.48 days.

F.5.2 Lower Respiratory Symptoms (Schwartz et al., 1994)

Schwartz et al. (1994) used logistic regression to link lower respiratory symptoms in children with SO_2, NO_2, ozone, PM_{10}, $PM_{2.5}$, sulfate and H^+ (hydrogen ion). Children were selected for the study if they were exposed to indoor sources of air pollution: gas stoves and parental smoking. The study enrolled 1,844 children into a year-long study that was conducted in different years (1984 to 1988) in six cities. The students were in grades two through five at the time of enrollment in 1984. By the completion of the final study, the cohort would then be in the eighth grade (ages 13-14); this suggests an age range of 7 to 14.

In single pollutant models SO_2, NO_2, $PM_{2.5}$, and PM_{10} were significantly linked to cough. In two-pollutant models, PM_{10} had the most consistent relationship with cough; ozone was marginally significant, controlling for PM_{10}. In models for upper respiratory symptoms, they reported a marginally significant association for PM_{10}. In models for lower respiratory symptoms, they reported significant single-pollutant models, using SO_2, O_3, $PM_{2.5}$, PM_{10}, SO_4, and H^+.

The C-R function used to estimate the change in lower respiratory symptoms is:

$$\Delta Lower\ Respiratory\ Symptoms = -\left[\frac{y_0}{(1-y_0)\cdot e^{\Delta PM_{2.5}\cdot b} + y_0} - y_0\right]\cdot pop\,.$$

where:

y_0 = daily lower respiratory symptom incidence rate per person = 0.0012
β = estimated $PM_{2.5}$ logistic regression coefficient = 0.01823
$\Delta PM_{2.5}$ = change in daily average $PM_{2.5}$ concentration
pop = population of ages 7-14
σ_β = standard error of β = 0.00586

Incidence Rate. The proposed incidence rate, 0.12 percent, is based on the percentiles in Schwartz et al. (Schwartz et al., 1994, Table 2). They did not report the mean incidence rate, but rather reported various percentiles from the incidence rate distribution. The percentiles and associated values are 10^{th} = 0 percent, 25^{th} = 0 percent, 50^{th} = 0 percent, 75^{th} = 0.29 percent, and 90^{th} = 0.34 percent. The most conservative estimate consistent with the data are to assume the incidence is zero up to the 75^{th} percentile, a constant 0.29 percent between the 75^{th} and 90^{th} percentiles, and a constant 0.34 percent between the 90^{th} and 100^{th} percentiles. Alternatively, assuming a linear slope between the 50^{th} and 75^{th}, 75^{th} and 90^{th}, and 90^{th} to 100^{th} percentiles, the estimated mean incidence rate is 0.12 percent,[43] which is used in this analysis.

[43]For example, the 62.5^{th} percentile would have an estimated incidence rate of 0.145 percent.

Coefficient Estimate (β). The coefficient β is calculated from the reported odds ratio (= 1.44) in a single-pollutant model associated with a 20 $\mu g/m^3$ change in $PM_{2.5}$ (Schwartz et al., 1994, Table 5):

$$b = \frac{\ln(1.44)}{20} = 0.01823.$$

Standard Error (σ_β). The standard error for the coefficient (σ_β) is calculated from the reported lower and upper bounds of the odds ratio (Schwartz et al., 1994, Table 5):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.82)}{20} - \frac{\ln(1.44)}{20}\right)}{1.96} = 0.00597$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.44)}{20} - \frac{\ln(1.15)}{20}\right)}{1.96} = 0.00574$$

$$s_b = \frac{s_{b,high} + s_{b,low}}{2} = 0.00586.$$

Population. Schwartz et al. (1994, Table 5 and p. 1235) enrolled 1,844 children into a year-long study that was conducted in different years in different cities; the students were in grades two through five and lived in six U.S. cities. All study participants were enrolled in September 1984; the actual study was conducted in Watertown, MA in 1984/85; Kingston-Harriman, TN, and St. Louis, MO in 1985/86; Steubenville, OH, and Portage, WI in 1986/87; and Topeka, KS in 1987/88. The study does not publish the age range of the children when they participated. As a result, the study is somewhat unclear about the appropriate age range for the resulting C-R function. If all the children were in second grade in 1984 (ages 7-8) then the Topeka cohort would be in fifth grade (ages 10-11) when they participated in the study. It appears from the published description, however, that the students were in grades two through five in 1984.[44] By the completion of the study, some students in the Topeka cohort would then be in the eighth grade (ages 13-14); this suggests an age range of 7 to 14.

[44]Neas et al. (1994, p. 1091) used the same data set; their description suggests that grades two to five were represented initially.

F.5.3 Upper Respiratory Symptoms (Pope et al., 1991)

Using logistic regression, Pope et al. (1991) estimated the impact of PM_{10} on the incidence of a variety of minor symptoms in 55 subjects (34 "school-based" and 21 "patient-based") living in the Utah Valley from December 1989 through March 1990. The children in the Pope et al. study were asked to record respiratory symptoms in a daily diary. With this information, the daily occurrences of upper respiratory symptoms (URS) and lower respiratory symptoms (LRS) were related to daily PM_{10} concentrations. Pope et al. describe URS as consisting of one or more of the following symptoms: runny or stuffy nose; wet cough; and burning, aching, or red eyes. Levels of ozone, NO_2, and SO_2 were reported low during this period, and were not included in the analysis. The sample in this study is relatively small and is most representative of the asthmatic population, rather than the general population. The school-based subjects (ranging in age from 9 to 11) were chosen based on "a positive response to one or more of three questions: ever wheezed without a cold, wheezed for 3 days or more out of the week for a month or longer, and/or had a doctor say the 'child has asthma' (Pope et al., 1991, p. 669)." The patient-based subjects (ranging in age from 8 to 72) were receiving treatment for asthma and were referred by local physicians. Regression results for the school-based sample (Pope et al., 1991, Table 5) show PM_{10} significantly associated with both upper and lower respiratory symptoms. The patient-based sample did not find a significant PM_{10} effect. The results from the school-based sample are used here.

The C-R function used to estimate the change in upper respiratory symptoms is:

$$\Delta Upper\ \mathrm{Re}spiratory\ Symptoms = -\left[\frac{y_0}{(1-y_0)\cdot e^{\Delta PM_{10}\cdot b} + y_0} - y_0\right]\cdot pop\,,$$

where:

y_0 = daily upper respiratory symptom incidence rate per person = 0.3419
β = estimated PM_{10} logistic regression coefficient (Pope et al., 1991, Table 5) = 0.0036
ΔPM_{10} = change in daily average PM_{10} concentration
pop = asthmatic population[45] ages 9 to 11 = 6.91% of population ages 9 to 11
σ_β = standard error of β (Pope et al., 1991, Table 5) = 0.0015

Incidence Rate. The incidence rate is published in Pope et al. (Pope et al., 1991, Table 2). Taking a sample-size-weighted average, one gets an incidence rate of 0.3419.

F.5.4 Minor Restricted Activity Days (Ostro and Rothschild, 1989)

Ostro and Rothschild (1989) estimated the impact of $PM_{2.5}$ on the incidence of minor restricted activity days (MRADs) and respiratory-related restricted activity days (RRADs) in a national sample of the adult working population, ages 18 to 65, living in metropolitan areas. The annual national survey results used in this analysis were conducted in 1976-1981. Controlling for $PM_{2.5}$, two-week average O_3 has highly variable association with RRADs and MRADs. Controlling for O_3, two-week average $PM_{2.5}$ was significantly linked to both health endpoints in most years.

[45] Adams (1995, Table 57) reported that in 1994, 6.91% of individuals under the age of 18 have asthma.

The study is based on a "convenience" sample of individuals ages 18-65. Applying the C-R function to this age group is likely a slight underestimate, as it seems likely that elderly are at least as susceptible to PM as individuals 65 and younger. The elderly appear more likely to die due to PM exposure than other age groups (e.g., Schwartz, 1994c, p. 30) and a number of studies have found that hospital admissions for the elderly are related to PM exposures (e.g., Schwartz, 1994a; Schwartz, 1994b).

Using the results of the two-pollutant model, we developed separate coefficients for each year in the analysis, which were then combined for use in this analysis. The coefficient used in this analysis is a weighted average of the coefficients (Ostro, 1987, Table IV) using the inverse of the variance as the weight. The C-R function to estimate the change in the number of minor restricted activity days (MRAD) is:

$$\Delta MRAD = \Delta y \cdot pop = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{2.5}} - 1) \right] \cdot pop,$$

where:

y_0 = daily MRAD daily incidence rate per person = 0.02137
β = inverse-variance weighted $PM_{2.5}$ coeffcient = 0.00741
$\Delta PM_{2.5}$ = change in daily average $PM_{2.5}$ concentration[46]
pop = adult population ages 18 to 65
σ_β = standard error of β = 0.0007

Incidence Rate. The annual incidence rate (7.8) provided by Ostro and Rothschild (1989, p. 243) was divided by 365 to get a daily rate of 0.02137.

Coefficient Estimate (β). The coefficient is a weighted average of the coefficients in Ostro and Rothschild (1989, Table 4) using the inverse of the variance as the weight:

$$b = \left(\frac{\sum_{i=1976}^{1981} \frac{b_i}{s_{b_i}^2}}{\sum_{i=1976}^{1981} \frac{1}{s_{b_i}^2}} \right) = 0.00741.$$

[46]The study used a two-week average pollution concentration; the daily rate used here is assumed to be a reasonable approximation.

Standard Error (σ_β). The standard error of the coefficient (σ_β) is calculated as follows, assuming that the estimated year-specific coefficients are independent:

$$s_b^2 = \text{var}\left(\frac{\sum_{i=1976}^{1981} \frac{b_i}{s_{b_i}^2}}{\sum_{i=1976}^{1981} \frac{1}{s_{b_i}^2}}\right) = \left(\frac{\sum_{i=1976}^{1981} \frac{b_i}{s_{b_i}^2}}{g}\right) = \sum_{i=1976}^{1981} \text{var}\left(\frac{b_i}{s_{b_i}^2 \cdot g}\right).$$

This reduces down to:

$$s_b^2 = \frac{1}{g} \Rightarrow s_b = \sqrt{\frac{1}{g}} = 0.00070.$$

F.5.5 Work Loss Days (Ostro, 1987)

Ostro (1987) estimated the impact of $PM_{2.5}$ on the incidence of work-loss days (WLDs), restricted activity days (RADs), and respiratory-related RADs (RRADs) in a national sample of the adult working population, ages 18 to 65, living in metropolitan areas. The annual national survey results used in this analysis were conducted in 1976-1981. Ostro reported that two-week average $PM_{2.5}$ levels were significantly linked to work-loss days, RADs, and RRADs, however there was some year-to-year variability in the results. Separate coefficients were developed for each year in the analysis (1976-1981); these coefficients were pooled. The coefficient used in the concentration-response function used here is a weighted average of the coefficients in Ostro (1987, Table III) using the inverse of the variance as the weight.

The study is based on a "convenience" sample of individuals ages 18-65. Applying the C-R function to this age group is likely a slight underestimate, as it seems likely that elderly are at least as susceptible to PM as individuals 65 and younger. The elderly appear more likely to die due to PM exposure than other age groups (e.g., Schwartz, 1994c, p. 30) and a number of studies have found that hospital admissions for the elderly are related to PM exposures (e.g., Schwartz, 1994a; Schwartz, 1994b). On the other hand, the number of workers over the age of 65 is relatively small; it was under 3% of the total workforce in 1996 (U.S. Bureau of the Census, 1997, Table 633).

The C-R function to estimate the change in the number of work-loss days is:

$$\Delta WLD = \Delta y \cdot pop = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{2.5}} - 1)\right] \cdot pop,$$

where:

y_0 = daily work-loss-day incidence rate per person = 0.00648
β = inverse-variance weighted $PM_{2.5}$ coefficient = 0.0046
$\Delta PM_{2.5}$ = change in daily average $PM_{2.5}$ concentration[47]
pop = population of ages 18 to 65
σ_β = standard error of β = 0.00036

[47]The study used a two-week average pollution concentration; the daily rate used here is assumed to be a reasonable approximation.

Incidence Rate. The estimated 1994 annual incidence rate is the annual number (376,844,000) of WLD per person in the age 18-64 population divided by the number of people in 18-64 population (159,361,000). The 1994 daily incidence rate is calculated as the annual rate divided by 365.[48] Data are from U.S. Bureau of the Census (1997, Table 14) and Adams (1995, Table 41).

Coefficient Estimate (β). The coefficient used in the C-R function is a weighted average of the coefficients in Ostro (1987, Table III) using the inverse of the variance as the weight:

$$b = \left(\frac{\sum_{i=1976}^{1981} \frac{b_i}{s_{b_i}^2}}{\sum_{i=1976}^{1981} \frac{1}{s_{b_i}^2}} \right) = 0.0046 .$$

Standard Error (σ_β). The standard error of the coefficient (σ_β) is calculated as follows, assuming that the estimated year-specific coefficients are independent:

$$s_b^2 = \text{var}\left(\frac{\sum_{i=1976}^{1981} \frac{b_i}{s_{b_i}^2}}{\sum_{i=1976}^{1981} \frac{1}{s_{b_i}^2}} \right) = \left(\frac{\sum_{i=1976}^{1981} \frac{b_i}{s_{b_i}^2}}{g} \right) = \sum_{i=1976}^{1981} \text{var}\left(\frac{b_i}{s_{b_i}^2 \cdot g} \right).$$

This eventually reduces down to:

$$s_b^2 = \frac{1}{g} \Rightarrow s_b = \sqrt{\frac{1}{g}} = 0.00036 .$$

[48]Ostro (1987) analyzed a sample aged 18 to 65. It is assumed that the age 18-64 rate is a reasonably good approximation to the rate for individuals 18-65. Data are from U.S. Bureau of the Census (1997, Table 14) and Adams (1995, Table 41).

F.5.6 Asthma Attacks: Whittemore and Korn (1980)

Whittemore and Korn (1980) examined the relationship between air pollution and asthma attacks in a survey of 443 children and adults, living in six communities in southern California during three 34-week periods in 1972-1975. The analysis focused on TSP and ozone. Respirable PM, NO_2, SO_2 were highly correlated with TSP and excluded from the analysis. In a two pollutant model, daily levels of both TSP and O_x were significantly related to reported asthma attacks.

The C-R function to estimate the change in the number of asthma attacks is:

$$\Delta asthma\ attacks = -\left[\frac{y_0}{(1-y_0)\cdot e^{\Delta PM_{10}\cdot b} + y_0} - y_0\right]\cdot pop,$$

where:

y_0 = daily incidence of asthma attacks = 0.027 (Krupnick, 1988, p. 4-6)
β = PM_{10} coefficient = 0.00144
ΔPM_{10} = change in daily PM_{10} concentration
pop = population of asthmatics of all ages = 5.61% of the population of all ages (Adams and Marano, 1995 Table 57).
σ_β = standard error of β = 0.000556

Incidence Rate. The annual rate of 9.9 asthma attacks per astmatic is divided by 365 to get a daily rate. A figure of 9.9 is roughly consistent with the recent statement that "People with asthma have more than 100 million days of restricted activity" each year (National Heart, 1997, p. 1). This 100 million incidence figure coupled with the 1996 population of 265,557,000 (U.S. Bureau of the Census, 1997, Table 2) and the latest asthmatic prevalence rate of 5.61% (Adams and Marano, 1995, Table 57), suggest an annual asthma attach rate per asthmatic of 6.7.

Coefficient Estimate (β). Based on a model with ozone, the coefficient is based on a TSP coefficient (0.00079) (Whittemore and Korn, 1980, Table 5). Assuming that PM_{10} is 55 percent of TSP[49] and that particulates greater than ten micrometers are harmless, the coefficient is calculated as follows:

$$b = \frac{0.00079}{0.55} = 0.00144.$$

Standard Error (σ_β). The standard error (σ_β) is calculated from the two-tailed p-value (<0.01) reported by Whittemore and Korn (1980, Table 5), which implies a t-value of at least 2.576 (assuming a large number of degrees of freedom).

$$s_b = \frac{b}{t} = \frac{0.144}{2.576} = 0.000556.$$

[49]The conversion of TSP to PM_{10} is from ESEERCO (1994, p. V-5), who cited studies by EPA (1986) and the California Air Resources Board (1982).

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Client Alert Commentary

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The Supreme Court Upholds EPA's Cross-State Air Pollution Rule

Decision in* EME Homer City *upholds CSAPR, but additional legal challenges and EPA revisions may still significantly alter CSAPR and delay its implementation.

On Tuesday, April 29, the United States Supreme Court upheld EPA's Cross-State Air Pollution Rule (**CSAPR**) in a 6-2 decision.[1] The Court's ruling in *EPA v. EME Homer City Generation* follows a long and highly contentious regulatory process that included: EPA's development of a prior rule regulating interstate transport of air pollution, the Clean Air Interstate Rule (**CAIR**) in 2005; the U.S. Court of Appeals for the D.C. Circuit's (**D.C. Circuit**) rejection of CAIR in 2008; EPA's development of CSAPR as a replacement rule in 2011; and the D.C. Circuit's decision to vacate CSAPR in August 2012. The Supreme Court's decision on Tuesday overturns the D.C. Circuit's 2012 ruling and reinstates CSAPR. EPA has announced that CAIR will continue to remain in place pending EPA's review of the Court's recent holding.[2]

Overview of the Decision

The majority opinion in *EME Homer City*, authored by Justice Ruth Bader Ginsberg,[3] includes two key holdings: (1) EPA was not required to provide the States another opportunity to develop state implementation plans (**SIPs**) allocating in-state emissions of air pollutants after EPA set emissions budgets for the States in CSAPR and could proceed to issue a federal implementation plan (**FIP**) without States' input; and (2) EPA's decision to allocate emission reductions in upwind States on the basis of control costs rather than proportional contributions of pollution to downwind States was a reasonable interpretation of the Clean Air Act's Good Neighbor Provision and EPA's decision is therefore entitled to *Chevron* deference.[4]

Justice Antonin Scalia issued a vocal dissent,[5] arguing EPA was required to provide the States a meaningful opportunity to allocate air emission reductions within their own borders before issuing a FIP, and arguing the Good Neighbor Provision provided no basis for EPA's consideration of control costs, rather than proportional allocation.

Background

Cross-Border Emissions Regulation under Section 110 of the Clean Air Act

The Cross-State Air Pollution Rule regulates cross-border emissions of criteria air pollutants including sulfur dioxide (**SO_2**) and nitrogen oxides (**NO_X**), as well as their byproducts, fine particulates (**$PM_{2.5}$**) and ozone, under Section 110 of the Clean Air Act.[6] Section 110 of the Clean Air Act requires States to create SIPs[7] to limit emissions from sources that "contribute significantly" to noncompliance with primary and secondary National Ambient Air Quality Standards (**NAAQS**) for the criteria air pollutants.[8] If the ambient levels of criteria air pollutants are above the NAAQS threshold set by EPA, a region is considered to be in

"nonattainment" for that pollutant and EPA applies more stringent control standards for sources of air emissions located in the region. The Good Neighbor Provision, found in Section 110(a)(2)(D)(i) of the Clean Air Act, requires SIPs to contain adequate provisions to prohibit "any source or other types of emissions activity within the State from emitting any air pollutant in amounts which will ... contribute significantly to nonattainment in, or interfere with maintenance by, any other State with respect to any" NAAQS. 42 U.S.C. § 7410(a)(2)(D)(i).

CAIR

According to EPA, as the result of SO_2 and NO_x moving across state borders and breaking down into fine particulates and ozone, some downwind States have been in nonattainment for air pollutants despite their own measures to control these pollutants. EPA claimed that these cross-border impacts were caused, in part, by upwind States that were not implementing adequate measures to reduce emissions. In 2005, EPA addressed interstate transport of air pollution with CAIR, which regulated NOx and SO_2 emissions, and created a market for trading emission credits. EPA permitted the States to develop SIPs in response to proposed allocations under CAIR. In 2008, the D.C. Circuit rejected CAIR, in part, on the basis of the Court's findings that EPA had failed to establish links between air pollution in upwind States and nonattainment with NAAQS in downwind States.[9] The Court left CAIR temporarily in place, but instructed EPA to "redo its analysis from the ground up."[10]

CSAPR

EPA issued CSAPR as a replacement rule in 2011. CSAPR regulates interstate transport of SO_2 and NO_x and $PM_{2.5}$ in 28 Eastern states. EPA used a two-step approach to allocate interstate emission reductions under CSAPR. First, EPA used a screening analysis to eliminate any upwind State that contributed less than one percent of the NAAQS for SO_2 and NO_x and $PM_{2.5}$ to downwind receptor States. If the upwind State contributed less than one percent of the NAAQS to all downwind States, EPA determined that the State had not "contributed significantly" to interstate pollution and was not subject to the Good Neighbor Provision. Second, EPA generated a "cost-effective" allocation of emissions reductions based on the cost per ton of reducing emissions. Finally, after compiling these data points, EPA created a budget or allocation of SO_2 and NO_X emissions for each source in the upwind States and issued a FIP. EPA did not permit the States an opportunity, as it had done under CAIR, to issue SIPs in response to EPA's emission allocations.

EPA's allocations depend wholly on emission reductions from electrical generating units (**EGUs**).[11] No other emitting sources were targeted. In the final version of CSAPR, EPA allocated emissions at sources on the basis of heat input, rather than just the cost-effectiveness of reductions, allegedly resulting in some sources receiving more emission allowances than they needed, and others falling far short of the allocations needed to continue operating.

CSAPR also establishes a market for trading SO_2 and NO_x emission allowances. Facilities are permitted to trade emission allowances with other sources in the same state. Interstate trading is also permitted, but is subject to significant limitations, including a cap on the amounts of emission credits that can be traded with out-of-state sources.[12] The first phase of the emissions trading program began in January 2012, with the second phase slated to start in January 2014.

Vacatur of CSAPR

In August 2012, in a split 2-1 decision, the D.C. Circuit vacated CSAPR on two independent grounds:[13] (1) EPA could only require States to reduce their significant contributions to downwind States and could not consider costs in allocating emission reductions under CSAPR; and (2) EPA was required to provide States a reasonable opportunity to review EPA's proposed allocation of emissions and develop SIPs prior

to EPA issuing a FIP implementing CSAPR. The prices of NO_x and SO_2 emission allowances dropped precipitously following the D.C. Circuit's decision.[14] Following CSAPR's vacatur, EPA reinstated the CAIR program on a temporary basis. EPA and the American Lung Association appealed the D.C. Circuit's decision and the Supreme Court granted certiorari on June 24, 2013.

The Supreme Court's Decision

EPA Did Not Have to Provide States Another Opportunity to Promulgate SIPs

In the first of its two key holdings, the Supreme Court reversed the D.C. Circuit's decision that EPA was required to provide the States an opportunity to develop SIPs and allocate emission reductions within their own borders after EPA developed state emissions budgets for air pollutants under CSAPR. The Supreme Court held that EPA was not required under the plain language of Section 110 of the Clean Air Act, to give the States an opportunity to review EPA's emissions budgets for the States and issue new SIPs prior to EPA issuing a FIP.

The Supreme Court held that Section 110 included no requirement that EPA provide the States another opportunity to develop a SIP after EPA rejected the initial SIP. Section 110 allows EPA to issue a FIP "at any time" within two years after it rejects the SIP. Nothing in Section 110 of the Act "places EPA under an obligation to provide specific metrics to States before they undertake to fulfill their good neighbor obligations." *Id*. at 17. Justice Scalia's dissent, echoing the D.C. Circuit's opinion, criticized EPA's approach as inconsistent with the principles of cooperative federalism and EPA's past practice with the NO_x SIP call[15] and CAIR.[16]

The majority acknowledged that EPA did provide the States an opportunity to review their emissions budgets prior to finalizing their SIPs under the NO_x SIP call and CAIR, but EPA was under no similar obligation to do so again when it issued CSAPR: "Whatever pattern the Agency followed in its NO_x SIP call and CAIR proceedings, EPA retained discretion to alter its course provided it gave a reasonable explanation for doing so." *Id.* at 17. In this case, EPA decided to act "expeditiously" because CAIR had been invalidated by the D.C. Circuit and EPA decided it was "inappropriate" for the Agency to undertake the "lengthy transition period" required for States to propose new or amended SIPs addressing EPA's CSAPR emissions budgets.

EPA's Decision to Allocate Emissions Using Cost is Entitled to *Chevron* Deference

The Supreme Court then turned to EPA's decision to allocate emissions among the States, not based on proportional contributions to air pollution, but based on the relative costs of reducing air pollution. Citing the *Chevron* case,[17] the Court held that because Congress had been silent on allocating emission reductions under the Good Neighbor Provision and EPA's cost allocation approach to apportionment was reasonable, EPA's approach should be afforded administrative deference and upheld.

The majority described EPA's task under Section 110 of the Act as a difficult one: "How should EPA allocate among multiple contributing upwind States responsibility for a downwind State's excess pollution?" *Id*. at 21. While the Good Neighbor Provision prohibits only those emissions from upwind States that "contribute significantly" to downwind attainment with the NAAQS, EPA had to determine what quantity of emissions should be reduced from each contributing State. EPA's solution to this problem was first to screen out any State that contributed less than one percent of the relevant NAAQS in a nonattainment, downwind State. For the States not screened out, EPA then determined which "amounts" of air pollution could be cost-effectively reduced. The Supreme Court held that EPA's methodology was an "efficient and equitable solution" to the excess emissions problem because it reduced the overall costs

of compliance with CSAPR and "subjects to stricter regulation those States that have done relatively less in the past to control their pollution." *Id*. at 27.

The Court rejected the respondents' arguments, the D.C. Circuit, and the dissenting Justices, that EPA could not consider costs in determining which emissions should be reduced because costs were not mentioned in the Good Neighbor Provision. The Court also rejected the argument that the Good Neighbor Provision required EPA to allocate responsibility for reducing emissions in "a manner proportional to each State's contribution to the problem." *Id.* at 22. In the Court's words, "Nothing in the text of the Good Neighbor Provision propels EPA down this path." *Id.* at 23. Indeed, according to the Court, the D.C. Circuit's "proportionality edict" does not work "mathematically" or "in practical application." *Id.* at 23. Finally, the Court rejected respondents' concerns that EPA's cost-allocation approach could lead to over-control of emissions in upwind States.[18] However, the Court left open the possibility that States falling below EPA's one percent threshold for control might bring individual challenges to the application of CSAPR.

Dissent

Justice Scalia's dissent, which he read in part from the bench, rebuked the majority opinion. The dissent began with a sharp jab at EPA and the Obama Administration: "Too many important decisions of the Federal Government are made nowadays by unelected agency officials exercising broad lawmaking authority, rather than by the people's representatives in Congress." Slip Op., Dissent at 1. Contrary to the majority's holding, Justice Scalia argued that there was no gap in the Good Neighbor Provision that permitted EPA to consider costs and that the majority had essentially approved EPA's "undemocratic revision of the Clean Air Act." *Id.* at 2. Justice Scalia expressed concern that the majority did not address EPA's primary argument that the phrase "significantly" in the statute was ambiguous and could be interpreted to allow EPA to consider costs. Instead, the majority relied on an "imaginary gap" in the Good Neighbor Provision to sanction EPA's cost allocation approach. *Id.* at 7.

The dissent also agreed with the D.C. Circuit that Section 110 of the Clean Air Act could only be interpreted to require the proportional reduction of air pollution from upwind States. There was no ambiguity or gap in the statute that allowed cost considerations. Congress could have, as it has done with many other environmental statutes, required EPA to consider costs, but Congress chose not to include such language. Justice Scalia disagreed that a proportional-reduction approach was impossible to apply. If it was impossible to apply, EPA must let the law fail: "I know of no legal authority that and no democratic principle that would derive from it the consequence that EPA could rewrite the statute, rather than the consequence that the statute would be inoperative." *Id.* at 7. Finally, as noted above, Justice Scalia criticized EPA's decision to abandon the principle of "cooperative federalism" and issue a FIP without providing the States "a meaningful opportunity to allocate reduction responsibilities among the sources within their borders." *Id.* at 15.

Implications of Decision

While the Supreme Court's decision upholding CSAPR represents a significant victory for EPA, the implications of the Court's ruling for regulating interstate transport of air pollution remain somewhat unclear.[19] The *EME Homer City* case will be remanded to the D.C. Circuit. The D.C. Circuit may request supplemental briefing and review additional challenges to CSAPR that were not considered in its initial decision setting aside the rule (and therefore not reviewed by the Supreme Court). If CSAPR remains in place, clarifications will be required — possibly in the form of new rulemakings — in light of the deadlines in the rule for implementing the rule's NO_x and SO_2 trading programs that passed while the rule was under review by the D.C. Circuit and Supreme Court. EPA may also significantly revise its emissions allocations in light of major changes in emissions following recent large-scale conversions of coal-fired

EGUs to natural gas, improvements in available emission control technologies, and EPA's pending release of a new NAAQS for ozone — although EPA may not be inclined to do so.

The Supreme Court's decision allowing EPA to dismiss SIPs and issue a FIP on the basis of requirements in CSAPR not yet announced and not subject to public notice or comment, may represent a dramatic shift away from the principles of cooperative federalism engrained in the Clean Air Act.[20] While the Court's decision may be interpreted as a limited exception[21] to the normal SIP process in which States have an opportunity to work with EPA to implement Clean Air Act standards, the decision may also be interpreted as recognizing EPA's virtually unfettered authority to deny a SIP and impose air regulations on its own. Several challenges to EPA's SIP disapprovals under CSAPR are still pending following their stay during the Supreme Court's review of the *EME Homer City* case. These cases will now be heard[22] and could limit EPA's authority to regulate under CSAPR without the input of the States. The outcome of these cases and others will almost certainly have a significant impact on CSAPR and its implementation.

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Endnotes

[1] *EPA v. EME Homer City Generation*, No. 12-1182, Slip op. (April 29, 2014).

[2] U.S. Environmental Protection Agency, Interstate Air Pollution Transport, *available at* http://www.epa.gov/airtransport/ ("At this time, CAIR remains in place and no immediate action from States or affected sources is expected.") (last visited May 2, 2014).

[3] Chief Justice John Roberts and Justices Anthony Kennedy, Stephen Breyer, Sonia Sotomayor, and Elena Kagan joined the majority opinion.

[4] The Court also rejected a jurisdictional challenge EPA brought relating to respondents' comments on CSAPR. *EME Homer City*, at 18-19.

[5] Justice Clarence Thomas joined the dissent. Justice Samuel Alito recused himself from the case.

[6] Six additional States – Iowa, Kansas, Michigan, Missouri, Oklahoma and Wisconsin — are required to make additional reductions in NO_X emissions during the summertime (ozone season) under CSAPR.

[7] Under Section 110 of the Act, if a State does not implement an acceptable control plan to comply with the NAAQS within three years, EPA must promulgate a FIP for the State within the next two years.

[8] Criteria air pollutants include lead, particulate matter (PM), ozone, sulfur dioxide, nitrogen oxides, and carbon monoxide.

[9] *North Carolina v. EPA*, 531 F.3d 896 (D.C. Cir. 2008).

[10] *Id.* at 929.

[11] Facilities may reduce emissions by burning low sulfur coal, increasing generation at more efficient units, or by installing control technologies including low NO_X burners, scrubbers (flue gas desulfurization), or dry sorbent injection systems.

[12] Limitations on interstate trading in CSAPR resulted in a highly inefficient emissions trading market in which sources left short on emission credits were required to buy allowances from competitors with few incentives to sell them, or curtail generation.

[13] 15 of the 28 States subject to regulation under CSAPR challenged the Rule. Coral Davenport, *Justices Back Rule Limiting Coal Pollution*, NY Times, April 29, 2014, *available at* http://www.nytimes.com/2014/04/30/us/politics/supreme-court-backs-epa-coal-pollution-rules.html.

[14] Following the D.C. Circuit's decision striking down CSAPR, prices dropped precipitously in allowance trading markets, falling from $150-$275/ton for Group 1 SO_2 allowances and $150-$300/ton for Group 2 SO_2 allowances to $10-$50/ton after the decision was announced.

[15] EPA finalized the NO_x SIP Call rule in September 1998, which required 22 States and the District of Columbia to submit SIPs that addressed regional transport of ground-level ozone. The rule required the States to put NO_x emission reduction measures into place by May 1, 2003. U.S. Environmental Protection Agency, Technology Transfer Network, NAAQS Ozone Implementation, *available at* http://www.epa.gov/ttn/naaqs/ozone/rto/sip/ (last accessed May 1, 2014).

[16] The D.C. Circuit previously held that EPA essentially "set the States up to fail" by failing to provide them with the emissions budgets prior to review of their initial SIPs and the Agency was required to provide the States with a "reasonable" period of time to review the budgets and propose implementation of the rule. *EME v. Homer City Generation*, 696 F.3d 7, 36-37 (D.C. Cir. 2012) (rev'd and remanded).

[17] *See Chevron U.S.A. Inc. v. Natural Resources Defense Council, Inc.*, 467 U.S. 837 (1984).

[18] The Court held that some "incidental" over-control might be necessary because EPA has a mandate to achieve attainment in every downwind state. Respondents identified few examples of potentially unnecessary emissions reductions and EPA was entitled to some leeway in fulfilling its statutory mandate. *EME Homer City*, at 31.

[19] Combined with existing state and EPA rules, EPA estimates CSAPR requires power plants in 23 States covered by the rule to reduce SO_2 emissions by 73 percent and NO_X emissions by 54 percent from 2005 levels. U.S. Environmental Protection Agency, EPA Fact Sheet, *available at* http://www.epa.gov/airtransport/CSAPR/pdfs/CSAPRFactsheet.pdf (last visited April 30, 2014).

[20] *See Michigan v. EPA*, 268 F.3d 1075, 1078 (D.C. Cir. 2001) (describing the Clean Air Act as an "experiment in federalism.").

[21] The Supreme Court indicated that immediate issuance of the FIP was reasonable in these circumstances given the D.C. Circuit's vacatur of CAIR and EPA's desire to avoid a lengthy delay in issuing a new rule. *EME Homer City*, at 18.

[22] Indeed, the Supreme Court specifically left open these avenues for individual sources and States to bring particularized challenges to CSAPR. *Id.* at 31.

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	) ss	ELECTRONIC FILING
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Kristie Lindstrom of the City of Duluth, County of St. Louis, State of Minnesota, says that on the 9th day of May, 2014, she served Minnesota Power's Comments in Docket No. E-999/CI-00-1636 to the Minnesota Public Utilities Commission and the Energy Resources Division of the Minnesota Department of Commerce via electronic filing. The remaining parties on the attached service list were served as so indicated on the list.

/s/ Kristie Lindstrom

Subscribed and sworn to before
me this 9th day of May, 2014.

/s/ Mary K Johnson

Notary Public - Minnesota
My Commission Expires Jan. 31, 2016

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