Abstract

Section 812 of the Clean Air Act Amendments of 1990 requires the Environmental Protection Agency (EPA) to periodically assess the effect of the Clean Air Act on the “public health, economy, and environment of the United States,” and to report the findings and results of its assessments to the Congress. Section 812 further directs EPA to evaluate the benefits and costs of the Clean Air Act’s implementation, taking into consideration the Act’s effects on public health, economic growth, the environment, employment, productivity, and the economy as a whole. This EPA Report to Congress presents the results and conclusions of the first section 812 assessment, a retrospective analysis of the benefits and costs of the Clean Air Act from 1970 to 1990. Future reports will detail the findings of prospective analyses of the benefits and costs of the Clean Air Act Amendments of 1990, as required by section 812.

This retrospective analysis evaluates the benefits and costs of emissions controls imposed by the Clean Air Act and associated regulations. The focus is primarily on the criteria pollutants sulfur dioxide, nitrogen oxides, carbon monoxide, particulate matter, ozone, and lead since essential data were lacking for air toxics. To determine the range and magnitude of effects of these pollutant emission reductions, EPA compared and contrasted two regulatory scenarios. The “control scenario” reflects the actual conditions resulting from the historical implementation of the 1970 and 1977 Clean Air Acts. In contrast, the “no-control” scenario reflects expected conditions under the assumption that, absent the passage of the 1970 Clean Air Act, the scope, form, and stringency of air pollution control programs would have remained as they were in 1970. The no-control scenario represents a hypothesized “baseline” against which to measure the effects of the Clean Air Act. The differences between the public health, air quality, and economic and environmental conditions resulting from these two scenarios represent the benefits and costs of the Act’s implementation from 1970 to 1990.

To identify and quantify the various public health, economic, and environmental differences between the control and no-control scenarios, EPA employed a sequence of complex modeling and analytical procedures. Data for direct compliance costs were used in a general equilibrium macroeconomic model to estimate the effect of the Clean Air Act on the mix of economic and industrial activity comprising the nation’s economy. These differences in economic activity were used to model the corresponding changes in pollutant emissions, which in turn provided the basis for modeling resulting differences in air quality conditions. Through the use of concentration-response functions derived from the scientific literature, changes in air quality provided the basis for calculating differences in physical effects between the two scenarios (e.g, reductions in the incidence of a specific adverse health effect, improvements in visibility, or changes in acid deposition rates). Many of the changes in physical effects were assigned an economic value on the basis of a thorough review and analysis of relevant studies from the economics, health effects, and air quality literature. The final analytical step involved aggregating these individual economic values and assessing the related uncertainties to generate a range of overall benefits estimates.

Comparison of emissions modeling results for the control and no-control scenarios indicates that the Clean Air Act has yielded significant pollutant emission reductions. The installation of stack gas scrubbers and the use of fuels with lower sulfur content produced a 40 percent reduction in 1990 sulfur dioxide emissions from electric utilities; total suspended particulate emissions were 75 percent lower as a result of controls on industrial and utility smokestacks. Motor vehicle pollution controls adopted under the Act were largely responsible for a 50 percent reduction in carbon monoxide emissions, a 30 percent reduction in emissions of nitrogen oxides, a 45

percent reduction in emissions of volatile organic compounds, and a near elimination of lead emissions. Several of these pollutants (primarily sulfur dioxide, nitrogen oxides, and volatile organic compounds) are precursors for the formation of ozone, particulates, or acidic aerosols; thus, emissions reductions have also yielded air quality benefits beyond those directly associated with reduced concentrations of the individual pollutants themselves.

The direct benefits of the Clean Air Act from 1970 to 1990 include reduced incidence of a number of adverse human health effects, improvements in visibility, and avoided damage to agricultural crops. Based on the assumptions employed, the estimated economic value of these benefits ranges from $5.6 to $49.4 trillion, in 1990 dollars, with a mean, or central tendency estimate, of $22.2 trillion. These estimates do not include a number of other potentially important benefits which could not be readily quantified, such as ecosystem changes and air toxics-related human health effects. The estimates are based on the assumption that correlations between increased air pollution exposures and adverse health outcomes found by epidemiological studies indicate causal relationships between the pollutant exposures and the adverse health effects.

The direct costs of implementing the Clean Air Act from 1970 to 1990, including annual compliance expenditures in the private sector and program implementation costs in the public sector, totaled $523 billion in 1990 dollars. This point estimate of direct costs does not reflect several potentially important uncertainties, such as the degree of accuracy of private sector cost survey results, that could not be readily quantified. The estimate also does not include several potentially important indirect costs which could not be readily quantified, such as the possible adverse effects of Clean Air Act implementation on capital formation and technological innovation.

Thus, the retrospective analysis of the benefits and costs of implementing the Clean Air Act from 1970 to 1990 indicates that the mean estimate of total benefits over the period exceeded total costs by more than a factor of 42. Taking into account the aggregate uncertainty in the estimates, the ratio of benefits to costs ranges from 10.7 to 94.5.

The assumptions and data limitations imposed by the current state of the art in each phase of the modeling and analytical procedure, and by the state of current research on air pollution’s effects, necessarily introduce some uncertainties in this result. Given the magnitude of difference between the estimated benefits and costs, however, it is extremely unlikely that eliminating these uncertainties would invalidate the fundamental conclusion that the Clean Air Act’s benefits to society have greatly exceeded its costs. Nonetheless, these uncertainties do serve to highlight the need for additional research into the public health, economic, and environmental effects of air pollution to reduce potential uncertainties in future prospective analyses of the benefits and costs of further pollution controls mandated by the Clean Air Act Amendments of 1990.

The Benefits and Costs of the Clean Air Act, 1970 to 1990

Prepared for
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by
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Contents

Tables .... ***xi***

Figures .... ***xv***

Acronyms and Abbreviations .... ***xvii***

Acknowledgments .... ***xxiii***

Executive Summary **ES-1**
- Purpose of the Study ES-1
- Study Design ES-1
- Study Review ES-1
- Summary of Results ES-2
  - Direct Costs ES-2
  - Emissions ES-2
  - Air Quality ES-3
  - Physical Effects ES-5
  - Economic Valuation ES-7
  - Monetized Benefits and Costs ES-8
- Alternative Results ES-9
- Conclusions and Future Directions ES-9

Chapter 1: Introduction **1**
- Background and Purpose 1
- Clean Air Act Requirements, 1970 to 1990 1
- Section 812 of the Clean Air Act Amendments of 1990 2
- Analytical Design and Review 2
  - Target Variable 2
  - Key Assumptions 2
  - Analytic Sequence 3
- Review Process 6
- Report Organization 6

Chapter 2: Cost and Macroeconomic Effects **7**
- Direct Compliance Costs 7
- Indirect Effects of the CAA 9
  - Sectoral Impacts 9
  - Aggregate Effects 9
- Uncertainties and Sensitivities in the Cost and Macroeconomic Analysis 10
  - Productivity and Technical Change 10
  - Discount Rates 11
  - Exclusion of Health Benefits from the Macroeconomic Model 12

Chapter 3: Emissions ... 13
- Sector-Specific Approach ... 15
- Summary of Results ... 15
- Uncertainty in the Emissions Estimates ... 17

Chapter 4: Air Quality ... 19
- General Methodology ... 20
- Sample Results ... 21
  - Carbon Monoxide ... 21
  - Sulfur Dioxide ... 22
  - Nitrogen Dioxide ... 22
  - Particulate Matter ... 23
  - Ozone ... 23
    - Urban Ozone ... 23
    - Rural Ozone ... 24
  - Acid Deposition ... 24
  - Visibility ... 25
- Uncertainty in the Air Quality Estimates ... 25

Chapter 5: Physical Effects ... 29
- Human Health and Welfare Effects Modeling Approach ... 29
  - Air Quality ... 29
  - Population ... 29
  - Health and Welfare Effects ... 29
- Key Analytical Assumptions ... 30
  - Mapping Populations to Monitors ... 32
  - Choice of Study ... 33
  - Variance Within Studies ... 33
  - PM-Related Mortality ... 34
    - Short-Term Exposure Studies ... 34
    - Long-Term Exposure Studies ... 35
- Health Effects Modeling Results ... 37
  - Avoided Premature Mortality Estimates ... 37
  - Non-Fatal Health Impacts ... 37
- Other Physical Effects ... 38
  - Ecological Effects ... 38
    - Aquatic and Forest Effects ... 38
    - Quantified Agricultural Effects ... 39
  - Effects of Air Toxics ... 39
  - Uncertainty in the Physical Effects Estimates ... 41

Chapter 6: Economic Valuation ... 43
- Methods for Valuation of Health and Welfare Effects ... 43
  - Mortality ... 44
  - Survey-Based Values ... 45
    - Chronic Bronchitis ... 45
    - Respiratory-Related Ailments ... 46
    - Minor Restricted Activity Days ... 46
    - Visibility ... 46
- Avoided Cost Estimates ... 46
  - Hypertension and Hospital Admissions ... 46
  - Household Soiling ... 47

Other Valuation Estimates ... 47
Changes in Children's IQ ... 47
Work Loss Days and Worker Productivity ... 48
Agricultural Benefits ... 48
Valuation Uncertainties ... 48
Mortity Risk Benefits Transfer ... 48

Chapter 7: Results and Uncertainty ... 51
Quantified Uncertainty in the Benefits Analysis ... 51
Aggregate Monetized Benefits ... 52
Comparison of Monetized Benefits and Costs ... 55
Major Sources of Uncertainty ... 56
Alternative Results ... 57
PM Mortality Valuation Based on Life-Years Lost ... 57
Alternative Discount Rates ... 58

Appendix A: Cost and Macroeconomic Modeling ... A-1
Introduction ... A-1
Macroeconomic Modeling ... A-1
Choice of Macroeconomic Model ... A-2
Overview of the Jorgenson-Wilcoxen Model ... A-2
Structure of the Jorgenson-Wilcoxen Model ... A-3
The Business Sector ... A-4
The Household Sector ... A-4
The Government Sector ... A-5
The Rest-of-the-World Sector ... A-5
Environmental Regulation, Investment, and Capital Formation ... A-5
The General Equilibrium ... A-5
Configuration of the No-control Scenario ... A-6
Elimination of Compliance Costs in the No-Control Case ... A-7
Capital Costs - Stationary Sources ... A-7
Operating and Maintenance Costs - Stationary Sources ... A-8
Capital Costs - Mobile Sources ... A-8
Operating and Maintenance - Mobile Sources ... A-8
Direct Compliance Expenditures Data ... A-8
Sources of Cost Data ... A-8
Cost of Clean Data ... A-8
EPA Data ... A-8
Commerce Data ... A-9
Stationary Source Cost Data ... A-9
Capital Expenditures Data ... A-9
Operation and Maintenance Expenditures Data ... A-10
Recovered Costs ... A-10
Mobile Source Cost Data ... A-11
Capital Expenditures Data ... A-11
Operation and Maintenance Expenditures Data ... A-11
Fuel Price Penalty ... A-11
Fuel Economy Penalty ... A-12
Inspection and Maintenance Programs ... A-13
Maintenance Credits ... A-13
Fuel Density Credits ... A-13
Other Direct Cost Data ... A-13

- Assessment Results A-14
  - Compliance Expenditures and Costs A-14
    - Annualization Method A-16
    - Discounting Costs and Expenditures A-19
  - Indirect Economic Effects of the CAA A-20
  - GNP and Personal Consumption A-20
  - Prices A-23
  - Sectoral Effects: Changes in Prices and Output by Industry A-23
  - Changes in Employment Across Industries A-25
- Uncertainties in the Cost Analysis A-26
  - Potential Sources of Error in the Cost Data A-26
  - Mobile Source Costs A-28
  - Stationary Source Cost Estimate Revisions A-29
  - Endogenous Productivity Growth in the Macro Model A-29
  - Amortization Period for Stationary Source Plant and Equipment A-30
- Cost and Macroeconomic Modeling References A-31

Appendix B: Emissions Modeling B-1
- Introduction B-1
- Comparison of Emissions Projections with Other EPA Data B-1
  - Control Scenario Projections Versus EPA Trends Projections B-1
  - No-Control Scenario Projections Versus Historical EPA Trends Data B-3
- Industrial Boilers and Processes B-4
  - Overview of Approach B-4
    - Industrial Boilers B-4
    - Industrial Processes and In-Process Fuel Combustion B-4
    - Establishment of Control Scenario Emissions B-5
    - Control Scenario Boiler Emissions B-5
    - Control Scenario Industrial Process Emissions B-7
- Development of Economic Driver Data for the Control Scenario - Industrial Boilers and Processes B-7
  - Economic Driver Data for Industrial Boiler Approach B-7
  - Economic Driver Data for the Industrial Process Approach B-8
  - No-control Scenario Emissions B-8
    - Industrial Boiler Emissions of SO2, NOx, and TSP B-8
    - Industrial Boiler Emissions of CO and VOC B-9
    - Industrial Process Emissions B-9
    - Lead Emissions B-9
- Off-Highway Vehicles B-10
  - Overview of Approach B-10
    - Development of Control Scenario B-11
    - No-control Scenario Emissions Estimates B-11
  - National and State-Level Off-Highway Emission Estimates B-11
- On-Highway B-12
  - Overview of Approach B-13
    - Personal Travel B-13
      - Iterative Proportional Fitting (IPF) B-13
      - Vehicle Ownership Projection (VOP) B-14
      - Projection of Vehicle Fleet Composition B-14
      - Activity/Energy Computation B-14
    - Goods Movement B-15
    - Other Transportation Activities B-15
  - Lead Emissions B-15
  - Estimation of No-control Scenario Emissions B-15
    - Development of Emission Factors B-15
    - Allocation of Highway Activity to States B-16

Development of Highway Pollutant Estimates ... B-16
Control Scenario Emissions Calculation ... B-16
No-control Scenario Emissions ... B-21
Utilities ... B-24
Overview of Approach ... B-24
Establishment of Control Scenario Emissions ... B-24
Key Assumptions in the Development of the ICF Analysis ... B-24
ARGUS Modeling Assumptions ... B-26
No-control Scenario Emissions ... B-27
ICF Estimates of SO2, TSP, and NOx Emissions in the No-control Scenario ... B-27
ARGUS No-control Scenario ... B-29
Estimation of Lead Emissions from Utilities ... B-29
CEUM Sensitivity Case ... B-30
Commercial/Residential ... B-30
Control Scenario Emissions ... B-31
Emissions Data ... B-32
Energy Data ... B-33
Economic/Demographic Data ... B-33
No-control Scenario Emissions ... B-34
Emissions Data ... B-34
Energy Data ... B-34
Economic/Demographic Data ... B-35
Emissions Modeling References ... B-39

Appendix C: Air Quality Modeling ... C-1
Introduction ... C-1
Carbon Monoxide ... C-1
Control scenario carbon monoxide profiles ... C-1
No-control scenario carbon monoxide profiles ... C-2
Summary differences in carbon monoxide air quality ... C-4
Key caveats and uncertainties for carbon monoxide ... C-4
Sulfur Dioxide ... C-5
Control scenario sulfur dioxide profiles ... C-5
No-control scenario sulfur dioxide profiles ... C-5
Summary differences in sulfur dioxide air quality ... C-6
Key caveats and uncertainties for sulfur dioxide ... C-6
Nitrogen Oxides ... C-6
Control scenario nitrogen oxides profiles ... C-7
No-control scenario nitrogen oxides profiles ... C-8
Summary differences in nitrogen oxides air quality ... C-8
Key caveats and uncertainties for nitrogen oxides ... C-8
Acid Deposition ... C-8
Control scenario acid deposition profiles ... C-9
No-control scenario acid deposition profiles ... C-11
Summary differences in acid deposition ... C-12
Key caveats and uncertainties for acid deposition ... C-12
Particulate Matter ... C-13
Control scenario particulate matter profiles ... C-14
No-control scenario particulate matter profiles ... C-15
Summary differences in particulate matter air quality ... C-16
Key caveats and uncertainties for particulate matter ... C-16
Ozone ... C-18
Control scenario ozone profiles ... C-21
No-control scenario ozone profiles ... C-21
Summary differences in ozone air quality ... C-23
Key caveats and uncertainties for ozone ... C-24

Visibility C-25
  Control scenario visibility C-25
  No-control scenario visibility C-26
  Summary differences in visibility C-26
    DeciView Haze Index C-26
    Modeling Results C-28
  Key caveats and uncertainties for visibility C-28
Air Quality Modeling References C-30

Appendix D: Human Health and Welfare Effects of Criteria Pollutants D-1
Introduction D-1
Principles for the Section 812 Benefits Analysis D-1
General Modeling Approach D-2
Quantifying Changes in Pollutant Exposures D-2
  Air Quality D-2
  Population Distribution D-3
    Census Data D-3
    Gridding U.S. Population D-4
    Allocating Exposure Estimates to the Population D-4
      Method One D-4
      Method Two D-4
Estimating Human Health Effects of Exposure D-5
  Types of Health Studies D-5
    Epidemiological Studies D-6
    Human Clinical Studies D-7
  Issues in Selecting Studies To Estimate Health Effects D-9
    Peer-Review of Research D-9
    Confounding Factors D-9
    Uncertainty D-10
    Magnitude of Exposure D-11
    Duration of Exposure D-11
    Thresholds D-11
    Target Population D-11
    Statistical Significance of Exposure-Response Relationships D-12
    Relative Risks D-12
    Baseline Incidence Data D-12
  Estimating Mortality Effects D-13
    Using PM as an Indicator D-13
    Estimating the Relationship Between PM and Premature Mortality D-13
    Prematurity of Mortality: Life-Years Lost as a Unit of Measure D-16
  Estimating Morbidity Effects D-19
    Overlapping Health Effects D-19
    Studies Requiring Adjustments D-19
  Concentration-Response Functions: Health Effects D-19
    Particulate Matter D-19
    Ozone D-26
    Nitrogen Oxides D-34
    Carbon Monoxide D-36
    Sulfur Dioxide D-38
Estimating Welfare Effects of Exposure D-40
  Agricultural Effects D-40
  Materials Damage D-41
  Visibility D-41
  Worker Productivity D-41
  Ecological Effects D-41
Modeling Results D-44
Human Health and Welfare Effects References D-48

Appendix E: Ecological Effects of Criteria Pollutants ... E-1
Introduction ... E-1
Benefits From Avoidance of Damages to Aquatic Ecosystems ... E-1
  Acid Deposition ... E-2
    Background ... E-2
    Current Impacts of Acid Deposition ... E-2
      Effects on Water Chemistry ... E-2
      Effects on Fish Habitat Quality ... E-4
      Economic Damages to Recreational Fishing ... E-5
    Benefits From Acid Deposition Avoidance Under the CAA ... E-5
      Recreational Fishing ... E-5
  Eutrophication ... E-6
    Atmospheric Deposition and Eutrophication ... E-7
    Valuing Potential Benefits from Eutrophication Avoidance Under the CAA ... E-7
  Mercury ... E-8
Benefits from Avoided Damages to Wetland Ecosystems ... E-9
  Introduction ... E-9
  Effects of Acidification ... E-9
  Effects of Nutrient Loading ... E-10
  Summary of Wetland Ecosystem Effects ... E-11
Benefits from Avoided Damages to Forests ... E-11
  Introduction ... E-11
  Current Air Pollutant Effects on Forests ... E-12
    Acid Deposition Impacts ... E-12
    Ozone Impacts ... E-12
      Experimental Evidence ... E-12
      Observational Evidence ... E-13
    Endangered species ... E-14
  Valuation of Benefits From CAA-Avoided Damages to Forests ... E-14
    Background ... E-14
    Commercial Timber Harvesting ... E-15
    Non-marketed Forest Services ... E-16
Ecosystem Effects References ... E-18

Appendix F: Effects of Criteria Pollutants on Agriculture ... F-1
Introduction ... F-1
Ozone Concentration Data ... F-1
  Control and No-control Scenario Ozone Concentration Data ... F-2
  Calculation of the W126 Statistic ... F-2
  Aggregating Ozone Data to the County Level ... F-3
Yield Change Estimates ... F-3
  Exposure-Response Functions ... F-3
    Minimum/Maximum Exposure-Response Functions ... F-4
  Calculation of Ozone Indices ... F-4
  Calculations of County Weights ... F-5
  Calculation of Percent Change in Yield ... F-5
Economic Impact Estimates ... F-5
  Agricultural Simulation Model (AGSIM) ... F-5
Conclusions ... F-9
Agricultural Effects References ... F-10

Appendix G: Lead Benefits Analysis ... G-1
Introduction ... G-1
Methods Used to Measure and Value Health Effects ... G-2
  Health Benefits to Children ... G-2

Changes in IQ G-2
Quantifying the Relationship Between Blood Lead Levels and IQ G-2
Valuing Changes in Children's Intelligence G-3
Children with IQs Less Than 70 ..... G-7
Quantifying the Number of Children with IQs Less than 70 ..... G-7
Valuing the Reduction in Number of Children with IQs less than 70 ..... G-8
Changes in Neonatal Mortality G-8
Quantifying the relationship between PbB levels and neonatal mortality G-8
Valuing changes in neonatal mortality G-8
Health Benefits to Men G-8
Hypertension G-9
Quantifying the relationship between PbB levels and hypertension G-9
Valuing reductions in hypertension G-9
Quantifying the relationship between blood lead and blood pressure G-9
Changes In Coronary Heart Disease G-10
Quantifying the relationship between blood pressure and coronary heart disease G-10
Valuing reductions in CHD events G-11
Changes in Initial Cerebrovascular Accidents and Initial Atherothrombotic Brain Infarctions G-12
Quantifying the relationship between blood pressure and first-time stroke G-12
Valuing reductions in strokes G-12
Changes in Premature Mortality G-13
Quantifying the relationship between blood pressure and premature mortality G-13
Valuing reductions in premature mortality G-13
Health Benefits to Women G-13
Changes in Coronary Heart Disease G-14
Quantifying the relationship between blood pressure and coronary heart disease G-14
Valuing reductions in CHD events G-14
Changes in Atherothrombotic Brain Infarctions and Initial Cerebrovascular Accidents G-14
Quantifying the relationship between blood pressure and first-time stroke G-14
Valuing reductions in strokes G-15
Changes in Premature Mortality G-15
Quantifying the relationship between blood pressure and premature mortality G-15
Quantifying Uncertainty G-15
Characterizing Uncertainty Surrounding the Dose-Response Relationships G-15
Characterizing Uncertainty Surrounding the Valuation Estimates G-15
Industrial Processes and Boilers and Electric Utilities G-16
Methods Used to Determine Changes in Lead Emissions from Industrial Processes from 1970 to 1990 ..... G-16
TRI Data G-16
Derivation of Industrial Process Emissions Differentials 1970-1990 ..... G-17
Data sources G-17
Estimates of industrial process emissions in the control scenario G-17
Estimates of industrial process emissions in the no-control scenario G-18
Matching TRI Data to Industrial Process Emissions Differentials G-18
Methods Used to Determine Changes in Lead Emissions from Industrial Boilers from 1970 to 1990 ..... G-19
TRI Data G-19
Derivation of Industrial Combustion Emissions 1970-1990 ..... G-20
Estimates of combustion emissions under the control scenario G-20
Estimates of combustion emissions under the no-control scenario G-20
Matching TRI Data to Industrial Combustion Emissions Data G-21
Methods Used to Determine Changes in Lead Emissions from Electric Utilities from 1975 to 1990 ..... G-21
Coal-Use Data G-21
The EPA Interim Emissions Inventory G-21
Matching the Coal-Use Data to the Interim Emissions Inventory G-22
Emissions Factors and Control Efficiencies G-22

      - Use of Air Dispersion Modeling to Estimate Ambient Air Lead Levels G-23
      - Determination of Blood Lead Levels from Air Lead Concentrations G-23
        - Relationship Between Air Lead Concentrations and Blood Lead Levels G-23
          - Children G-25
          - Adults G-25
          - Individuals with initial blood lead levels of 30 µg/dL and greater G-26
        - Estimates of Initial Blood Lead Concentrations G-26
      - Combination of Air Concentration Estimates with Population Data G-27
      - Results G-28
    - Reduction in Health Effects Attributable to Gasoline Lead Reductions G-31
      - Estimating Changes in Amount of Lead in Gasoline from 1970 to 1990 .... G-31
      - Estimating the Change in Blood Lead Levels from the Change in the Amount of Lead in Gasoline G-31
        - 1970-Forward and 1990-Backward Approaches G-32
        - Relating Blood Lead Levels to Population Health Effects G-32
        - Changes in Leaded Gasoline Emissions and Resulting Decreased Blood Lead Levels and Health Effects G-32
    - Lead Benefits Analysis References G-36

Appendix H: Air Toxics H-1
- Introduction H-1
- Limited Scope of this Assessment H-1
- History of Air Toxics Standards under the Clean Air Act of 1970 .... H-2
- Quantifiable Stationary Source Air Toxics Benefits H-3
  - EPA Analyses of Cancer Risks from Selected Air Toxic Pollutants H-3
  - Cancer Risk Estimates from NESHAP Risk Assessments H-4
- Non-utility Stationary Source Cancer Incidence Reductions H-4
  - PES Study H-5
    - Methodology H-5
    - Findings H-6
  - ICF Re-analysis H-7
    - Methodology H-7
    - Findings H-8
- Mobile Source HAP Exposure Reductions H-9
  - Methodology H-10
  - Results H-10
- Non-Cancer Health Effects H-11
- Ecological Effects H-11
- Conclusions — Research Needs H-12
  - Health Effects H-12
  - Exposure Assessment H-13
  - Ecosystem Effects H-13
  - Economic Valuation H-13
- Air Toxics References H-14

Appendix I: Valuation of Human Health and Welfare Effects of Criteria Pollutants I-1
- Methods Used to Value Health and Welfare Effects I-1
  - Valuation of Specific Health Endpoints I-3
    - Valuation of Premature Mortality Avoided I-3
    - Valuation of Hospital Admissions Avoided I-3
    - Valuation of Chronic Bronchitis Avoided I-4
    - Valuation of Other Morbidity Endpoints Avoided I-6
  - Valuation of Welfare Effects I-6
    - Visibility Valuation I-6
- Results of Valuation of Health and Welfare Effects I-16

- Uncertainties I-16
  - The Effect of Discount Rates I-20
  - The Relative Importance of Different Components of Uncertainty I-20
  - Economic Benefits Associated with Reducing Premature Mortality I-21
- Economic Valuation References I-27

Appendix J: Future Directions J-1

- Research Implications J-1
  - Research Topics to Reduce Uncertainty J-1
  - Research Topics to Improve Comprehensiveness J-3
- Future Section 812 Analyses J-4

Tables

Table ES-1 Criteria Pollutant Health Benefits - Distributions of 1990 Incidences of Avoided Health Effects (In Thousands of Incidences Reduced) for 48 State Population. ES-4
Table ES-2 Major Nonmonetized, Adverse Effects Reduced by the Clean Air Act. ES-5
Table ES-3 Central Estimates of Economic Value per Unit of Avoided Effect (In 1990 Dollars). ES-6
Table ES-4 Total Monetized Benefits by Endpoint Category for 48 State Population for 1970 to 1990 Period (In Billions of 1990 Dollars) ES-7
Table ES-5 Alternative Mortality Benefits Mean Estimates for 1970 to 1990 (In Trillions of 1990 Dollars) Compared to Total 1970 to 1990 Compliance Costs ES-9

Table 1 Estimated Annual CAA Compliance Costs ($Billions). 8
Table 2 Compliance Cost, GNP, and Consumption Impacts Discounted to 1990 ($1990 Billions) 11
Table 3 Summary of Sector-Specific Emission Modeling Approaches. 14
Table 4 Uncertainties Associated with Emissions Modeling. 18
Table 5 Key Uncertainties Associated with Air Quality Modeling. 26
Table 6 Human Health Effects of Criteria Pollutants. 31
Table 7 Selected Welfare Effects of Criteria Pollutants. 32
Table 8 Percent of Population (of the Continental US) Within 50km of a Monitor (Or in a County with PM monitors), 1970-1990. 33
Table 9 Criteria Pollutants Health Benefits — Distributions of 1990 Avoided Premature Mortalities (Thousands of Cases Reduced) for 48 State Population 37
Table 10 Criteria Pollutants Health Benefits — Distributions of 1990 Non-Fatal Avoided Incidence (Thousands of Cases Reduced) for 48 State Population 38
Table 11. Health and Welfare Effects of Hazardous Air Pollutants. 40
Table 12 Uncertainties Associated with Physical Effects Modeling. 42
Table 13 Health and Welfare Effects Unit Valuation (1990 Dollars). 44
Table 14 Summary of Mortality Valuation Estimates (Millions of $1990) 45
Table 15 Estimating Mortality Risk Based on Wage-Risk Studies: Potential Sources and Likely Direction of Bias. 50
Table 16 Present Value of 1970 to 1990 Monetized Benefits by Endpoint Category for 48 State Population (Billions of $1990, Discounted to 1990 at 5 Percent) 52
Table 17 Total Monetized Benefits for 48 State Population (Present Value in Billions of 1990 Dollars, Discounted to 1990 at 5 Percent) 53
Table 18 Quantified Uncertainty Ranges for Monetized Annual Benefits and Benefit/Cost Ratios, 1970-1990 (In Billions of 1990-Value Dollars). 55
Table 19 Alternative Mortality Benefits Mean Estimates for 1970 to 1990 (in Trillions of 1990 Dollars, Discounted at 5 percent) Compared to Total 1970 to 1990 Compliance Costs 57
Table 20 Effect of Alternative Discount Rates on Present Value of Total Monetized Benefits/Costs for 1970 to 1990 (In Trillions of 1990 Dollars). 57

Table A-1 Key Distinguishing Characteristics of the Jorgenson-Wilcoxen Model. A-3
Table A-2 Definitions of Industries Within the J/W Model. A-4
Table A-3 Estimated Capital and O&M Expenditures for Stationary Source Air Pollution Control (Millions of Current Dollars). A-10
Table A-4 Estimated Recovered Costs for Stationary Source Air Pollution Control (Millions of Current Dollars). A-11
Table A-5 Estimated Capital and Operation and Maintenance Expenditures for Mobile Source Air Pollution Control (Millions of Current Dollars). A-12

Table A-6 O&M Costs and Credits (Millions of Current Dollars). A-12
Table A-7 Other Air Pollution Control Expenditures (Millions of Current Dollars). A-14
Table A-8 Summary of Expenditures and Conversion to 1990 Dollars (Millions of Dollars) A-15
Table A-9 Annualized Costs, 1973-1990 (Millions of 1990 Dollars; Capital Expenditures Annualized at 5 Percent) A-16
Table A-10 Amortization of Capital Expenditures for Stationary Sources (Millions of 1990 Dollars) A-17
Table A-11 Amortization of Capital Expenditures for Mobile Sources (Millions of 1990 Dollars) A-18
Table A-12 Compliance Expenditures and Annualized Costs, 1973 to 1990 ($1990 millions) A-19
Table A-13 Costs Discounted to 1990 ($1990 Millions) A-20
Table A-14 Differences in Gross National Product Between the Control and No-Control Scenarios A-20
Table A-15 Difference in Personal Consumption Between the Control and No-Control Scenarios A-21
Table A-16 GNP and Consumption Impacts Discounted to 1990 ($1990 Billions) A-21
Table A-17 Percentage Difference in Energy Prices Between the Control and No-Control Scenarios. A-23
Table A-18 Potential Sources of Error and Their Effect on Total Costs of Compliance. A-26
Table A-19 Stationary Source O&M Expenditures as a Percentage of Capital Stock (Millions of 1990 Dollars) A-27
Table A-20 Comparison of EPA and BEA Stationary Source Expenditure Estimates (Millions of Current Dollars) A-28
Table A-21 BEA Estimates of Mobile Source Costs. A-29
Table A-22 Annualized Costs Assuming 40-Year Stationary Source Capital Amortization Period, 1973 to 1990 ($1990 Millions). A-30
Table A-23 Effect of Amortization Periods on Annualized Costs Discounted to 1990 (Billions of $1990) A-30

Table B-1 Correspondence Between Process Emissions Categories Used by MSCET, *Trends*, and J/W Industrial Sectors and Identifier Codes. B-6
Table B-2 Fuel Use Changes Between Control and No-control Scenarios. B-9
Table B-3 Difference in Control and No-control Scenario Off-Highway Mobile Source Emissions. B-12
Table B-4 Sources of Data for Transportation Sector Control Scenario Activity Projection. B-17
Table B-5 Distribution of Households by Demographic Attributes for Control Scenario. B-18
Table B-6 Economic and Vehicle Usage Data for Vehicle Ownership Projection Control Scenario. B-19
Table B-7 Control Scenario Personal Characteristics. B-20
Table B-8 Distribution of Households by Income Class for No-Control Scenario. B-21
Table B-9 Economic and Vehicle Usage Data for Vehicle Ownership Projection No-Control Scenario B-22
Table B-10 Percent Changes in Key Vehicle Characteristics Between the Control and No-Control Scenarios. B-23
Table B-11 J/W Estimates of Percentage Increases in National Electricity Generation Under No-Control Scenario. B-29
Table B-12 *Trends* Source Categories and (1975 to 1985) Scaling Factors for TSP and CO. B-33
Table B-13 Percentage Change in Real Energy Demand by Households from Control to No-Control Scenario. B-34
Table B-14 Percentage Change in Commercial Energy Demand from Control to No-Control Scenario. B-35
Table B-15 J/W Percent Differential in Economic Variables Used in CRESS. B-35
Table B-16 TSP Emissions Under the Control and No-Control Scenarios by Target Year (In Thousands of Short Tons). B-36
Table B-17 SO2 Emissions Under the Control and No-Control Scenarios by Target Year (In Thousands of Short Tons). B-36

Table B-18 NOx Emissions Under the Control and No-Control Scenarios by Target Year (In Thousands of Short Tons). B-37
Table B-19 VOC Emissions Under the Control and No-Control Scenarios by Target Year (In Thousands of Short Tons). B-37
Table B-20 CO Emissions Under the Control and No-Control Scenarios by Target Year (In Thousands of Short Tons). B-38
Table B-21 Lead (Pb) Emissions Under the Control and No-Control Scenarios by Target Year (In Thousands of Short Tons) B-38

Table C-1 Summary of CO Monitoring Data C-2
Table C-2 Format of Air Quality Profile Databases C-3
Table C-3 Summary of SO2 Monitoring Data. C-5
Table C-4 Summary of NO2 Monitoring Data. C-7
Table C-5 Summary of NO Monitoring Data. C-7
Table C-6 Summary of TSP Monitoring Data C-14
Table C-7 Summary of PM10 Monitoring Data C-15
Table C-8 Fine Particle (PM2.5) Chemical Composition by U.S. Region. C-16
Table C-9 Coarse Particle (PM2.5 to PM10) Chemical Composition by U.S. Region. C-17
Table C-10 PM Control Scenario Air Quality Profile Filenames. C-17
Table C-11 PM No-Control Scenario Air Quality Profile Filenames. C-18
Table C-12 Urban Areas Modeled with OZIPM4. C-19
Table C-13 Summary of Ozone Monitoring Data. C-21
Table C-14 Apportionment of Emissions Inventories for SAQM Runs. C-22
Table C-15 1990 Control Scenario Visibility Conditions for 30 Southwestern U.S. Cities. C-27
Table C-16 1990 No-control Scenario Visibility Conditions for 30 Southwestern U.S. Cities. C-27
Table C-17 Summary of Relative Change in Visual Range and DeciView Between 1990 Control and No-Control Scenario Visibility Conditions for 30 Southwestern U.S. Cities. C-29

Table D-1 Criteria Air Pollutant Monitors in the U.S., 1970 - 1990. D-3
Table D-2 Population Coverage in the “Within 50 km” Model Runs (Percent of Continental U.S. Population). D-4
Table D-3 Population Coverage for “Extrapolated to All U.S.” Model Runs (Percent of Continental U.S. Population). D-5
Table D-4 Human Health Effects of Criteria Pollutants. D-6
Table D-5 PM2.5/PM10 Ratios Used to Estimate PM2.5 Data Used With Pope et al. (1995) Mortality Relationship D-16
Table D-6 Summary of Concentration-Response Functions for Particulate Matter D-20
Table D-7 Summary of Concentration-Response Functions for Ozone D-27
Table D-8 Summary of Concentration-Response Functions for NO2 D-35
Table D-9 Summary of Concentration-Response Functions for Carbon Monoxide D-37
Table D-10 Summary of Concentration-Response Functions for Sulfur Dioxide D-39
Table D-11 Selected Welfare Effects of Criteria Pollutants D-40
Table D-12 Summary of Functions Quantifying Welfare Benefits D-42
Table D-13 Criteria Pollutants Health Effects — Extrapolated to 48 State U.S. Population (Cases Per Year-Mean Estimates) D-45
Table D-14 Mortality Distribution by Age: Proportion of PM- and Pb-related Premature Mortalities and Associated Life Expectancies D-46
Table D-15 Quantified Benefits Which Could Not Be Monetized — Extrapolated to the Entire 48 State Population. D-47

Table E-1 Summary of Biological Changes with Surface Water Acidification. E-3
Table E-2 Comparison of Population of Acidic National Surface Water Survey (NSWS) by Chemical Category E-4
Table E-3 Results from Benefits Assessments of Aquatic Ecosystem Use Values from Acid Deposition Avoidance E-6

Table F-1 Agriculture Exposure-Response Functions. F-4
Table F-2 Relative No-Control to Control Percent Yield Change (harvested acres) for the Minimum Scenario. F-6
Table F-3 Relative No-Control to Control Percent Yield Change (harvested acres) for the Maximum Scenario. F-6
Table F-4 Change in Farm Program Payments, Net Crop Income, Consumer Surplus, and Net Surplus Due to the CAA (Millions 1990 $). F-8

Table G-1 Quantified and Unquantified Health Effects of Lead. G-1
Table G-2 Uncertainty Analysis: Distributions Associated With Dose-Response Coefficients Used to Estimate Lead Health Effects G-16
Table G-3 Air Modeling Parameters. G-24
Table G-4 Estimated Indirect Intake Slopes: Increment of Blood Lead Concentration (in µg/dL) per Unit of Air Lead Concentration (µg/m3). G-26
Table G-5 Estimated Lead Emissions from Electric Utilities, Industrial Processes, and Industrial Combustion (in Tons). G-28
Table G-6 Yearly Differences in Number of Health Effects Between the Control and No-Control Scenarios: Industrial Processes, Boilers, and Electric Utilities (Holding Other Lead Sources at Constant 1970 Levels) G-29
Table G-7 Yearly Differences in Number of Health Effects Between the Controlled and Uncontrolled Scenarios: Industrial Processes, Boilers, and Electric Utilities (Holding Other Lead Sources at Constant 1990 Levels) G-30
Table G-8 Lead Burned in Gasoline (In Tons). G-33
Table G-9 Yearly Differences in Number of Health Effects Between the Control and No-Control Scenarios: Lead in Gasoline only (Holding Other Lead Sources at Constant 1970 Levels). G-34
Table G-10 Yearly Differences in Number of Health Effects Between the Control and No-Control Scenarios: Lead in Gasoline only (Holding Other Lead Sources at Constant 1990 Levels). G-35

Table H-1 Health and Welfare Effects of Hazardous Air Pollutants. H-2
Table H-2 Cancer Incidence Reductions and Monetized Benefits for NESHAPs. H-5

Table I-1 Summary of Mortality Valuation Estimates (Millions of 1990 Dollars) I-3
Table I-2 Unit Values Used for Economically Valuing Health and Welfare Endpoints I-8
Table I-3 Criteria Pollutants Health and Welfare Benefits — Extrapolated to Entire 48 State Population Present Value (In 1990 Using 5% Discount Rate) of Benefits from 1970-1990 (In Billions of 1990 Dollars). I-17
Table I-4 Present Value of 1970 to 1990 Monetized Benefits by Endpoint Category for 48 State Population (Billions of 1990 Dollars, Discounted to 1990 at 5 Percent) I-18
Table I-5 Monte Carlo Simulation Model Results for Target Years, Plus Present Value in 1990 Terms of Total Monetized Benefits for Entire 1970 to 1990 Period (In Billions of 1990-Value Dollars). I-18
Table I-6 Comparison of 1990 (Single Year) Monetized Benefits by Endpoint for 48 State Population and Monitored Areas (In Millions of 1990 Dollars). I-19
Table I-7 Effect of Alternative Discount Rates on Present Value of Total Monetized Benefits for 1970 to 1990 (In Trillions of 1990 Dollars). I-20
Table I-8 Alternative Estimates of the Present Value of Mortality Associated With PM (Based on Pope et al., 1996, in Trillions of 1990 Dollars). I-25

Figures

Figure ES-1 Total Direct Compliance Costs of the CAA (in billions of inflation-adjusted dollars.) ES-1
Figure ES-2 1990 Control and No-control Scenario Emissions (in millions of short tons). ES-2
Figure ES-3 Total Direct Costs and Monetized Direct Benefits of the Clean Air Act, 1970 to 1990 (in trillions of 1990 dollars). ES-8

Figure 1 Summary of Analytical Sequence and Modeled versus Historical Data Basis. 4
Figure 2 Control and No-control Scenario Total SO_2 Emission Estimates. 16
Figure 3 Control and No-control Scenario Total NO_x Emission Estimates. 16
Figure 4 Control and No-control Scenario Total VOC Emission Estimates. 16
Figure 5 Control and No-control Scenario Total CO Emission Estimates. 16
Figure 6 Control and No-control Scenario Total TSP Emission Estimates. 16
Figure 7 Control and No-control Scenario Total Pb Emission Estimates. 16
Figure 8 Frequency Distribution of Estimated Ratios for 1990 Control to No-control Scenario 95th Percentile 1-Hour Average CO Concentrations, by Monitor. 21
Figure 9 Frequency Distribution of Estimated Ratios for 1990 Control to No-control Scenario 95th Percentile 1-Hour Average SO_2 Concentrations, by Monitor. 22
Figure 10 Frequency Distribution of Estimated Ratios for 1990 Control to No-control Scenario 95th Percentile 1-Hour Average NO_2 Concentrations, by Monitor. 23
Figure 11 Distribution of Estimated Ratios for 1990 Control to No-Control Annual Mean TSP Concentrations, by Monitored County. 23
Figure 12 Distribution of Estimated Ratios for 1990 Control to No-control OZIPM4 Simulated 1-Hour Peak Ozone Concentrations, by Urban Area. 23
Figure 13 Distribution of Estimated Ratios for 1990 Control to No-control SAQM Simulated Daytime Average Ozone Concentrations, by SAQM Monitor. 24
Figure 14 Distribution of Estimated Ratios for 1990 Control to No-control RADM Simulated Daytime Average Ozone Concentrations, by RADM Grid Cell. 24
Figure 15 RADM-Predicted Percent Increase in Total Sulfur Deposition (Wet + Dry) Under the No-control Scenario. 24
Figure 16 RADM-Predicted Percent Increase in Total Nitrogen Deposition (Wet + Dry) Under the No-control Scenario. 25
Figure 17 RADM-Predicted Increase in Visibility Degradation, Expressed in DeciViews, for Poor Visibility Conditions (90th Percentile) Under the No-control Scenario. 25
Figure 18 Monte Carlo Simulation Model Results for Target Years (in billions of 1990 dollars) 54
Figure 19 Distribution of 1990 Monetized Benefits of CAA (in billions of 1990 dollars) 54
Figure 20 Uncertainty Ranges Deriving From Individual Uncertainty Factors 55

Figure A-1 Percent Difference in Real Investment Between Control and No-control Scenarios. A-22
Figure A-2 Percent Difference in Price of Output by Sector Between Control and No-control Scenario for 1990. A-22
Figure A-3 Percent Difference in Quantity of Output by Sector Between Control and No-control Scenario for 1990. A-24
Figure A-4 Percent Difference in Employment by Sector Between Control and No-control Scenario for 1990. A-24

Figure B-1 Comparison of Control, No-control, and *Trends* SO_2 Emission Estimates. B-2
Figure B-2 Comparison of Control, No-control, and *Trends* NO_x Emission Estimates. B-2
Figure B-3 Comparison of Control, No-control, and *Trends* VOC Emission Estimates. B-2
Figure B-4 Comparison of Control, No-control, and *Trends* CO Emission Estimates. B-2
Figure B-5 Comparison of Control, No-Control, and *Trends* TSP Emission Estimates B-2

Figure C-1 Frequency Distribution of Estimated Ratios for 1990 Control to No-Control Scenario 95th Percentile 1-Hour Average CO Concentrations, by Monitor C-4
Figure C-2 Frequency Distribution of Estimated Ratios for 1990 Control to No-control Scenario 95th Percentile 1-Hour Average SO_2 Concentrations, by Monitor. C-6
Figure C-3 Frequency Distribution of Estimated Ratios for 1990 Control to No-control Scenario 95th Percentile 1-Hour Average NO_2 Concentrations, by Monitor. C-8
Figure C-4 Location of the High Resolution RADM 20-km Grid Nested Inside the 80-km RADM Domain. C-9
Figure C-5 RADM-Predicted 1990 Total Sulfur Deposition (Wet + Dry; in kg/ha) Under the Control Scenario. C-10
Figure C-6 RADM-Predicted 1990 Total Nitrogen Deposition (Wet + Dry; in kg/ha) Under the Control Scenario. C-10
Figure C-7 RADM-Predicted 1990 Total Sulfur Deposition (Wet + Dry; in kg/ha) Under the No-control Scenario. C-11
Figure C-8 RADM-Predicted 1990 Total Nitrogen Deposition (Wet + Dry; in kg/ha) Under the No-control Scenario. C-11
Figure C-9 RADM-Predicted Percent Increase in Total Sulfur Deposition (Wet + Dry; in kg/ha) Under the No-control Scenario. C-12
Figure C-10 RADM-Predicted Percent Increase in Total Nitrogen Deposition (Wet + Dry; in kg/ha) Under the No-control Scenario. C-12
Figure C-11 Distribution of Estimated Ratios for 1990 Control to No-Control Annual Mean TSP Concentrations, by Monitored County. C-18
Figure C-12 RADM and SAQM Modeling Domains, with Rural Ozone Monitor Locations. C-20
Figure C-13 Distribution of Estimated Ratios for 1990 Control to No-control OZIPM4 Simulated 1-Hour Peak Ozone Concentrations, by Urban Area. C-23
Figure C-14 Distribution of Estimated Ratios for 1990 Control to No-control RADM-Simulated Daytime Average Rural Ozone Concentrations, by RADM Grid Cell. C-23
Figure C-15 Distribution of Estimated Ratios for 1990 Control to No-control SAQM-Simulated Daytime Average Ozone Concentrations, by SAQM Monitor. C-23
Figure C-16 RADM-Predicted Visibility Degradation, Expressed in Annual Average DeciView, for Poor Visibility Conditions (90th Percentile) Under the Control Scenario. C-26
Figure C-17 RADM-Predicted Visibility Degradation, Expressed in Annual Average DeciView, for Poor Visibility Conditions (90th Percentile) Under the No-Control Scenario. C-26
Figure C-18 RADM-Predicted Increase in Visibility Degradation, Expressed in Annual Average DeciView, for Poor Visibility Conditions (90th Percentile) Under the No-Control Scenario. C-28

Figure H-1 PES Estimated Reductions in HAP-Related Cancer Cases. H-7
Figure H-2 ICF Estimated Reductions in Total HAP-Related Cancer Cases Using Upper Bound Asbestos Incidence and Lower Bound Non-Asbestos HAP Incidence. H-8
Figure H-3 ICF Estimated Reduction in Total HAP-Related Cancer Cases Using Upper Bound Incidence for All HAPs. H-8
Figure H-4 National Annual Average Motor Vehicle HAP Exposures (µg/m3). H-11

Figure I-1 Monte Carlo Simulation Model Results for Target Years (in billions of 1990 dollars) I-19
Figure I-2 Uncertainty Ranges Deriving From Individual Uncertainty Factors I-21

Acronyms and Abbreviations

μeq/L	microequivalents per liter
μg/m^3	micrograms per cubic meter
μg	micrograms
μm	micrometers, also referred to as microns
ACCACAPERS	SAB Advisory Council on Clean Air Compliance Analysis Physical Effects Review Subcommittee
AGSIM	AGricultural SImulation Model
AIRS	EPA Aerometric Information Retrieval System
Al^{3+}	aluminum
ANC	acid neutralizing capacity
ANL	Argonne National Laboratories
APPI	Argonne Power Plant Inventory
AQCR	Air Quality Control Region
ARGUS	Argonne Utility Simulation Model
ASI	Acid Stress Index
ATERIS	Air Toxic Exposure and Risk Information System
ATLAS	Aggregate Timberland Assessment System
AUSM	Advanced Utility Simulation Model
BEA	Bureau of Economic Analysis
b_{ext}	total light extinction
BG/ED	Block Group / Enumeration District
BI	atherothrombotic brain infarction
BID	Background Information Document
BP	blood pressure
BTU	British Thermal Unit
c.i.	confidence interval
CA	cerebrovascular accident
CAA	Clean Air Act
CAAA90	Clean Air Act Amendments of 1990
CAPMS	EPA’s Criteria Air Pollutant Modeling System
CARB	California Air Resources Board
CASAC	SAB Clean Air Scientific Advisory Committee
CDC	Centers for Disease Control (now CDCP, Centers for Disease Control and Prevention)
CERL	EPA/ORD Corvallis Environmental Research Laboratory (old name; see NERL)
CEUM	ICF Coal and Electric Utility Model
CHD	coronary heart disease
CIPP	changes in production processes
CO	carbon monoxide
CO_2	carbon dioxide
COH	coefficient of haze
COHb	blood level of carboxyhemoglobin
COPD	chronic obstructive pulmonary disease
Council	SAB Advisory Council on Clean Air Compliance Analysis
CPUE	catch per unit effort

CR	concentration-response
CRESS	Commercial and Residential Simulation System model
CSTM	Coal Supply and Transportation Model
CTG	Control Techniques Guidelines
CV	contingent valuation
CVM	contingent valuation method
D.C.	District of Columbia
DBP	diastolic blood pressure
DDE	dichlorodiphenyldichloroethylene
DDT	dichlorodiphenyltrichloroethane
$DFEV_1$	decrement of forced expiratory volume (in one second)
dL	deciliter
DOC	Department of Commerce
DOE	Department of Energy
DOI	Department of Interior
DRI	Data Resources Incorporated
dV	DeciView Haze Index
DVSAM	Disaggregate Vehicle Stock Allocation Model
EC	extinction coefficient
EDB	ethylene dibromide
EDC	ethylene dichloride
EFI	Electronic Fuel Injection
EI	Electronic Ignition
EIA	Energy Information Administration
EKMA	Empirical Kinetics Modeling Approach
ELI	Environmental Law Institute
EOL	end-of-line
EPA	Environmental Protection Agency
EPRI	Electric Power Research Institute
ESEERCO	Empire State Electric Energy Research Corporation
ESP	electrostatic precipitator
FERC	Federal Energy Regulatory Commission
FEV_1	forced expiratory volume (in one second)
FGD	flue gas desulfurization
FHWA	Federal Highway Administration
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
FIP	Federal Information Processing System
FR	Federal Register
FRP	Forest Response Program
GDP	gross domestic product
GEMS	Graphical Exposure Modeling System
GM	geometric mean
GNP	Gross National Product
GSD	geometric standard deviation
H_2SO_4	sulfuric acid
ha	hectares
HAP	Hazardous Air Pollutant
HAPEM-MS	Hazardous Air Pollutant Exposure Model - Mobile Source
HNO_3	nitric acid
hp	horsepower
HTCM	Hedonic Travel-Cost Model
ICARUS	Investigation of Costs and Reliability in Utility Systems

ICD-9	International Classification of Diseases, Ninth Version (1975 Revision)
ICE	Industrial Combustion Emissions model
IEc	Industrial Economics, Incorporated
IEUBK	EPA's Integrated Exposure Uptake Biokinetic model
IMS	Integrated Model Set
IPF	iterative proportional fitting
IQ	intelligence quotient
ISCLT	Industrial Source Complex Long Term air quality model
J/W	Jorgenson / Wilcoxen
kg	kilograms
km	kilometers
lbs	pounds
LRI	lower respiratory illness
m/s	meters per second
m	meters
m^3	cubic meters
Mm	megameters
MMBTU	million BTU
MOBILE5a	EPA's mobile source emission factor model
mpg	miles per gallon
MRAD	minor restricted activity day
MSCET	Month and State Current Emission Trends
MTD	metric tons per day
MVATS	EPA's Motor Vehicle-Related Air Toxics Study
MVMA	Motor Vehicle Manufacturers Association
Mwe	megawatt equivalent
N	nitrogen
NA	not available
NAAQS	National Ambient Air Quality Standard
NAPAP	National Acid Precipitation Assessment Program
NARSTO	North American Research Strategy for Tropospheric Ozone
NATICH	National Air Toxics Information Clearinghouse
NCLAN	National Crop Loss Assessment Network
NEA	National Energy Accounts
NERA	National Economic Research Associates
NERC	North American Electric Reliability Council
NERL	EPA/ORD National Exposure Research Laboratory (new name for CERL)
NESHAP	National Emission Standard for Hazardous Air Pollutants
NHANES	First National Health and Nutrition Examination Survey
NHANES II	Second National Health and Nutrition Examination Survey
NIPA	National Income and Product Accounts
NMOCs	nonmethane organic compounds
NO	nitric oxide
NO_2	nitrogen dioxide
NO_3-	nitrate ion
NO_x	nitrogen oxides
NPTS	Nationwide Personal Transportation Survey
NSPS	New Source Performance Standards
NSWS	National Surface Water Survey
O&M	operating and maintenance
O_3	ozone

OAQPS	EPA/OAR Office of Air Quality Planning and Standards
OAR	EPA Office of Air and Radiation
OMS	EPA/OAR Office of Mobile Sources
OPAR	EPA/OAR Office of Policy Analysis and Review
OPPE	EPA Office of Policy Planning and Evaluation
ORD	EPA Office of Research and Development
OZIPM4	Ozone Isopleth Plotting with Optional Mechanism-IV
PACE	Pollution Abatement Costs and Expenditures survey
PAN	peroxyacetyl nitrate
PAPE	Pollution Abatement Plant and Equipment survey
Pb	lead
PbB	blood lead level
PCB	polychlorinated biphenyl
PES	Pacific Environmental Services
pH	the logarithm of the reciprocal of hydrogen ion concentration, a measure of acidity
PIC	product of incomplete combustion
PM_{10}	particulates less than or equal to 10 microns in aerometric diameter
$PM_{2.5}$	particulates less than or equal to 2.5 microns in aerometric diameter
POP	population
Pop_{mild}	exposed population of exercising mild asthmatics
Pop_{mod}	exposed population of exercising moderate asthmatics
ppb	parts per billion
PPH	people per household
pphm	parts per hundred million
ppm	parts per million
PPRG	Pooling Project Research Group
PRYL	percentage relative yield loss
PURHAPS	PURchased Heat And Power
PVC	polyvinyl chloride
r^2	statistical correlation coefficient, squared
RAD	restricted activity day
RADM	Regional Acid Deposition Model
RADM/EM	RADM Engineering Model
RAMC	Resource Allocation and Mine Costing model
RfD	reference dose
RIA	Regulatory Impact Analysis
ROM	Regional Oxidant Model
RRAD	respiratory restricted activity day
RUM	Random Utility Model
s.e.	standard error
SAB	Science Advisory Board
SAI	Systems Applications International
SAQM	SARMAP Air Quality Model
SARA	Superfund Amendment Reauthorization Act
SARMAP	SJVAQS/AUSPEX Regional Modeling Adaptation Project
SCC	Source Classification Code
SEDS	State Energy Data System
SIC	Standard Industrial Classification
SIP	State Implementation Plan
SJVAQS	San Joaquin Valley Air Quality Study
SMSA	Standard Metropolitan Statistical Area

SO_2	sulfur dioxide
SO_4^{2-}	sulfate ion
SOS/T	State of Science and Technology (refers to a series of NAPAP reports)
SRaw	Specific Airway Resistance
STAR	Stability Array weather database
TAMM90	Timber Assessment Market Model (revised version)
TEEMS	Transportation Energy and Emissions Modeling System
TIUS	Truck Inventory and Use Surveys
TRI	Toxic Release Inventory
TSP	total suspended particulate
U.S.	United States
UAM	Urban Airshed Model
URI	upper respiratory illness
USDA	United States Department of Agriculture
USEPA	United States Environmental Protection Agency
VC	vinyl chloride
VMT	vehicle miles traveled
VOC	volatile organic compounds
VOP	Vehicle Ownership Projection
VR	visual range
W126	index of peak weighted average of cumulative ozone concentrations
WLD	Work Loss Day
WTP	willingness to pay

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This report could not have been produced without the support of key administrative support staff. The project managers are grateful to Catrice Jefferson, Nona Smoke, Carolyn Hicks, Eunice Javis, Gloria Booker, Thelma Butler, Wanda Farrar, Ladonya Langston, Michelle Olawuyi, and Eileen Pritchard for their timely and tireless support on this project.

Executive Summary

Purpose of the Study

Throughout the history of the Clean Air Act, questions have been raised as to whether the health and environmental benefits of air pollution control justify the costs incurred by industry, taxpayers, and consumers. For the most part, questions about the costs and benefits of individual regulatory standards continue to be addressed during the regulatory development process through Regulatory Impact Analyses (RIAs) and other analyses which evaluate regulatory costs, benefits, and such issues as scope, stringency, and timing. There has never been, however, any comprehensive, long-term, scientifically valid and reliable study which answered the broader question:

> ***"How do the overall health, welfare, ecological, and economic benefits of Clean Air Act programs compare to the costs of these programs?"***

To address this void, Congress added to the 1990 Clean Air Act Amendments a requirement under section 812 that EPA conduct periodic, scientifically reviewed studies to assess the benefits and the costs of the Clean Air Act. Congress further required EPA to conduct the assessments to reflect central tendency, or "best estimate," assumptions rather than the conservative assumptions sometimes deemed appropriate for setting protective standards.

This report is the first in this ongoing series of Reports to Congress. By examining the benefits and costs of the 1970 and 1977 Amendments, this report addresses the question of the overall value of America's historical investment in cleaner air. The first Prospective Study, now in progress, will evaluate the benefits and costs of the 1990 Amendments.

Study Design

Estimates of the benefits and costs of the historical Clean Air Act are derived by examining the differences in economic, human health, and environmental outcomes under two alternative scenarios: a "control scenario" and a "no-control scenario." The control scenario reflects actual historical implementation of clean air programs and is based largely on historical data. The no-control scenario is a hypothetical scenario which reflects the assumption that no air pollution controls were established beyond those in place prior to enactment of the 1970 Amendments. Each of the two scenarios is evaluated by a sequence of economic, emissions, air quality, physical effect, economic valuation, and uncertainty models to measure the differences between the scenarios in economic, human health, and environmental outcomes. Details of this analytical sequence are presented in Chapter 1 and are summarized in Figure 1 of that chapter.

Study Review

EPA is required, under section 812, to consult both a panel of outside experts and the Departments of Labor and Commerce in designing and implementing the study.

The expert panel was organized in 1991 as the Advisory Council on Clean Air Act Compliance Analysis (hereafter "Council") under the auspices of EPA's Science Advisory Board (SAB). Organizing the external panel under the auspices of the SAB ensured that the peer review of the study would be conducted in a rigorous, objective, and publicly open manner. Eminent scholars and practitioners with expertise in economics, human health sciences, environmental sciences, and air quality modeling served on the Council and its technical subcommittees, and these reviewers met many times throughout the design and implementation phases of the study. During this ongoing, in-depth review, the Council provided valuable advice pertaining to the development and selection of data, selection of models and assumptions, evaluation and interpretation of the analytical findings, and characterization of those findings in several successive drafts of the Report to Congress. The present report was vastly improved as a result of the Council's rigorous and constructive review effort.

With respect to the interagency review process, EPA expanded the list of consulted agencies and convened a series of meetings during the design and early implementation phases from 1991 through late 1994. In late 1994, to ensure that all interested parties and the public received consistent information about remaining analytical issues and emerging results, EPA decided to use the public SAB review process as the primary forum for presenting and discussing issues and results. The Interagency Review Group was therefore discontinued as a separate process in late 1994.

A final, brief interagency review, pursuant to Circular A-19, was organized in August 1997 by the Office of Management and Budget and conducted following the completion of the extensive expert panel peer review by the SAB Council. During the course of the final interagency discussions, it became clear that several agencies held different views pertaining to several key assumptions in this study as well as to the best techniques to apply in the context of environmental program benefit-cost analyses, including the present study. The concerns include: (1) the extent to which air quality would have deteriorated from 1970 to 1990 in the absence of the Clean Air Act, (2) the methods used to estimate the number of premature deaths and illnesses avoided due to the CAA, (3) the methods used to estimate the value that individuals place on avoiding those risks, and (4) the methods used to value non-health related benefits. However, due to the court deadline the resulting concerns were not resolved during this final, brief interagency review. Therefore, this report reflects the findings of EPA and not necessarily other agencies in the Administration. Interagency discussion of some of these issues will continue in the context of the future prospective section 812 studies and potential regulatory actions.

Summary of Results

Direct Costs

To comply with the Clean Air Act, businesses, consumers, and government entities all incurred higher costs for many goods and services. The costs of providing goods and services to the economy were higher primarily due to requirements to install, operate, and maintain pollution abatement equipment. In addition, costs were incurred to design and implement regulations, monitor and report regulatory compliance, and invest in research and development. Ultimately, these higher costs of production were borne by stockholders, business owners, consumers, and taxpayers.

Figure ES-1 summarizes the historical data on Clean Air Act compliance costs by year, adjusted both for inflation and for the value of long-term investments in equipment. Further adjusting the direct costs incurred each year to reflect their equivalent worth in the year 1990, and then summing these annual results, yields an estimate of approximately $523 billion for the total value of 1970 to 1990 direct expenditures (see Appendix A for calculations).

Emissions

Emissions were substantially lower by 1990 under the control scenario than under the no-control scenario, as shown in Figure ES-2. Sulfur dioxide (SO_2) emissions were 40 percent lower, primarily due to utilities installing scrubbers and/or switching to lower sulfur fuels. Nitrogen oxides (NO_X) emissions were 30 percent lower by 1990, mostly because of the installation of catalytic converters on highway vehicles. Volatile organic compound (VOC) emissions were 45 percent lower and carbon monoxide (CO) emissions were 50 percent lower, also primarily due to motor vehicle controls.

For particulate matter, it is important to recognize the distinction between reductions in directly emitted particulate matter and reductions in ambient concentrations of particulate matter in the atmosphere. As discussed further in the next section, changes in particulate matter air quality depend both on changes in emissions of primary particles (i.e., air pollution which is already in solid particle form) and on changes in emissions of gaseous pollutants, such as sulfur dioxide and nitrogen oxides, which can be converted to particulate matter through chemical transformation in the atmosphere. Emissions of primary particulates

Figure ES-1. Total Estimated Direct Compliance Costs of the CAA (in billions of inflation-adjusted dollars).

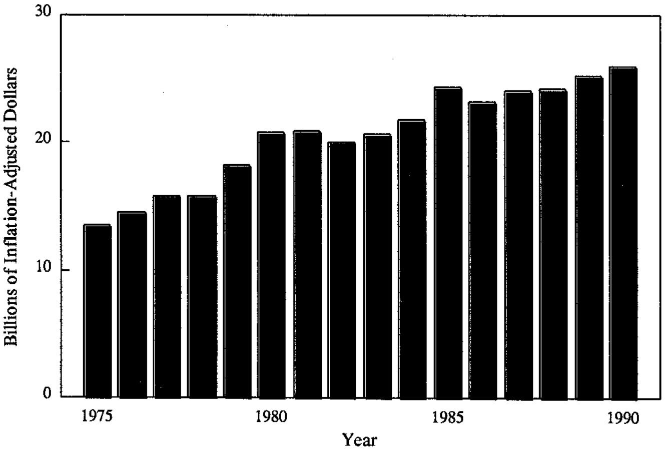
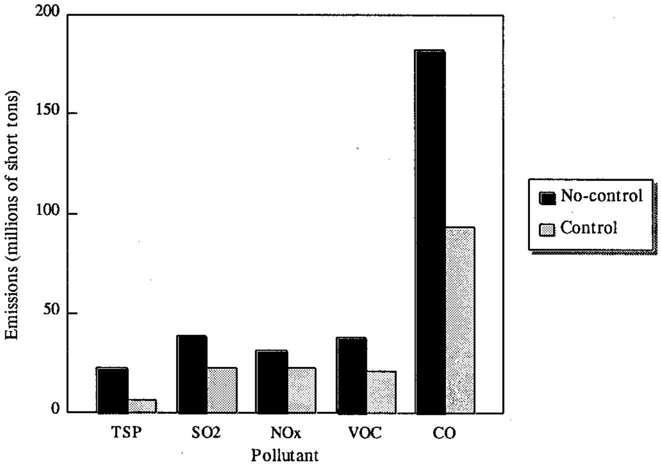


Figure ES-2. 1990 Control and No-control Scenario Emissions (in millions of short tons).



were 75 percent lower under the control scenario by 1990 than under the no-control scenario. This substantial difference is primarily due to vigorous efforts in the 1970s to reduce visible emissions from utility and industrial smokestacks.

Lead (Pb) emissions for 1990 are reduced by about 99 percent from a no-control level of 237,000 tons to about 3,000 tons under the control scenario.[1] The vast majority of the difference in lead emissions under the two scenarios is attributable to reductions in the use of leaded gasoline.

These reductions were achieved during a period in which population grew by 22.3 percent and the national economy grew by 70 percent.

Air Quality

The substantial reductions in air pollutant emissions achieved by the Clean Air Act translate into significantly improved air quality throughout the U.S. For sulfur dioxide, nitrogen oxides, and carbon monoxide, the improvements in air quality under the control scenario are assumed to be proportional to the estimated reduction in emissions. This is because, for these pollutants, changes in ambient concentrations in a particular area are strongly related to changes in emissions in that area. While the differences in control and no-control scenario air quality for each of these pollutants vary from place to place because of local variability in emissions reductions, by 1990 the national average improvements in air quality for these pollutants were: 40 percent reduction in sulfur dioxide, 30 percent reduction in nitrogen oxides, and 50 percent reduction in carbon monoxide.

Ground-level ozone is formed by the chemical reaction of certain airborne pollutants in the presence of sunlight. Reductions in ground-level ozone are therefore achieved through reductions in emissions of its precursor pollutants, particularly volatile organic compounds (VOCs) and nitrogen oxides (NO_X).[2] The differences in ambient ozone concentrations estimated under the control scenario vary significantly from one location to another, primarily because of local differences in the relative proportion of VOCs and NO_X, weather conditions, and specific precursor emissions reductions. On a national average basis, ozone concentrations in 1990 are about 15 percent lower under the control scenario. For several reasons, this overall reduction in ozone is significantly less than the 30 percent reduction in precursor NO_X and 45 percent reduction in precursor VOCs. First, significant natural (i.e., biogenic) sources of VOCs limit the level of ozone reduction achieved by reductions in man-made (i.e., anthropogenic) VOCs. Second, current knowledge of atmospheric photochemistry suggests that ozone reductions will tend to be proportionally smaller than reductions in precursor emissions. Finally, the plume model system used to estimate changes in urban ozone for this study is incapable of handling long-range transport of ozone from upwind areas and multi-day pollution events in a realistic manner.

There are many pollutants which contribute to ambient concentrations of particulate matter. The relative contributions of these individual pollutant species to ambient particulate matter concentrations vary from one region of the country to the next, and from urban areas to rural areas. The most important particle species, from a human health standpoint, may be the fine particles which can be respired deep into the lungs. While some fine particles are directly emitted by sources, the most important fine particle species are formed in the atmosphere through chemical conversion of gaseous pollutants. These species are referred to as secondary particles. The three most important secondary particles are (1) sulfates, which derive primarily from sulfur dioxide emissions; (2) nitrates, which derive primarily from nitrogen oxides emissions; and (3) organic aerosols, which can be directly emitted or can form from volatile organic com-

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[1] Results for lead are not shown in Figure ES-2 because the absolute levels of lead emissions are measured in thousands, not millions, of tons and will not be discernible on a graph of this scale.

[2] Ambient NOx concentrations are driven by anthropogenic emissions whereas ambient VOCs result from both anthropogenic and biogenic sources (e.g., terpenes emitted by trees).

Table ES-1. Criteria Pollutant Health Benefits — Estimated Distributions of 1990 Incidences of Avoided Health Effects (in thousands of incidences reduced) for 48 State Population. [1]

Endpoint	**Pollutant(s)**	**Affected Population**	**Annual Effects Avoided[2] (thousands)**			**Unit**
			5th %ile	**Mean**	**95th %ile**	
Premature Mortality	PM [3]	30 and over	112	184	257	cases
Premature Mortality	Lead	all	7	22	54	cases
Chronic Bronchitis	PM	all	498	674	886	cases
Lost IQ Points	Lead	children	7,440	10,400	13,000	points
IQ less than 70	Lead	children	31	45	60	cases
Hypertension	Lead	men 20-74	9,740	12,600	15,600	cases
Coronary Heart Disease	Lead	40-74	0	22	64	cases
Atherothrombotic brain infarction	Lead	40-74	0	4	15	cases
Initial cerebrovascular accident	Lead	40-74	0	6	19	cases
Hospital Admissions						
All Respiratory	PM & Ozone	all	75	89	103	cases
Chronic Obstructive Pulmonary Disease & Pneumonia	PM & Ozone	over 65	52	62	72	cases
Ischemic Heart Disease	PM	over 65	7	19	31	cases
Congestive Heart Failure	PM & CO	65 and over	28	39	50	cases
Other Respiratory-Related Ailments						
Shortness of breath, days	PM	children	14,800	68,000	133,000	days
Acute Bronchitis	PM	children	0	8,700	21,600	cases
Upper & Lower Respiratory Symptoms	PM	children	5,400	9,500	13,400	cases
Any of 19 Acute Symptoms	PM & Ozone	18-65	15,400	130,000	244,000	cases
Asthma Attacks	PM & Ozone	asthmatics	170	850	1,520	cases
Increase in Respiratory Illness	NO2	all	4,840	9,800	14,000	cases
Any Symptom	SO2	asthmatics	26	264	706	cases
Restricted Activity and Work Loss Days						
Minor Restricted Activity Days	PM & Ozone	18-65	107,000	125,000	143,000	days
Work Loss Days	PM	18-65	19,400	22,600	25,600	days

[1] The following additional human welfare effects were quantified directly in economic terms: household soiling damage, visibility impairment, decreased worker productivity, and agricultural yield changes.

[2] The 5th and 95th percentile outcomes represent the lower and upper bounds, respectively, of the 90 percent credible interval for each effect as estimated by uncertainty modeling. The mean is the arithmetic average of all estimates derived by the uncertainty modeling. See Chapter 7 and Appendix I for details.

[3] In this analysis, PM is used as a proxy pollutant for all non-Lead (Pb) criteria pollutants which may contribute to premature mortality. See Chapter 5 and Appendix D for additional discussion.

pound emissions. This highlights an important and unique feature of particulate matter as an ambient pollutant: more than any other pollutant, reductions in particulate matter are actually achieved through reductions in a wide variety of air pollutants. In other words, controlling particulate matter means controlling "air pollution" in a very broad sense. In the present analysis, reductions in sulfur dioxide, nitrogen oxides, volatile organic compounds, and directly-emitted primary particles achieved by the Clean Air Act result in a national average reduction in total suspended particulate matter of about 45 percent by 1990. For the smaller particles which are of greater concern from a health effects standpoint (i.e., PM_{10} and $PM_{2.5}$), the national average reductions were also about 45 percent.

Reductions in sulfur dioxide and nitrogen oxides also translate into reductions in formation, transport, and deposition of secondarily formed acidic compounds such as sulfate and nitric acid. These are the principal pollutants responsible for acid precipitation, or "acid rain." Under the control scenario, sulfur and nitrogen deposition are significantly lower by 1990 than under the no-control scenario throughout the 31 eastern states covered by EPA's Regional Acid Deposition Model (RADM). Percentage decreases in sulfur deposition range up to more than 40 percent in the upper Great Lakes and Florida-Southeast Atlantic Coast areas, primarily because the no-control scenario projects significant increases in the use of high-sulfur fuels by utilities in the upper Great Lakes and Gulf

Coast states. Nitrogen deposition is also significantly lower under the control scenario, with percentage decreases reaching levels of 25 percent or higher along the Eastern Seaboard, primarily due to higher projected emissions of motor vehicle nitrogen oxides under the no-control scenario.

Finally, decreases in ambient concentrations of light-scattering pollutants, such as sulfates and nitrates, are estimated to lead to perceptible improvements in visibility throughout the eastern states and southwestern urban areas modeled for this study.

Physical Effects

The lower ambient concentrations of sulfur dioxide, nitrogen oxides, particulate matter, carbon monoxide, ozone and lead under the control scenario yield a substantial variety of human health, welfare and ecological benefits. For a number of these benefit categories, quantitative functions are available from the scientific literature which allow estimation of the reduction in incidence of adverse effects. Examples of these categories include the human mortality and morbidity effects of a number of pollutants, the neurobehavioral effects among children caused by exposure to lead, visibility impairment, and effects on yields for some agricultural products.

A number of benefit categories, however, can not be quantified and/or monetized for a variety of reasons. In some cases, substantial scientific uncertainties prevail regarding the existence and magnitude of adverse effects (e.g., the contribution of ozone to air pollution-related mortality). In other cases, strong scientific evidence of an effect exists, but data are still too limited to support quantitative estimates of incidence reduction (e.g., changes in lung function associated with long-term exposure to ozone). Finally, there are effects for which there is sufficient information to estimate incidence reduction, but for which there are no available economic value measures; thus reductions in adverse effects cannot be expressed in monetary terms. Examples of this last category include relatively small pulmonary function decrements caused by acute exposures to ozone and reduced time to onset of angina pain caused by carbon monoxide exposure.

Table ES-1 provides a summary of the key differences in quantified human health outcomes estimated under the control and no-control scenarios. Results are presented as thousands of cases avoided in 1990 due to control of the pollutants listed in the table and reflect reductions estimated for the entire U.S. population living in the 48 continental states. Epidemiological research alone cannot prove whether a cause-effect relationship exists between an individual

Table ES-2. Major Nonmonetized, Adverse Effects Reduced by the Clean Air Act.

Pollutant	Nonmonetized Adverse Effects
Particulate Matter	Large Changes in Pulmonary Function Other Chronic Respiratory Diseases Inflammation of the Lung Chronic Asthma and Bronchitis
Ozone	Changes in Pulmonary Function Increased Airway Responsiveness to Stimuli Centroacinar Fibrosis Inflammation of the Lung Immunological Changes Chronic Respiratory Diseases Extrapulmonary Effects (i.e., other organ systems) Forest and other Ecological Effects Materials Damage
Carbon Monoxide	Decreased Time to Onset of Angina Behavioral Effects Other Cardiovascular Effects Developmental Effects
Sulfur Dioxide	Respiratory Symptoms in Non-Asthmatics Hospital Admissions Agricultural Effects Materials Damage Ecological Effects
Nitrogen Oxides	Increased Airway Responsiveness to Stimuli Decreased Pulmonary Function Inflammation of the Lung Immunological Changes Eye Irritation Materials Damage Eutrophication (e.g., Chesapeake Bay) Acid Deposition
Lead	Cardiovascular Diseases Reproductive Effects in Women Other Neurobehavioral, Physiological Effects in Children Developmental Effects from Maternal Exposure, inc IQ Loss [/1] Ecological Effects
Air Toxics	All Human Health Effects Ecological Effects

[/1] IQ loss from direct, as opposed to maternal, exposure is quantified and monetized. See Tables ES-1 And ES-3.

pollutant and an observed health effect. Although not universally accepted, this study uses the epidemiological findings about correlations between pollution and observed health effects to estimate changes in the number of health effects that would occur if pollution levels change. A range is presented along with the mean estimate for each effect, reflecting uncertainties which have been quantified in the underlying health effects literature.

Adverse human health effects of the Clean Air Act "criteria pollutants" sulfur dioxide, nitrogen oxides, ozone, particulate matter, carbon monoxide, and lead dominate the quantitative estimates in part because, although there are important residual uncertainties, evidence of physical consequences is greatest for these pollutants. The Clean Air Act yielded other benefits, however, which are important even though they are uncertain and/or difficult to quantify. These other benefit categories include (a) all benefits accruing from reductions in hazardous air pollutants (also referred to as air toxics), (b) reductions in damage to cultural resources, buildings, and other materials, (c) reductions in adverse effects on wetland, forest, and aquatic ecosystems, and (d) a variety of additional human health and welfare effects of criteria pollutants. A more complete list of these nonmonetized effects is presented in Table ES-2.

In addition to controlling the six criteria pollutants, the 1970 and 1977 Clean Air Act Amendments led to reductions in ambient concentrations of a small number of hazardous air pollutants. Although they are not fully quantified in this report, control of these pollutants resulted both from regulatory standards set specifically to control hazardous air pollutants and from incidental reductions achieved through programs aimed at controlling criteria pollutants.

Existing scientific research suggests that reductions in both hazardous air pollutants and criteria pollutants yielded widespread improvements in the functioning and quality of aquatic and terrestrial ecosystems. In addition to any intrinsic value to be attributed to these ecological systems, human welfare is enhanced through improvements in a variety of ecological services. For example, protection of freshwater ecosystems achieved through reductions in deposition of acidic air pollutants may improve commercial and recreational fishing. Other potential ecological benefits of reduced acid deposition include improved wildlife viewing, maintenance of biodiversity, and nutrient cycling. Increased growth and productivity of U.S. forests may have resulted from reduc-

Table ES-3. Central Estimates of Economic Value per Unit of Avoided Effect (in 1990 dollars).

Endpoint	Pollutant	Valuation (mean est.)	
Mortality	PM & Lead	$4,800,000	per case [1]
Chronic Bronchitis	PM	$260,000	per case
IQ Changes			
Lost IQ Points	Lead	$3,000	per IQ point
IQ less than 70	Lead	$42,000	per case
Hypertension	Lead	$680	per case
Strokes [2]	Lead	$200,000 $150,000	per case-males[3] per case-females[3]
Coronary Heart Disease	Lead	$52,000	per case
Hospital Admissions			
Ischemic Heart Disease	PM	$10,300	per case
Congestive Heart Failure	PM	$8,300	per case
COPD	PM & Ozone	$8,100	per case
Pneumonia	PM & Ozone	$7,900	per case
All Respiratory	PM & Ozone	$6,100	per case
Respiratory Illness and Symptoms			
Acute Bronchitis	PM	$45	per case
Acute Asthma	PM & Ozone	$32	per case
Acute Respiratory Symptoms	PM, Ozone, NO_2, SO_2	$18	per case
Upper Respiratory Symptoms	PM	$19	per case
Lower Respiratory Symptoms	PM	$12	per case
Shortness of Breath	PM	$5.30	per day
Work Loss Days	PM	$83	per day
Mild Restricted Activity Days	PM & Ozone	$38	per day
Welfare Benefits			
Visibility	DeciView	$14	per unit change in DeciView
Household Soiling	PM	$2.50	per household per PM-10 change
Decreased Worker Productivity	Ozone	$1	[4]
Agriculture (Net Surplus)	Ozone	Change in Economic Surplus	

[1] Alternative results, based on assigning a value of $293,000 for each life-year lost are presented on pg. ES-9.

[2] Strokes are comprised of atherothrombotic brain infarctions and cerebrovascular accidents; both are estimated to have the same monetary value.

[3] The different valuations for stroke cases reflect differences in lost earnings between males and females. See Appendix G for a more complete discussion of valuing reductions in strokes.

[4] Decreased productivity valued as change in daily wages: $1 per worker per 10% decrease in ozone.

tions in ground-level ozone. More vigorous forest ecosystems in turn yield a variety of benefits, including increased timber production; improved forest aesthetics for people enjoying outdoor activities such as hunting, fishing, and camping; and improvements in ecological services such as nutrient cycling and temporary sequestration of global warming gases. These improvements in ecological structure and function have not been quantified in this assessment.

Economic Valuation

Estimating the reduced incidence of physical effects provides a valuable measure of health benefits for individual endpoints. However, to compare or aggregate benefits across endpoints, the benefits must be monetized. Assigning a monetary value to avoided incidences of each effect permits a summation, in terms of dollars, of monetized benefits realized as a result of the Clean Air Act, and allows that summation to be compared to the cost of the Clean Air Act.

Before proceeding through this step, it is important to recognize the substantial controversies and uncertainties which pervade attempts to characterize adverse human health and ecological effects of pollution in dollar terms. To many, dollar-based estimates of the value of avoiding outcomes such as loss of human life, pain and suffering, or ecological degradation do not capture the full and true value to society as a whole of avoiding or reducing these effects. Adherents to this view tend to favor assessment procedures which (a) adopt the most technically defensible dollar-based valuation estimates for analytical purposes but (b) leave the moral dimensions of policy evaluation to those who must decide whether, and how, to use cost-benefit results in making public policy decisions. This is the paradigm adopted in the present study. Given the Congressional mandate to perform a cost-benefit study of the Clean Air Act, the Project Team has endeavored to apply widely-recognized, customary techniques of Applied Economics to perform this cost-benefit analysis. However, EPA believes there are social and personal values furthered by the Clean Air Act which have not been effectively captured by the dollar-based measures used in this study. Therefore, EPA strongly encourages readers to look beyond the dollar-based comparison of costs and benefits of the Clean Air Act and consider the broader value of the reductions in adverse health and environmental effects which have been achieved as well as any additional adverse consequences of regulation which may not be reflected in the cost estimates reported herein.

For this study, unit valuation estimates are derived from the economics literature and reported in dollars per case (or, in some cases, episode or symptom-day) avoided for health effects and dollars per unit of

Table ES-4. Total Estimated Monetized Benefits by Endpoint Category for 48 State Population for 1970 to 1990 Period (in billions of 1990 dollars).

		Present Value		
Endpoint	**Pollutant(s)**	**5th %ile**	**Mean**	**95th %ile**
Mortality	PM	$2,369	$16,632	$40,597
Mortality	Lead	$121	$1,339	$3,910
Chronic Bronchitis	PM	$409	$3,313	$10,401
IQ (Lost IQ Pts. + Children w/ IQ<70)	Lead	$271	$399	$551
Hypertension	Lead	$77	$98	$120
Hospital Admissions	PM, Ozone, Lead, & CO	$27	$57	$120
Respiratory-Related Symptoms, Restricted Activity, & Decreased Productivity	PM, Ozone, NO2, & SO2	$123	$182	$261
Soiling Damage	PM	$6	$74	$192
Visibility	particulates	$38	$54	$71
Agriculture (Net Surplus)	Ozone	$11	$23	$35

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[3] All of these summary results are present values of the 1970 to 1990 streams of benefits and costs, discounted at five percent.

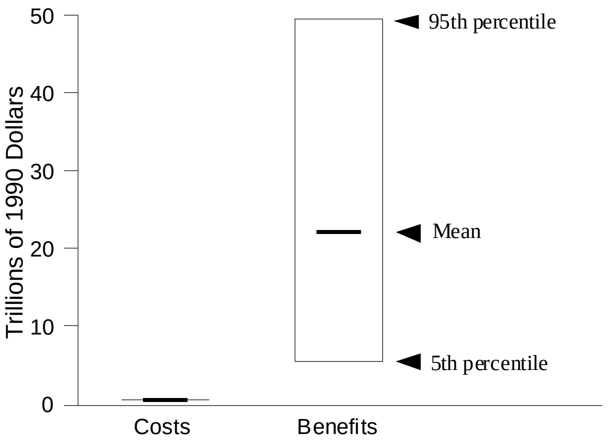
avoided damage for human welfare effects. Similar to estimates of physical effects provided by health studies, each of the monetary values of benefits applied in this analysis can be expressed in terms of a mean value and a range around the mean estimate. This range reflects the uncertainty in the economic valuation literature associated with a given effect. These value ranges, and the approaches used to derive them, are described in Chapter 6 and Appendix I for each of the effects monetized in this study. The mean values of these ranges are shown in Table ES-3.

Monetized Benefits and Costs

The total monetized economic benefit attributable to the Clean Air Act is derived by applying the unit values (or ranges of values) to the stream of monetizable physical effects estimated for the 1970 to 1990 period. In developing these estimates, steps are taken to avoid double-counting of benefits. In addition, a computer simulation model is used to estimate ranges of plausible outcomes for the benefits estimates reflecting uncertainties in the physical effects and economic valuation literature (see Chapter 7 and Appendix I for details).

The economic benefit estimation model then generated a range of economic values for the differences in physical outcomes under the control and no-control scenarios for the target years of the benefits analysis: 1975, 1980, 1985, and 1990. Linear interpolation between these target years is used to estimate benefits in intervening years. These yearly results are then adjusted to their equivalent value in the year 1990 and summed to yield a range and mean estimate for the total monetized benefits of the Clean Air Act from 1970 to 1990. These results are summarized in Table ES-4.

Figure ES-3. Total Estimated Direct Compliance Costs of the CAA (in trillions of inflation-adjusted dollars).



Combining these benefits results with the cost estimates presented earlier yields the following analytical outcomes.[3]

- **The total monetized benefits of the Clean Air Act realized during the period from 1970 to 1990 range from 5.6 to 49.4 trillion dollars, with a central estimate of 22.2 trillion dollars.**

- **By comparison, the value of direct compliance expenditures over the same period equals approximately 0.5 trillion dollars.**

- **Subtracting costs from benefits results in net, direct, monetized benefits ranging from 5.1 to 48.9 trillion dollars, with a central estimate of 21.7 trillion dollars, for the 1970 to 1990 period.**

- **The lower bound of this range may go down and the upper bound may go up if analytical uncertainties associated with compliance costs, macroeconomic effects, emissions projections, and air quality modeling could be quantified and incorporated in the uncertainty analysis. While the range already reflects many important uncertainties in the physical effects and economic valuation steps, the range might also broaden further if additional uncertainties in these two steps could be quantified.**

- **The central estimate of 22.2 trillion dollars in benefits may be a significant underestimate due to the exclusion of large numbers of benefits from the monetized benefit estimate (e.g., all air toxics effects, ecosystem effects, numerous human health effects).**

Figure ES-3 provides a graphical representation of the estimated range of total monetized benefits and compares this range to estimated direct compliance costs. Clearly, even the lower bound estimate of monetized benefits substantially exceeds the costs of the historical Clean Air Act. As shown by the yearly data presented in Chapter 7, monetized benefits consistently and substantially exceeded costs throughout the 1970 to 1990 period.

Table ES-5. Alternative Mortality Benefits Mean Estimates for 1970 to 1990 (in trillions of 1990 dollars) Compared to Total 1970 to 1990 Compliance Costs.

Benefit Estimation Method	Mortality Benefits (trillions of dollars) PM	 PM+Pb
Statistical life method ($4.8M/case)	16.6	18.0
Life-years lost method ($293,000/year)	9.1	10.1
Total compliance cost	---	0.5

Alternative Results

The primary results of this analysis, including aggregate cost and benefit estimates which reflect many elements of the uncertainty associated with them, are presented above. However, some additional analysis is required to address an important issue raised by the EPA Science Advisory Board Council on Clean Air Act Compliance Analysis (a.k.a. Council) charged with reviewing the present study. Specifically, the Council believes it is appropriate to also display alternative premature mortality results based on an approach which estimates, and assigns a value to, the loss of life-years (i.e., the reduction in years of remaining life expectancy) resulting from the pollution exposure. The Council's position is based on the conclusion that older individuals are more susceptible to air pollution-induced mortality. EPA believes, however, that the simplifying assumptions which must be adopted to implement a life-years lost approach render its results less reliable, even for the purposes of economic efficiency analysis, than a value of statistical life approach. In addition, EPA is concerned about any analytical methodology which may be interpreted to justify conferring less environmental protection on particular individuals or groups of individuals (e.g., the elderly and/or sick). EPA therefore prefers at this time to continue with its current practice of assigning the same economic value to incidences of premature mortality regardless of the age and health status of those affected, and the primary results presented above reflect this view. Nevertheless, complete alternative results based on a value of statistical life-years lost (VSLY) approach are presented in Chapter 7 and Appendix I and are summarized below.

Table ES-5 summarizes and compares the results of the mortality benefits estimates based on the value of statistical life (VSL) and VSLY approaches. Estimated 1970 to 1990 benefits from PM-related mortality alone and total mortality (i.e., PM plus Lead) benefits are reported, along with total compliance costs for the same period. Adding the VSLY-based mortality benefits estimates to the non-mortality benefits estimates from Table ES-4 yields the following results for the overall analysis.

- ***Alternate Result:* The total monetized benefits of the Clean Air Act realized during the period from 1970 to 1990 range from 4.8 to 28.7 trillion dollars, with a central estimate of 14.3 trillion dollars.**

- ***Alternate Result:* Subtracting costs from benefits results in net, direct, monetized benefits ranging from 4.3 to 28.2 trillion dollars, with a central estimate of 13.7 trillion dollars, for the 1970 to 1990 period.**

The results indicate that the choice of valuation methodology significantly affects the estimated monetized value of historical reductions in air pollution-related premature mortality. However, the downward adjustment which would result from applying a VSLY approach in lieu of a VSL approach does not change the basic outcome of this study, viz. the estimated monetized benefits of the historical Clean Air Act substantially exceed the estimated historical costs of compliance.

Conclusions and Future Directions

First and foremost, these results indicate that the benefits of the Clean Air Act and associated control programs substantially exceeded costs. Even considering the large number of important uncertainties permeating each step of the analysis, it is extremely unlikely that the converse could be true.

A second important implication of this study is that a large proportion of the monetized benefits of the historical Clean Air Act derive from reducing two pollutants: lead and particulate matter[4] (see Table ES-4). Some may argue that, while programs to control these two pollutants may have yielded measurable benefits in excess of measurable costs, estimates of measurable benefits of many other historical Clean Air Act programs and standards considered in isolation might not have exceeded measurable costs. While this may or may not be true, this analysis provides no evidence to support or reject such conjectures. On the cost side, the historical expenditure data used in this analysis are not structured in ways which allow attribution of control costs to specific programs or standards. On the benefit side, most control programs yielded a variety of benefits, many of which included reductions in other pollutants such as ambient particulate matter. For example, new source performance standards for sulfur dioxide emissions from coal-fired utility plants yielded benefits beyond those associated with reducing exposures to gaseous sulfur dioxide. The reductions in sulfur dioxide emissions also led to reductions in ambient fine particle sulfates, yielding human health, ecological, and visibility benefits.

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[4] Ambient particulate matter results from emissions of a wide array of precursor pollutants, including sulfur dioxide, nitrogen oxides, and organic compounds.

This retrospective study highlights important areas of uncertainty associated with many of the monetized benefits included in the quantitative analysis and lists benefit categories which could not be quantified or monetized given the current state of the science. Additional research in these areas may reduce critical uncertainties and/or improve the comprehensiveness of future assessments. Particularly important areas where further research might reduce critical uncertainties include particulate matter-related mortality incidence, valuation of premature mortality, and valuation of particulate-related chronic bronchitis and cardiovascular disease. Additional research on hazardous air pollutants and on air pollution-related changes in ecosystem structure and function might help improve the comprehensiveness of future benefit studies. (See Appendix J for further discussion.)

Finally, the results of this retrospective study provide useful lessons with respect to the value and the limitations of cost-benefit analysis as a tool for evaluating environmental programs. Cost-benefit analysis can provide a valuable framework for organizing and evaluating information on the effects of environmental programs. When used properly, cost-benefit analysis can help illuminate important effects of changes in policy and can help set priorities for closing information gaps and reducing uncertainty. Such proper use, however, requires that sufficient levels of time and resources be provided to permit careful, thorough, and technically and scientifically sound data-gathering and analysis. When cost-benefit analyses are presented without effective characterization of the uncertainties associated with the results, cost-benefit studies can be used in highly misleading and damaging ways. Given the substantial uncertainties which permeate cost-benefit assessment of environmental programs, as demonstrated by the broad range of estimated benefits presented in this study, cost-benefit analysis is best used to inform, but not dictate, decisions related to environmental protection policies, programs, and research.

1

Introduction

Background and Purpose

As part of the Clean Air Act Amendments of 1990, Congress established a requirement under section 812 that EPA develop periodic Reports to Congress estimating the benefits and costs of the Clean Air Act itself. The first such report was to be a retrospective analysis, with a series of prospective analyses to follow every two years thereafter. This report represents the retrospective study, covering the period beginning with passage of the Clean Air Act Amendments of 1970, until 1990 when Congress enacted the most recent comprehensive amendments to the Act.

Since the legislative history associated with section 812 is sparse, there is considerable uncertainty regarding Congressional intent behind the requirement for periodic cost-benefit evaluations of the Clean Air Act (CAA). However, EPA believes the principal goal of these amendments was that EPA should develop, and periodically exercise, the ability to provide Congress and the public with up-to-date, comprehensive information about the economic costs, economic benefits, and health, welfare, and ecological effects of CAA programs. The results of such analyses might then provide useful information for refinement of CAA programs during future reauthorizations of the Act.

The retrospective analysis presented in this Report to Congress has been designed to provide an unprecedented examination of the overall costs and benefits of the historical Clean Air Act. Many other analyses have attempted to identify the isolated effects of individual standards or programs, but no analysis with the present degree of validity, breadth and integration has ever been successfully developed. Despite data limitations, considerable scientific uncertainties, and severe resource constraints; the EPA Project Team was able to develop a broad assessment of the costs and benefits associated with the major CAA programs of the 1970 to 1990 period. Beyond the statutory goals of section 812, EPA intends to use the results of this study to help support decisions on future investments in air pollution research. Finally, many of the methodologies and modeling systems developed for the retrospective study may be applied in the future to the ongoing series of section 812 prospective studies.

Clean Air Act Requirements, 1970 to 1990

The Clean Air Act establishes a framework for the attainment and maintenance of clean and healthful air quality levels. The Clean Air Act was enacted in 1970 and amended twice — in 1977 and most recently in 1990. The 1970 Clean Air Act contained a number of key provisions. First, EPA was directed to establish national ambient air quality standards for the major criteria air pollutants. The states were required to develop implementation plans describing how they would control emission limits from individual sources to meet and maintain the national standards. Second, the 1970 CAA contained deadlines and strengthened enforcement of emission limitations and state plans with measures involving both the states and the federal government. Third, the 1970 Act forced new sources to meet standards based on the best available technology. Finally, the Clean Air Act of 1970 addressed hazardous pollutants and automobile exhausts.

The 1977 Clean Air Act Amendments also set new requirements on clean areas already in attainment with the national ambient air quality standards. In addition, the 1977 Amendments set out provisions to help areas that failed to comply with deadlines for achievement of the national ambient air quality standards. For example, permits for new major sources and modifications were required.

The 1990 Clean Air Act Amendments considerably strengthened the earlier versions of the Act. With respect to nonattainment, the Act set forth a detailed and graduated program, reflecting the fact that problems in some areas are more difficult and complex than others. The 1990 Act also established a list of 189 regulated hazardous air pollutants and a multistep program for controlling emissions of these toxic air pollutants. Significant control programs were also established for emissions of acid rain precursors and stratospheric ozone-depleting chemicals. The biggest regulatory procedural change in the Act is the new permit program where all major sources are now required to obtain an operating permit. Finally, the amendments considerably expanded the enforcement provisions of the Clean Air Act, adding administrative penalties and increasing potential civil penalties.

Section 812 of the Clean Air Act Amendments of 1990

Section 812 of the Clean Air Act Amendments of 1990 requires the EPA to perform a "retrospective" analysis which assesses the costs and benefits to the public health, economy and the environment of clean air legislation enacted prior to the 1990 amendments. Section 812 directs that EPA shall measure the effects on "employment, productivity, cost of living, economic growth, and the overall economy of the United States" of the Clean Air Act. Section 812 also requires that EPA consider all of the economic, public health, and environmental benefits of efforts to comply with air pollution standards. Finally, section 812 requires EPA to evaluate the prospective costs and benefits of the Clean Air Act every two years.

Analytical Design and Review

Target Variable

The retrospective analysis was designed to answer the following question:

> ***"How do the overall health, welfare, ecological, and economic benefits of Clean Air Act programs compare to the costs of these programs?"***

By examining the overall effects of the Clean Air Act, this analysis complements the Regulatory Impact Analyses (RIAs) developed by EPA over the years to evaluate individual regulations. Resources were used more efficiently by recognizing that these RIAs, and other EPA analyses, provide complete information about the costs and benefits of specific rules. Furthermore, in addition to the fact that the RIAs already provide rule-specific benefit and cost estimates, the broad-scale approach adopted in the present study precludes reliable re-estimation of the benefits and costs of individual standards or programs. On the cost side, this study relies on aggregated compliance expenditure data from existing surveys. Unfortunately, these data do not support reliable allocation of total costs incurred to specific emissions reductions for the various pollutants emitted from individual facilities. Therefore, it is infeasible in the context of this study to assign costs to specific changes in emissions. Further complications emerge on the benefit side. To estimate benefits, this study calculates the change in incidences of adverse effects implied by changes in ambient concentrations of air pollutants. However, reductions achieved in emitted pollutants contribute to changes in ambient concentrations of those, or secondarily formed, pollutants in ways which are highly complex, interactive, and often nonlinear. Therefore, even if costs could be reliably matched to changes in emissions, benefits cannot be reliably matched to changes in emissions because of the complex, nonlinear relationships between emissions and the changes in ambient concentrations which are used to estimate benefits.

Focusing on the broader target variables of "overall costs" and "overall benefits" of the Clean Air Act, the EPA Project Team adopted an approach based on construction and comparison of two distinct scenarios: a "no-control scenario" and a "control scenario." The no-control scenario essentially freezes federal, state, and local air pollution controls at the levels of stringency and effectiveness which prevailed in 1970. The control scenario assumes that all federal, state, and local rules promulgated pursuant to, or in support of, the CAA during 1970 to 1990 were implemented. This analysis then estimates the differences between the economic and environmental outcomes associated with these two scenarios. For more information on the scenarios and their relationship to historical trends, see Appendix B.

Key Assumptions

Two key assumptions were made during the scenario design process to avoid miring the analytical process in endless speculation. First, the "no-control" scenario was defined to reflect the assumption that no additional air pollution controls were imposed by any level of government or voluntarily initiated by private entities after 1970. Second, it is assumed that the geographic distribution of population and economic activity remains the same between the two scenarios.

The first assumption is an obvious oversimplification. In the absence of the CAA, one would expect to see some air pollution abatement activity, either voluntary or due to state or local regulations. It is conceivable that state and local regulation would have required air pollution abatement equal to—or even greater than—that required by the CAA; particularly since some states, most notably California, have done so. If one were to assume that state and local regulations would have been equivalent to CAA standards, then a cost-benefit analysis of the CAA would be a meaningless exercise since both costs and benefits would equal zero. Any attempt to predict how state and local regulations would have differed from the CAA would be too speculative to support the credibility of the ensuing analysis. Instead, the no-control scenario has been structured to reflect the assumption that states and localities would not have invested further in air pollution control programs after 1970 in the absence of the federal CAA. That is, this analysis accounts for the costs and benefits of all air pollution control from 1970 to 1990. Speculation about the precise fraction of costs and benefits attributable exclusively to the federal CAA is left to others. Nevertheless, it is important to note that state and local governments and private initiatives are responsible for a significant portion of these total costs and total benefits. At the same time, it must also be acknowledged that the federal CAA played an essential role in achieving these results by helping minimize the advent of pollution havens[1], establishing greater incentives for pollution control research and development than individual state or local rules could provide; organizing and promoting health and environmental research, technology transfer and other information management and dissemination services; addressing critical interstate air pollution problems, including the regional fine particle pollution which is responsible for much of the estimated monetary benefit of historical air pollution control; providing financial resources to state and local government programs; and many other services. In the end, however, the benefits of historical air pollution controls were achieved through partnerships among all levels of government and with the active participation and cooperation of private entities and individuals.

The second assumption concerns changing demographic patterns in response to air pollution. In the hypothetical no-control world, air quality is worse than that in the historical "control" world particularly in urban industrial areas. It is possible that in the no-control case more people, relative to the control case, would move away from the most heavily polluted areas. Rather than speculate on the scale of population movement, the analysis assumes no differences in demographic patterns between the two scenarios. Similarly, the analysis assumes no changes in the spatial pattern of economic activity. For example: if, in the no-control case, an industry is expected to produce greater output than it did in the control case, that increased output is produced by actual historical plants, avoiding the need to speculate about the location or other characteristics of new plants providing additional productive capacity.

Analytic Sequence

The analysis was designed and implemented in a sequential manner following seven basic steps which are summarized below and described in detail later in this report. The seven major steps were:

- direct cost estimation
- macroeconomic modeling
- emissions modeling
- air quality modeling
- health and environmental effects estimation
- economic valuation
- results aggregation and uncertainty characterization

By necessity, these components had to be completed sequentially. The emissions modeling effort had to be completed entirely before the air quality models could be configured and run; the air quality modeling results had to be completed before the health and environmental consequences of air quality changes could be derived; and so on. The analytical sequence, and the modeled versus actual data basis for each analytical component, are summarized in Figure 1 and described in the remainder of this section.

The first step of the analysis was to estimate the total direct costs incurred by public and private entities to comply with post-1970 CAA requirements. These data were obtained directly from Census Bureau and Bureau of Economic Analysis (BEA) data on compliance expenditures reported by sources, and from EPA analyses. These direct cost data were then adopted as inputs to the macroeconomic model used to project economic conditions–such as production levels, prices, employment patterns, and other economic indicators–under the two scenarios. To ensure a consistent basis for scenario comparison, the analysis applied the same macroeconomic modeling system to estimate control and no-control scenario economic conditions.[2] First, a control scenario was constructed by running the macroeconomic model using actual historical data for input factors such as economic growth rates during the 1970 to 1990 period. The model was then re-run for the no-control scenario by, in essence, returning all post-1970 CAA compliance expenditures to the economy. With these additional resources available for capital formation, personal consumption, and other purposes, overall economic conditions under the no-control scenario differed from those of the control scenario. In addition to providing estimates of the difference in overall economic growth and other outcomes under the two scenarios, these first two analytical steps were used to define specific economic conditions used as inputs to the emissions modeling effort, the first step in the estimation of CAA benefits.[3]

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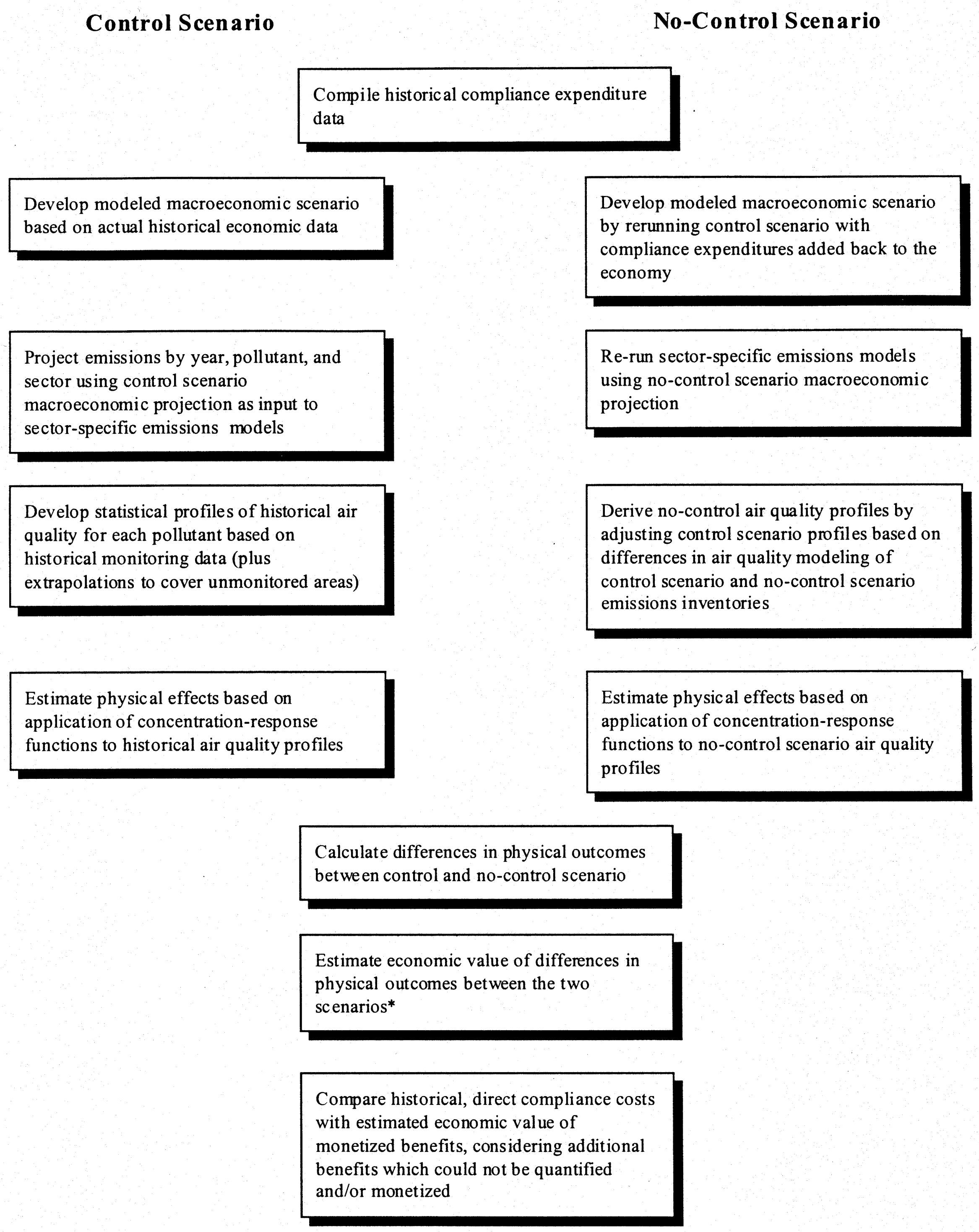
[1] "Pollution havens" is a term used to identify individual states or localities which permit comparatively high levels of pollution in order to attract and hold polluting industries and other activities.

[2] Using modeled economic conditions for both scenarios has both advantages and disadvantages. The principal disadvantage is that historical economic conditions "predicted" by a macroeconomic model will not precisely duplicate actual historical events and conditions. However, this disadvantage is outweighed by the avoidance of distortions and biases which would result from comparing a modeled no-control scenario with actual historical conditions. By using the same macroeconomic model for both scenarios, model errors and biases essentially cancel out, yielding more robust estimates of scenario differences, which are what this analysis seeks to evaluate.

[3] For example, the macroeconomic model projected different electricity sales levels under the two scenarios, and these sales levels were used as key input assumptions by the utility sector emissions model.

Figure 1. Summary of Analytical Sequence and Modeled versus Historical Data Basis.



Control Scenario

No-Control Scenario

Compile historical compliance expenditure data

Develop modeled macroeconomic scenario based on actual historical economic data

Develop modeled macroeconomic scenario by rerunning control scenario with compliance expenditures added back to the economy

Project emissions by year, pollutant, and sector using control scenario macroeconomic projection as input to sector-specific emissions models

Re-run sector-specific emissions models using no-control scenario macroeconomic projection

Develop statistical profiles of historical air quality for each pollutant based on historical monitoring data (plus extrapolations to cover unmonitored areas)

Derive no-control air quality profiles by adjusting control scenario profiles based on differences in air quality modeling of control scenario and no-control scenario emissions inventories

Estimate physical effects based on application of concentration-response functions to historical air quality profiles

Estimate physical effects based on application of concentration-response functions to no-control scenario air quality profiles

Calculate differences in physical outcomes between control and no-control scenario

Estimate economic value of differences in physical outcomes between the two scenarios*

Compare historical, direct compliance costs with estimated economic value of monetized benefits, considering additional benefits which could not be quantified and/or monetized

* In some cases, economic value is derived directly from physical effects modeling (e.g., agricultural yield loss).

Using appropriate economic indicators from the macroeconomic model results as inputs, a variety of emissions models were run to estimate emissions levels under the two scenarios. These emissions models provided estimates of emissions of six major pollutants[4] from each of six key emitting sectors: utilities, industrial processes, industrial combustion, on-highway vehicles, off-highway vehicles, and commercial/residential sources. The resulting emissions profiles reflect state-wide total emissions from each pollutant-sector combination for the years 1975, 1980, 1985, and 1990.[5]

The next step toward estimation of benefits involved translating these emissions inventories into estimates of air quality conditions under each scenario. Given the complexity, data requirements, and operating costs of state-of-the-art air quality models–and the afore-mentioned resource constraints–the EPA Project Team adopted simplified, linear scaling approaches for a number of pollutants. However, for ozone and other pollutants or air quality conditions which involve substantial non-linear formation effects and/or long-range atmospheric transport and transformation, the EPA Project Team invested the time and resources needed to use more sophisticated modeling systems. For example, urban area-specific ozone modeling was conducted for 147 urban areas throughout the 48 contiguous states.

Up to this point of the analysis, both the control and no-control scenario were based on modeled conditions and outcomes. However, at the air quality modeling step, the analysis returned to a foundation based on actual historical conditions and data. Specifically, actual historical air quality monitoring data from 1970 to 1990 were used to define the control scenario. Air quality conditions under the no-control scenario were then derived by scaling the historical data adopted for the control scenario by the ratio of the modeled control and no-control scenario air quality. This approach took advantage of the richness of the historical data on air quality, provided a realistic grounding for the benefit measures, and yet retained the analytical consistency conferred by using the same modeling approach for both scenarios. The outputs of this step of the analysis were statistical profiles for each pollutant characterizing air quality conditions at each monitoring site in the lower 48 states.[6]

The control and no-control scenario air quality profiles were then used as inputs to a modeling system which translates air quality to physical outcomes –such as mortality, emergency room visits, or crop yield losses– through the use of concentration-response functions. These concentration-response functions were in turn derived from studies found in the scientific literature on the health and ecological effects of air pollutants. At this point, estimates were derived of the differences between the two scenarios in terms of incidence rates for a broad range of human health and other effects of air pollution by year, by pollutant, and by monitor.[7]

In the next step, economic valuation models or coefficients were used to estimate the economic value of the reduction in incidence of those adverse effects which were amenable to such monetization. For example, a distribution of unit values derived from the economic literature was used to estimate the value of reductions in mortality risk associated with exposure to particulate matter. In addition, benefits which could not be expressed in economic terms were compiled and are presented herein. In some cases, quantitative estimates of scenario differences in the incidence of a nonmonetized effect were calculated.[8] In many other cases, available data and techniques were insufficient to support anything more than a qualitative characterization of the change in effects.

Finally, the costs and monetized benefits were combined to provide a range of estimates for the partial, net economic benefit of the CAA with the range reflecting quantified uncertainties associated with the physical effects and economic valuation steps.[9] The term “partial” is emphasized because only a subset of the total potential benefits of the CAA could be represented in economic terms due to limitations in ancal

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[4] These six pollutants are total suspended particulates (TSP), sulfur dioxide (SO_2), nitrogen oxides (NO_x), carbon monoxide (CO), volatile organic compounds (VOCs), and lead (Pb). The other CAA criteria pollutant, ozone (O_3), is formed in the atmosphere through the interaction of sunlight and ozone precursor pollutants such as NO_x and VOCs.

[5] By definition, 1970 emissions under the two scenarios are identical.

[6] The one exception is particulate matter (PM). For PM, air quality profiles for both Total Suspended Particulates (TSP) and particulates less than or equal to 10 microns in diameter (PM_{10}) were constructed at the county level rather than the individual monitor level.

[7] Or, for PM, by county.

[8] For example, changes in forced expiratory volume in one second (FEV_1) as a result of exposure to ozone were quantified but could not be expressed in terms of economic value.

[9] Although considerable uncertainties surround the direct cost, macroeconomic modeling, emissions modeling,, and air quality modeling steps, the ranges of aggregate costs and benefits presented in this analysis do not reflect these uncertainties. While the uncertainties in these components were assessed qualitatively, and in some cases quantitatively, resource limitations precluded the multiple macroeconomic model, emissions model, and air quality model runs which would have been required to propagate these uncertainties through the entire analytical sequence. As a result, complete quantitative measures of the aggregate uncertainty in the cost and benefit estimates could not be derived. However, the ranges presented do reflect quantitative measures of the uncertainties in the two most uncertain analytical steps: physical effects estimation and economic valuation.

cal resources, available data and models, and the state of the science.[10] Of paramount concern to the EPA Project Team was the paucity of concentration-response functions needed to translate air quality changes into measures of ecological effect. In addition, significant scientific evidence exists linking air pollution to a number of adverse human health effects which could not be effectively quantified and/or monetized.[11]

Review Process

The CAA requires EPA to consult with an outside panel of experts–referred to statutorily as the Advisory Council on Clean Air Act Compliance Analysis (the Council)–in developing the section 812 analyses. In addition, EPA is required to consult with the Department of Labor and the Department of Commerce.

The Council was organized in 1991 under the auspices and procedures of EPA's Science Advisory Board (SAB). Organizing the review committee under the SAB ensured that review of the section 812 studies would be conducted by highly qualified experts in an objective, rigorous, and publicly open manner. The Council has met many times during the development of the retrospective study to review methodologies and interim results. While the full Council retains overall review responsibility for the section 812 studies, some specific issues concerning physical effects and air quality modeling have been referred to subcommittees comprised of both Council members and members of other SAB committees. The Council's Physical Effects Review Subcommittee met several times and provided its own review findings to the full Council. Similarly, the Council's Air Quality Subcommittee, comprised of members and consultants of the SAB Clean Air Scientific Advisory Committee (CASAC), held several teleconference meetings to review methodology proposals and modeling results.

With respect to the interagency review process, EPA expanded the list of consulted agencies and convened a series of meetings during the design and early implementation phases from 1991 through late 1994. In late 1994, to ensure that all interested parties and the public received consistent information about remaining analytical issues and emerging results, EPA decided to use the public SAB review process as the primary forum for presenting and discussing issues and results. The Interagency Review Group was therefore discontinued as a separate process in late 1994.

A final, brief interagency review, pursuant to Circular A-19, was organized in August 1997 by the Office of Management and Budget and conducted following the completion of the extensive expert panel peer review by the SAB Council. During the course of the final interagency discussions, it became clear that several agencies held different views pertaining to several key assumptions in this study as well as to the best techniques to apply in the context of environmental program benefit-cost analyses, including the present study. The concerns include: (1) the extent to which air quality would have deteriorated from 1970 to 1990 in the absence of the Clean Air Act, (2) the methods used to estimate the number of premature deaths and illnesses avoided due to the CAA, (3) the methods used to estimate the value that individuals place on avoiding those risks, and (4) the methods used to value non-health related benefits. However, due to the court deadline the resulting concerns were not resolved during this final, brief interagency review. Therefore, this report reflects the findings of EPA and not necessarily other agencies in the Administration. Interagency discussion of some of these issues will continue in the context of the future prospective section 812 studies and potential regulatory actions.

Report Organization

The remainder of the main text of this report summarizes the key methodologies and findings of retrospective study. The direct cost estimation and macroeconomic modeling steps are presented in Chapter 2. The emissions modeling is summarized in Chapter 3. Chapter 4 presents the air quality modeling methodology and sample results. Chapter 5 describes the approaches used and principal results obtained through the physical effects estimation process. Economic valuation methodologies are described in Chapter 6. Chapter 7 presents the aggregated results of the cost and benefit estimates and describes and evaluates important uncertainties in the results.

Additional details regarding the methodologies and results are presented in the appendices and in the referenced supporting documents. Appendix A covers the direct cost and macroeconomic modeling. Appendix B provides additional detail on the sector-specific emissions modeling effort. Details of the air quality models used and results obtained are presented or referenced in Appendix C. The effects of the CAA on human health and visibility; aquatic, wetland, and forest ecosystems; and agriculture are presented in Appendices D, E, and F, respectively. Appendix G presents details of the lead (Pb) benefits analysis. Air toxics reduction benefits are discussed in Appendix H. The methods and assumptions used to value quantified effects of the CAA in economic terms are described in Appendix I. Appendix J describes some areas of research which may increase comprehensiveness and reduce uncertainties in effect estimates for future assessments, and describes plans for future section 812 analyses.

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[10] It should be noted that there is some uncertainty associated with the estimates of economic costs as well and that some omitted components of adverse economic consequences of pollution control programs may be significant. For example, some economists argue that the economic costs of the CAA reported herein may be significantly underestimated to the extent potential adverse effects of regulation on technological innovation are not captured. Nevertheless, it is clear that the geographic, population, and categorical coverage of monetary cost effects is significantly greater than coverage of monetized benefits in this analysis.

[11] For example, while there is strong evidence of a link between exposure to carbon monoxide and reduced time of onset of angina attack, there are no valuation functions available to estimate the economic loss associated with this effect.

2

Cost and Macroeconomic Effects

The costs of complying with Clean Air Act (CAA) requirements through the 1970 to 1990 period affected patterns of industrial production, capital investment, productivity, consumption, employment, and overall economic growth. The purpose of the analyses summarized in this chapter was to estimate those direct costs and the magnitude and significance of resulting changes to the overall economy. This was accomplished by comparing economic indicators under two alternative scenarios: a control scenario serving as the historical benchmark, including the historical CAA as implemented; and a no-control scenario which assumes historical CAA programs did not exist. The estimated economic consequences of the historical CAA were taken as the difference between these two scenarios.

Data used as inputs to the cost analysis can be classified into two somewhat overlapping categories based on the information source: survey-based information (generally gathered by the Census Bureau) and information derived from various EPA analyses. For the most part, cost estimates for stationary air pollution sources (e.g., factory smokestacks) are based on surveys of private businesses that attempt to elicit information on annual pollution control outlays by those businesses. Estimates of pollution control costs for mobile sources (e.g., automobiles) are largely based on EPA analyses, rather than on direct observation and measurement of compliance expenditures. For example, to determine one component of the cost of reducing lead emissions from mobile sources, the Project Team used an oil refinery production cost model to calculate the incremental cost required to produce unleaded (or less-leaded, as appropriate) rather than leaded gasoline, while maintaining the octane level produced by leaded gasoline.

As is the case with many policy analyses, a significant uncertainty arises in the cost analysis as a consequence of constructing a hypothetical scenario. With this retrospective analysis covering almost twenty years, difficulties arise in projecting alternative technological development paths. In some cases, the analytical assumptions used to project the alternative scenario are not immediately apparent. For example, the surveys covering stationary source compliance expenditures require respondents to report pollution abatement expenditures—implicitly asking them to determine by how much the company's costs would decline if there were no CAA compliance requirements. While a response might be relatively straightforward in the few years following passage of the CAA, a meaningful response becomes more difficult after many years of technical change and investment in less-polluting plant and equipment make it difficult to determine the degree to which total costs would differ under a "no CAA" scenario. In cases such as this, assumptions concerning the alternative hypothetical scenario are made by thousands of individual survey respondents. Where cost data are derived from EPA analyses, the hypothetical scenario assumptions are, at least in theory, more apparent. For example, when determining the incremental cost caused by pollution-control requirements, one needs to make assumptions (at least implicitly) about what an auto would look like absent pollution control requirements. In either case, the need to project hypothetical technology change for two decades introduces uncertainty into the assessment results, and this uncertainty may be difficult to quantify.

The remainder of this chapter summarizes the basic methods and results of the direct compliance cost and macroeconomic analyses. Further details regarding the modeling methods and assumptions employed, as well as additional analytical results, are presented in Appendix A.

Direct Compliance Costs

Compliance with the CAA imposed direct costs on businesses, consumers, and governmental units; and triggered other expenditures such as governmental regulation and monitoring costs and expenditures for

Table 1. Estimated Annual CAA Compliance Costs ($billions).

	Expenditures		**Annualized Costs** $1990 at:		
Year	**$current**	**$1990**	**3%**	**5%**	**7%**
1973	7.2	19.6	11.0	11.0	11.1
1974	8.5	21.4	13.2	13.4	13.7
1975	10.6	24.4	13.3	13.6	14.0
1976	11.2	24.1	14.1	14.6	15.1
1977	11.9	24.1	15.3	15.9	16.6
1978	12.0	22.6	15.0	15.8	16.7
1979	14.4	24.8	17.3	18.3	19.3
1980	16.3	25.7	19.7	20.8	22.0
1981	17.0	24.4	19.6	20.9	22.3
1982	16.0	21.6	18.6	20.1	21.7
1983	15.5	20.1	19.1	20.7	22.5
1984	17.3	21.6	20.1	21.9	23.8
1985	19.1	22.9	22.5	24.4	26.5
1986	17.8	20.8	21.1	23.2	25.4
1987	18.2	20.6	22.1	24.2	26.6
1988	18.2	19.8	22.0	24.3	26.7
1989	19.0	19.8	22.9	25.3	27.8
1990	19.0	19.0	23.6	26.1	28.7

research and development by both government and industry. Although expenditures unadjusted for inflation — that is, expenditures denominated in "current dollars"— increased steadily from $7 billion to $19 billion per year over the 1973 to 1990 period,[12] annual CAA compliance expenditures adjusted for inflation were relatively stable, averaging near $25 billion (in 1990 dollars) during the 1970s and close to $20 billion during most of the 1980s (see Table 1). Aggregate compliance expenditures were somewhat less than one half of one percent of total domestic output during that period, with the percentage falling from two thirds of one percent of total output in 1975 to one third of one percent in 1990.

Although useful for many purposes, a summary of direct annual expenditures may not the best cost measure to use when comparing costs to benefits. Capital expenditures are investments, generating a stream of benefits and opportunity cost[13] over the life of the investment. The appropriate accounting technique to use for capital expenditures in a cost/benefit analysis is to annualize the expenditure. This technique, analogous to calculating the monthly payment associated with a home mortgage, involves spreading the cost of the capital equipment over the useful life of the equipment using a discount rate to account for the time value of money.

For this cost/benefit analysis, "annualized" costs reported for any given year are equal to O&M expenditures — including R&D and other similarly recurring expenditures — plus amortized capital costs (i.e., depreciation plus interest costs associated with the existing capital stock) for that year. Stationary source air pollution control capital costs were amortized over 20 years; mobile source air pollution control costs were amortized over 10 years.[14] All capital expenditures were annualized using a five percent, inflation-adjusted rate of interest. Additionally, annualized costs were calculated using discount rates of three and seven percent to determine the sensitivity of the cost results to changes in the discount rate. Table 1 summarizes costs annualized at three, five, and seven percent, as well as annual expenditures.

Total expenditures over the 1973-1990 period, discounted to 1990 using a five percent (net of inflation) discount rate, amount to 628 billion dollars (in 1990 dollars). Discounting the annualized cost stream to 1990 (with both annualization and discounting procedures using a five percent rate) gives total costs of 523 billion dollars (in 1990 dollars). Aggregate annualized costs are less than expenditures because the annualization procedure spreads some of the capital cost beyond 1990.[15]

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[12] Due to data limitations, the cost analysis for this CAA retrospective starts in 1973, missing costs incurred in 1970-72. This limitation is not likely to be significant, however, because relatively little in the way of compliance with the "new" provisions of the 1970 CAA was required in the first two years following passage.

[13] In this context, "opportunity cost" is defined as the value of alternative investments or other uses of funds foregone as a result of the investment.

[14] Although complete data are available only for the period 1973-1990, EPA's Cost of Clean report includes capital expenditures for 1972 (see Appendix A for more details and complete citation). Those capital expenditure data have been used here. Therefore, amortized costs arising from 1972 capital investments are included in the 1973-1990 annualized costs, even though 1972 costs are not otherwise included in the analysis. Conversely, some capital expenditures incurred in the 1973-1990 period are not reflected in the 1973-1990 annualized costs — those costs are spread through the following two decades, thus falling outside of the scope of this study (e.g., only one year of depreciation and interest expense is included for 1989 capital expenditures). Similarly, benefits arising from emission reductions realized after 1990 as a result of capital investments made during the 1970 to 1990 period of this analysis are not included in the estimates of benefits included in this report.

[15] This adjustment is required because many 1970 to 1990 investments in control equipment continue to yield benefits beyond 1990. Annualization of costs beyond 1990 ensures that the costs and benefits of any particular investment are properly scaled and matched over the lifetime of the investment.

Indirect Effects of the CAA

Through changing production costs, CAA implementation induced changes in consumer good prices, and thus in the size and composition of economic output. The Project Team used a general equilibrium macroeconomic model to assess the extent of such second-order effects. This type of model is useful because it can capture the feedback effects of an action. In the section 812 macroeconomic modeling exercise, the feedback effects arising from expenditure changes were captured, but the analogous effects arising from improvements in human health were not captured by the model. For example, the macroeconomic model results do not reflect the indirect economic effects of worker productivity improvements and medical expenditure savings caused by the CAA. Consequently, the macroeconomic modeling exercise provides limited and incomplete information on the type and potential scale of indirect economic effects.

The effects estimated by the macroeconomic model can be grouped into two broad classes: sectoral impacts (i.e., changes in the composition of economic output), and aggregate effects (i.e., changes in the degree of output or of some measure of human welfare). The predicted sectoral effects were used as inputs to the emissions models as discussed in Chapter 3. In general, the estimated second-order macroeconomic effects were small relative to the size of the U.S. economy. See Appendix A for more detail on data sources, analytical methods, and results for the macroeconomic modeling performed for this assessment.

Sectoral Impacts

The CAA had variable compliance impacts across economic sectors. The greatest effects were on the largest energy producers and consumers, particularly those sectors which relied most heavily on consumption of fossil fuels (or energy generated from fossil fuels). In addition, production costs increased more for capital-intensive industries than for less capital-intensive industries under the control scenario due to a projected increase in interest rates. The interest rate increase, which resulted in an increase in the cost of capital, occurred under the control scenario because CAA-mandated investment in pollution abatement reduced the level of resources available for other uses, including capital formation.

Generally, the estimated difference in cost impacts under the control and no-control scenarios for a particular economic sector was a function of the relative energy-intensity and capital-intensity of that sector. Increased production costs in energy- and capital-intensive sectors under the control scenario were reflected in higher consumer prices, which resulted in reductions in the quantity of consumer purchases of goods and services produced by those sectors. This reduction in consumer demand under the control scenario led, ultimately, to reductions in output and employment in those sectors. The sectors most affected by the CAA were motor vehicles, petroleum refining, and electricity generation. The electricity generation sector, for example, incurred a two to four percent increase in consumer prices by 1990, resulting in a three to five and a half percent reduction in output. Many other manufacturing sectors saw an output effect in the one percent range.

Some other sectors, however, were projected to increase output under the control scenario. Apart from the pollution control equipment industry, which was not separately identified and captured in the macroeconomic modeling performed for this study, two example sectors for which output was higher and prices were lower under the control scenario are food and furniture. These two sectors showed production cost and consumer price reductions of one to two percent relative to other industries under the control scenario, resulting in output and employment increases of similar magnitudes.

Aggregate Effects

As noted above, the control and no-control scenarios yield different estimated mixes of investment. In particular, the control scenario was associated with more pollution control capital expenditure and less consumer commodity capital expenditure. As a result, the growth pattern of the economy under the control scenario differed from the no-control scenario. Under the control scenario, the macroeconomic model projected a rate of long-run GNP growth about one twentieth of one percent per year lower than under the no-control scenario. Aggregating these slower growth effects of the control scenario over the entire 1970 to 1990 period of this study results, by 1990, in a level of GNP one percent (or approximately $55 billion) lower than that projected under the no-control scenario.

Although small relative to the economy as a whole, the estimated changes in GNP imply that the potential impact of the CAA on the economy by 1990 was greater than that implied by expenditures ($19 billion in 1990) or annualized costs ($26 billion in 1990, annualized at five percent). Discounting the stream of 1973-1990 GNP effects to 1990 gives an aggregate impact on production of 1,005 billion dollars (in 1990 dollars discounted at five percent). Of that total, $569 billion represent reductions in household consumption, and another $200 billion represent government consumption, for an aggregate effect on U.S. consumption of goods and services equal to 769 billion dollars. Both the aggregate GNP effects and aggregate consumption effects exceed total 1973-1990 expenditures ($628 billion) and annualized costs ($523 billion, with all dollar quantities in $1990, discounted at five percent).

Changes in GNP (or, even, changes in the national product account category "consumption") do not necessarily provide a good indication of changes in social welfare. Social welfare is not improved, for example, by major oil tanker spills even though measured GNP is increased by the "production" associated with clean-up activities. Nevertheless, the effects of the CAA on long-term economic growth would be expected to have had some effect on economic welfare. One of the characteristics of the macroeconomic model used by the Project Team is its ability to estimate a measure of social welfare change which is superior to GNP changes. This social welfare measure estimates the monetary compensation which would be required to offset the losses in consumption (broadly defined) associated with a given policy change. The model reports a range of results, with the range sensitive to assumptions regarding how cost impacts are distributed through society. For the CAA, the model reports an aggregate welfare effect of 493 billion to 621 billion dollars (in 1990 dollars), depending on the distributional assumptions used. This range does not differ greatly from the range of results represented by 1973-1990 expenditures, compliance costs, and consumption changes.

Uncertainties and Sensitivities in the Cost and Macroeconomic Analysis

The cost and macroeconomic analyses for the present assessment relied upon survey responses, EPA analyses, and a macroeconomic simulation model. Although the Project Team believes that the results of the cost and macroeconomic analyses are reasonably reliable, it recognizes that every analytical step is subject to uncertainty. As noted at the beginning of this chapter, explicit and implicit assumptions regarding hypothetical technology development paths are crucial to framing the question of the cost impact of the CAA. In addition, there is no way to verify the accuracy of the survey results used;[16] alternative, plausible cost analyses exist that arrive at results that differ from some of the results derived from EPA analyses; and it is not clear how the use of a general equilibrium macroeconomic model affects the accuracy of macroeconomic projections in a macroeconomy characterized by disequilibrium. For many factors engendering uncertainty, the degree or even the direction of bias is unknown. In several areas, nevertheless, uncertainties and/or sensitivities can be identified that may bias the results of the analysis.

Productivity and Technical Change

An important component of the macroeconomic model used by the Project Team is its treatment of technical change and productivity growth. Three factors associated with productivity and technical change have been identified which may bias the results of the macroeconomic simulation: (1) the long-run effects of reducing the "stock" of technology, (2) the possible "chilling" effect of regulations on innovation and technical change, and (3) the role of endogenous productivity growth within the macroeconomic model.

The macroeconomic model projected a decrease in the growth of GNP as a result of CAA compliance. Decreased growth was due not only to decreased capital investment, but also to decreased factor productivity. The annual decrement in productivity can be thought of as a reduction of the stock of available technology. That reduction in stock could be expected to affect macroeconomic activity after 1990, as well as during the 1973-1990 period studied by the Project Team. Thus, to the extent that this effect exists, the Project Team has underestimated the macroeconomic impact of the CAA by disregarding the effect of 1973-1990 productivity change decrements on post-1990 GNP.

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[16] For an example of the difficulties one encounters in assessing the veracity of survey results, see the discussion in Appendix A on the apparently anomalous growth in stationary source O&M expenditures in relation to the size of the stationary source air pollution control capital stock.

Some economists contend that regulations have a "chilling" effect on technological innovation and, hence, on productivity growth. Two recent studies by Gray and Shadbegian,[17] which are sometimes cited in support of this contention, suggest that pollution abatement regulations may decrease productivity levels in some manufacturing industries. The macroeconomic model allowed policy-induced productivity change through the mechanism of price changes and resultant factor share changes. To the extent that additional policy-induced effects on productivity growth exist, the Project Team has underestimated the impact of the CAA on productivity growth during the 1973-1990 period, and, thus, has underestimated macroeconomic impacts during the 1973-1990 period and beyond.

The macroeconomic model allowed productivity growth to vary with changes in prices generated by the model. This use of "endogenous" productivity growth is not universal in the economic growth literature — that is, many similar macroeconomic models do not employ analogous forms of productivity growth. The Project Team tested the sensitivity of the model results to the use of endogenous productivity growth. If the model is run without endogenous productivity growth, then the predicted macroeconomic impacts (GNP, personal consumption, etc.) of the CAA are reduced by approximately 20 percent. That is, to the extent that use of endogenous productivity growth in the macroeconomic model is an inaccurate simulation technique, then the Project Team has overestimated the macroeconomic impact of the CAA.

Discount Rates

There is a broad range of opinion in the economics profession regarding the appropriate discount rate to use in analyses such as the current assessment. Some economists believe that the appropriate rate is one that approximates the social rate of time preference — that is, the rate of return at which individuals are willing to defer consumption to the future. A three percent rate would approximate the social rate of time preference (all rates used here are "real", i.e., net of price inflation impacts). Others believe that a rate that approximates the opportunity cost of capital (e.g., seven percent or greater) should be used.[18] A third school of thought holds that some combination of the social rate of time preference and the opportunity cost of capital is appropriate, with the combination effected either by use of an intermediate rate or by use of a multiple-step procedure employing the social rate of time preference as the "discount rate," but still accounting for the opportunity cost of capital.

The Project Team elected to use an intermediate rate (five percent), but recognizes that analytical results aggregated across the study period are sensitive to the discount rate used. Consequently, all cost measures are presented at three and seven percent, as well as the base case five percent. Table 2 summarizes major cost and macroeconomic impact measures expressed in constant 1990 dollars, and discounted to 1990 at rates of three, five, and seven percent.

Table 2. Compliance Cost, GNP, and Consumption Impacts Discounted to 1990 ($1990 billions)

	3%	5%	7%
Expenditures	$52	628	761
Annualized Costs	417	523	657
GNP	880	1005	1151
Household Consumption	500	569	653
HH and Gov't Consumption	676	769	881

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[17] Gray, Wayne B., and Ronald J. Shadbegian, "Environmental Regulation and Manufacturing Productivity at the Plant Level," Center for Economic Studies Discussion Paper, CES 93-6, March 1993. Gray, Wayne B., and Ronald J. Shadbegian, "Pollution Abatement Costs, Regulation, and Plant-Level Productivity," National Bureau of Economic Research, Inc., Working Paper Series, Working Paper No. 4994, January 1995.

[18] Some would argue that use of the opportunity cost of capital approach would be inappropriate in the current assessment if the results of the macroeconomic modeling (such as GNP) were used as the definition of "cost," since the macro model already accounts for the opportunity cost of capital. The appropriate rate would then be the social rate of time preference.

Exclusion of Health Benefits from the Macroeconomic Model

The macroeconomic modeling exercise was designed to capture the second-order macroeconomic effects arising from CAA compliance expenditures. Those predicted second-order effects are among the factors used to drive the emissions estimates and, ultimately, the benefits modeled for this assessment. The benefits of the CAA, however, would also be expected to induce second-order macroeconomic effects. For example, increased longevity and decreased incidence of non-fatal heart attacks and strokes would be expected to improve macroeconomic performance measures. The structure of the overall analysis, however, necessitated that these impacts be excluded from the macroeconomic simulation.

The first-order CAA beneficial effects have been included in the benefits analysis for this study, including measures that approximate production changes (e.g., income loss due to illness, or lost or restricted work days; income loss due to impaired cognitive ability; and income loss due to reduced worker production in certain economic sectors). These measures are analogous to compliance expenditures in the cost analysis. The second-order benefits impacts, which would result from price changes induced by CAA-related benefits, have not been estimated. It is likely that the estimated adverse second-order macroeconomic impacts would have been reduced had the impact of CAA benefits been included in the macroeconomic modeling exercise; however, the magnitude of this potential upward bias in the estimate of adverse macroeconomic impact was not quantitatively assessed.

3

Emissions

This chapter presents estimates of emissions reductions due to the Clean Air Act (CAA) for six criteria air pollutants. Reductions are calculated by estimating, on a sector-by-sector basis, the differences in emissions between the control and no-control scenarios. While the relevant years in this analysis are 1970 through 1990, full reporting of emissions was only made for the 1975 to 1990 period since 1970 emission levels are, by assumption, identical for the two scenarios. The criteria pollutants for which emissions are reported in this analysis are: total suspended particulates (TSP),[19] carbon monoxide (CO), volatile organic compounds (VOC), sulfur dioxide (SO_2), nitrogen oxides (NO_x), and Lead (Pb).

The purpose of the present study is to estimate the differences in economic and environmental conditions between a scenario reflecting implementation of historical CAA controls and a scenario which assumes that no additional CAA-related control programs were introduced after 1970. Because of the focus on differences in –rather than absolute levels of– emissions between the scenarios, the various sector-specific emission models were used to estimate both the control and no-control scenario emission inventories. This approach ensures that differences between the scenarios are not distorted by differences between modeled and actual historical emission estimates.[20]

Despite the use of models to estimate control scenario emission inventories, the models used were configured and/or calibrated using historical emissions estimates. The control scenario utility emissions estimates, for example, were based on the ICF CEUM model which was calibrated using historical emissions inventory data.[21] In other cases, such as the EPA Emissions Trends Report (Trends) methodology[22] used to estimate industrial process emissions, historical data were used as the basis for control scenario emissions with little or no subsequent modification. Nevertheless, differences in model selection, model configuration, and macroeconomic input data[23] result in unavoidable, but in this case justifiable, differences between national total historical emission estimates and national total control scenario emission estimates for each pollutant. Comparisons between no-control, control, and official EPA Trends Report historical emissions inventories are presented in Appendix B.[24]

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[19] In 1987, EPA replaced the earlier TSP standard with a standard for particulate matter of 10 microns or smaller (PM_{10}).

[20] By necessity, emission models must be used to estimate the hypothetical no-CAA scenario. If actual historical emissions data were adopted for the control scenario, differences between the monitoring data and/or models used to develop historical emission inventories and the models used to develop no-control scenario emission estimates would bias the estimates of the differences between the scenarios.

[21] See ICF Resources, Inc., “Results of Retrospective Electric Utility Clean Air Act Analysis - 1980, 1985 and 1990,” September 30, 1992, Appendix C.

[22] EPA, 1994a: U.S. Environmental Protection Agency, “National Air Pollutant Emission Trends, 1900-1993,” EPA-454/R-94-027, Office of Air Quality Planning and Standards, Research Triangle Park, NC, October 1994.

[23] The Jorgenson/Wilcoxen macroeconomic model outputs were used to configure both the control and no-control scenario emission model runs. While this satisfies the primary objective of avoiding “across model” bias between the scenarios, the macroeconomic conditions associated with the control scenario would not be expected to match actual historical economic events and conditions. To the extent actual historical economic conditions are used to estimate official historical emission inventories, conformity between these historical emissions estimates and control scenario emission estimates would be further reduced.

[24] In general, these comparisons show close correspondence between control scenario and Trends estimates with the largest differences occurring for VOC and CO emissions. The Trends report VOC estimates are generally higher than the control scenario estimates due primarily to the inclusion of Waste Disposal and Recycling as a VOC source in the Trends report. This inconsistency is of no consequence since Waste Disposal and Recycling sources were essentially uncontrolled by the historical CAA and therefore do not appear as a difference between the control and no-control scenarios. The higher CO emission estimates in the Trends Report are primarily associated with higher off-highway vehicle emissions estimates. Again, since off-highway emissions do not change between the control and no-control scenario in the present analysis, this inconsistency is of no consequence.

To estimate no-control scenario emissions, sector-specific historical emissions are adjusted based on changes in the following two factors: (1) growth by sector predicted to occur under the no-control scenario; and (2) the exclusion of controls attributable to specific provisions of the CAA.

To adjust emissions for economic changes under the no-control scenario, activity levels that affect emissions from each sector were identified. These activity levels include, for example, fuel use, industrial activity, and vehicle miles traveled (VMT). The Jorgenson-Wilcoxen (J/W) general equilibrium model was used to estimate changes in general economic conditions, as well as sector-specific economic outcomes used as inputs to the individual sector emission models.[25]

Table 3. Summary of Sector-Specific Emission Modeling Approaches.

Sector	Modeling Approach
On-Highway Vehicles	Modeled using ANL's TEEMS; adjusted automobile emission estimates by changes in personal travel and economic activity in the without CAA case. Truck VMT was obtained from the Federal Highway Administration (FHWA). MOBILE5a emission factors were used to calculate emissions. Lead emission changes from gasoline were estimated by Abt Associates based on historical gasoline sales and the lead content of leaded gasoline in each target year.
Off-Highway Vehicles	ELI analysis based on Trends methods. Recalculated historical emissions using 1970 control efficiencies from Trends. No adjustment was made to activity levels in the without the CAA case.
Electric Utilities	ICF's Coal and Electric Utility Model (CEUM) used to assess SO_2, NO_x, and TSP emission changes. Electricity sales levels were adjusted with results of the J/W model. The Argonne Utility Simulation Model (ARGUS) provided CO and VOC results. Changes in activity levels were adjusted with results of the J/W model. Lead emissions were calculated based on energy consumption data and Trends emission factors and control efficiencies.
Industrial Combustion	ANL industrial boiler analysis for SO_2, NO_x, and TSP using the Industrial Combustion Emissions (ICE) model. VOC and CO emissions from industrial boilers were calculated based on Trends methods; recalculated using 1970 control efficiencies. Lead emissions calculated for boilers and processes based on Trends fuel consumption data, emission factors, and 1970 control efficiencies.
Industrial Processes	ELI analyzed industrial process emissions based on Trends methods. Adjusted historical emissions with J/W sectoral changes in output, and 1970 control efficiencies from Trends. Lead emissions calculated for industrial processes and processes based on Trends fuel consumption data, emission factors, and 1970 control efficiencies.
Commercial / Residential	ANL's Commercial and Residential Simulation System (CRESS) model was used.

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[25] For example, the change in distribution of households by income class predicted by the J/W model was used as input to the transportation sector model system. Changes in household income resulted in changes in vehicle ownership and usage patterns which, in turn, influence VMT and emissions. (See Pechan, 1995, p. 43).

The specific outputs from the J/W model used in this analysis are the percentage changes in gross national product (GNP), personal consumption, and output for various economic sectors under the control and no-control scenario for the years 1975, 1980, 1985, and 1990.[26] The sectors for which the results of the J/W model are used include: industrial processes, electric utilities, highway vehicles, industrial boilers, and the commercial/residential sector. For the off-highway sector, economic growth was not taken into account as there was no direct correspondence between J/W sectors and the off-highway vehicle source category activity.

In addition to adjusting for economic activity changes, any CAA-related control efficiencies that were applied to calculate control scenario emissions were removed for the no-control scenario. In most instances, emissions were recalculated based on 1970 control levels.

Uncertainty associated with several key modeling inputs and processes may contribute to potential errors in the emission estimates presented herein. Although the potential errors are likely to contribute in only a minor way to overall uncertainty in the estimated monetary benefits of the Clean Air Act, the most significant emission modeling uncertainties are described at the end of this chapter.

Sector-Specific Approach

The approaches used to calculate emissions for each sector vary based on the complexity of estimating emissions in the absence of CAA controls, taking economic activity levels and CAA regulations into account. For the off-highway vehicle and industrial process sectors, a relatively simple methodology was developed. The approaches used for the highway vehicles, electric utilities, industrial boilers, and commercial/residential sectors were more complex because the J/W model does not address all of the determinants of economic activity in these sectors that might have changed in the absence of regulation. The approaches by sector used to estimate emissions for the two scenarios are summarized in Table 3, and are described in more detail in Appendix B.

Summary of Results

Figure 2 compares the total estimated sulfur dioxide emission from all sectors under the control and no-control scenarios over the period from 1975 to 1990. Figures 3, 4, 5, 6, and 7 provide similar comparisons for NO_x, VOCs, CO, TSP, and Lead (Pb) respectively.

Additional tables presented in Appendix B provide further breakdown of the emissions estimates by individual sector. The essential results are characterized below. For most sectors, emission levels under the control scenario were substantially lower than levels projected under the no-control scenario. For some pollutants, for example NO_x, most of the reductions achieved under the control scenario offset the growth in emissions which would have occurred under the no-control case as a result of increases in population and economic activity. For other pollutants, particularly lead, most of the difference in 1990 emissions projected under the two scenarios reflects significant improvement relative to 1970 emission levels. Appendix B also assesses the consistency of the control and no-control scenario estimates for 1970 to 1990 with pre-1970 historical emissions trends data.

The CAA controls that affected SO_2 emitting sources had the greatest proportional effect on industrial process emissions, which were 60 percent lower in 1990 than they would have been under the no-control scenario. SO_2 emissions from electric utilities and industrial boilers were each nearly 40 percent lower in 1990 as a result of the controls. In terms of absolute tons of emission reductions, controls on electric utilities account for over 10 million of the total 16 million ton difference between the 1990 control and no-control scenario SO_2 emission estimates.

CAA regulation of the highway vehicles sector led to the greatest percent reductions in VOC and NO_x. Control scenario emissions of these pollutants in 1990 were 66 percent and 47 percent lower, respectively, than the levels estimated under the no-control scenario. In absolute terms, highway vehicle VOC controls account for over 15 million of the roughly 17 million ton difference in control and no-control scenario emissions.

Differences between control and no-control scenario CO emissions are also most significant for highway vehicles. In percentage terms, highway vehicle CO emissions were 56 percent lower in 1990 under the control scenario than under the no-control scenario. Industrial process CO emission estimates under the control scenario were about half the levels projected under the no-control scenario. Of the roughly 89 mil-

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[26] For details regarding the data linkages between the J/W model and the various emission sector models, see Pechan (1995).

Figure 2. Control and No-control Scenario Total SO_2 Emission Estimates.

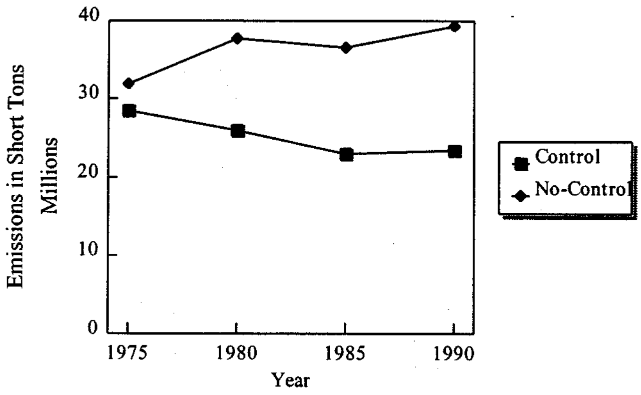


Figure 5. Control and No-control Scenario Total CO Emission Estimates.

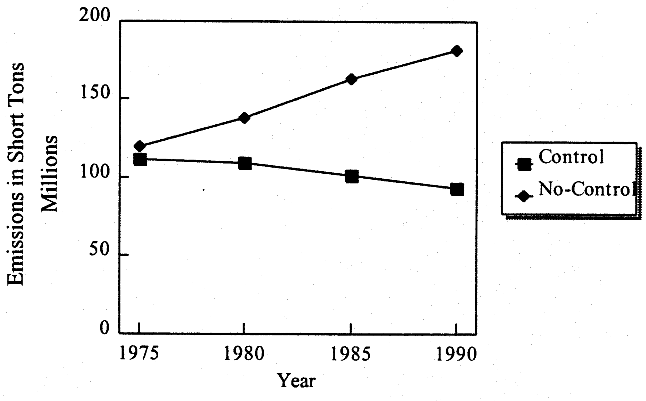


Figure 3. Control and No-control Scenario Total NO_X Emission Estimates.

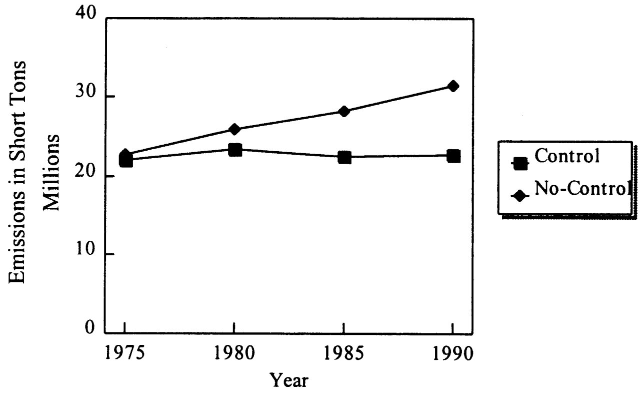


Figure 6. Control and No-control Scenario Total TSP Emission Estimates.

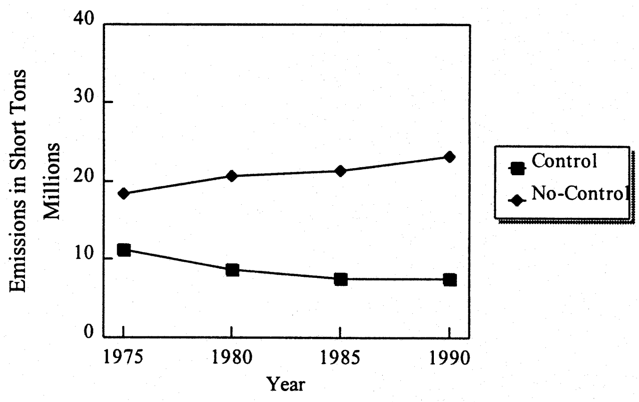


Figure 4. Control and No-control Scenario Total VOC Emission Estimates.

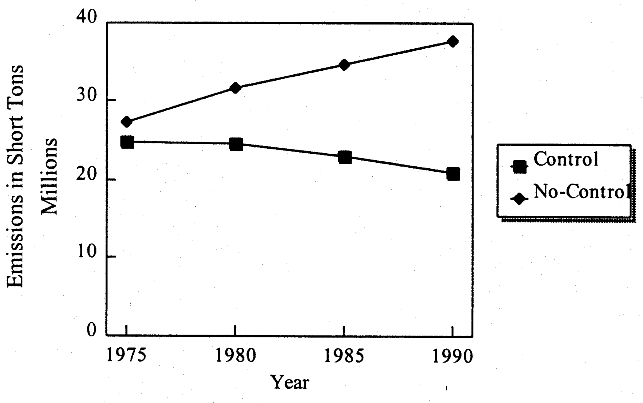
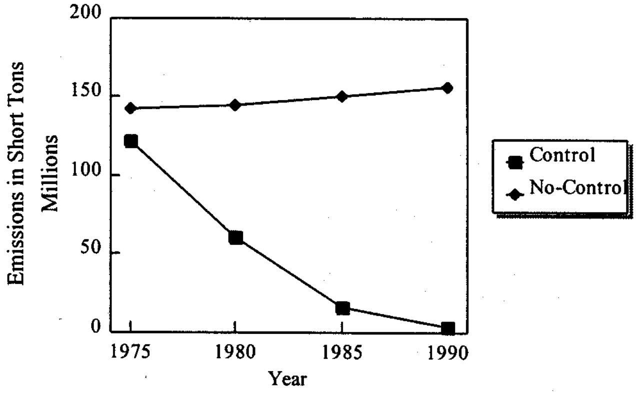


Figure 7. Control and No-control Scenario Total Pb Emission Estimates.



lion ton difference in CO emissions between the two scenarios, 84 million tons are attributable to highway vehicle controls and the rest is associated with reductions from industrial process emissions.

For TSP, the highest level of reductions on a percentage basis was achieved in the electric utilities sector. TSP emissions from electric utilities were 93 percent lower in 1990 under the control scenario than projected under the no-control scenario. TSP emissions from industrial processes were also significantly lower on a percentage basis under the control scenario, with the differential reaching 76 percent by 1990.

This is not an unexpected result as air pollution control regulations in the 1970's focused on solving the visible particulate problems from large fuel combustors. In terms of absolute tons, electric utilities account for over 5 million of the 16 million ton difference between the two scenarios and industrial processes account for almost 10 million tons.

The vast majority of the difference in lead emissions under the two scenarios is attributable to reductions in burning of leaded gasoline. By 1990, reductions in highway vehicle emissions account for 221 thousand of the total 234 thousand ton difference in lead emissions. As shown in more detail in Appendix B, airborne lead emissions from all sectors were virtually eliminated by 1990.

As described in the following chapter and in Appendix C, these emissions inventories were used as inputs to a series of air quality models. These air quality models were used to estimate air quality conditions under the control and no-control scenarios.

Uncertainty in the Emissions Estimates

The emissions inventories developed for the control and no-control scenarios reflect at least two major sources of uncertainty. First, potential errors in the macroeconomic scenarios used to configure the sector-specific emissions model contribute to uncertainties in the emissions model outputs. Second, the emissions models themselves rely on emission factors, source allocation, source location, and other parameters which may be erroneous.

An important specific source of potential error manifest in the present study relates to hypothetical emission rates from various sources under the no-control scenario. Emission rates from motor vehicles, for example, would have been expected to change during the 1970 to 1990 period due to technological changes not directly related to implementation of the Clean Air Act (e.g., advent of electronic fuel injection, or EFI). However, the lack of emissions data from vehicles with EFI but without catalytic converters compelled the Project Team to use 1970 emission factors throughout the 1970 to 1990 period for the no-control scenario. Although this creates a potential bias in the emissions inventories, the potential errors from this and other uncertainties in the emissions inventories are considered unlikely to contribute significantly to overall uncertainty in the monetary estimates of Clean Air Act benefits. This conclusion is based on the demonstrably greater influence on the monetary benefit estimates of uncertainties in other analytical components (e.g., concentration-response functions). A list of the most significant potential errors in the emissions modeling, and their significance relative to overall uncertainty in the monetary benefit estimate, is presented in Table 4.

Table 4. Uncertainties Associated with Emissions Modeling.

Potential Source of Error	**Direction of Potential Bias in Estimate of Emission Reduction Benefits**	**Significance Relative to Key Uncertainties in Overall Monetary Benefit Estimate**
Use of 1970 motor vehicle emission factors for no-control scenario without adjustment for advent of Electronic Fuel Injection (EFI) and Electronic Ignition (EI).	Overestimate.	Unknown, but likely to be minor due to overwhelming significance of catalysts in determining emission rates.
Use of ARGUS for utility CO and VOC rather than CEUM.	Unknown.	Negligible.
Use of historical fuel consumption to estimate 1975 SO_2, NO_x, TSP utility emissions.	Unknown.	Negligible.
Adoption of assumption that utility unit inventories remain fixed between the control and no-control scenarios.	Overestimate.	Unknown, but likely to be small since the CAA had virtually no effect on costs of new coal-fired plants built prior to 1975 and these plants comprise a large majority of total coal-fired capacity operating in the 1970 to 1990 period. (See ICF CEUM Report, p. 7).

4

Air Quality

Air quality modeling is the crucial analytical step which links emissions to changes in atmospheric concentrations of pollutants which affect human health and the environment. It is also one of the more complex and resource-intensive steps, and contributes significantly to overall uncertainty in the bottom-line estimate of net benefits of air pollution control programs. The assumptions required to estimate hypothetical no-control scenario air quality conditions are particularly significant sources of uncertainty in the estimates of air quality change, especially for those pollutants which are not linearly related to changes in associated emissions. Specific uncertainties are described in detail at the end of this chapter.

The key challenges faced by air quality modelers attempting to translate emission inventories into air quality measures involve modeling of pollutant dispersion and atmospheric transport, and modeling of atmospheric chemistry and pollutant transformation. These challenges are particularly acute for those pollutants which, rather than being directly emitted, are formed through secondary formation processes. Ozone is the paramount example since it is formed in the atmosphere through complex, nonlinear chemical interactions of precursor pollutants, particularly volatile organic compounds (VOCs) and nitrogen oxides (NO_x). In addition, atmospheric transport and transformation of gaseous sulfur dioxide and nitrogen oxides to particulate sulfates and nitrates, respectively, contributes significantly to ambient concentrations of fine particulate matter. In addition to managing the complex atmospheric chemistry relevant for some pollutants, air quality modelers also must deal with uncertainties associated with variable meteorology and the spatial and temporal distribution of emissions.

Given its comprehensive nature, the present analysis entails all of the aforementioned challenges, and involves additional complications as well. For many pollutants which cause a variety of human health and environmental effects, the concentration-response functions which have been developed to estimate those effects require, as inputs, different air quality indicators. For example, adverse human health effects of particulate matter are primarily associated with the respirable particle fraction;[27] whereas household soiling is a function of total suspended particulates, especially coarse particles. It is not enough, therefore, to simply provide a single measure of particulate matter air quality. Even for pollutants for which particle size and other characteristics are not an issue, different air quality indicators are needed which reflect different periods of cumulative exposure (i.e., “averaging periods”). For example, 3-month growing season averages are needed to estimate effects of ozone on yields of some agricultural crops, whereas adverse human health effect estimates require ozone concentration profiles based on a variety of short-term averaging periods.[28]

Fortunately, in responding to the need for scientifically valid and reliable estimation of air quality changes, air quality modelers and researchers have developed a number of highly sophisticated atmospheric dispersion and transformation models. These models have been employed for years supporting the development of overall federal clean air programs, national assessment studies, State Implementation Plans (SIPs), and individual air toxic source risk assessments. Some of these models, however, require massive amounts of computing power. For example, completing the 160 runs of the Regional Acid Deposition Model (RADM) required for the present study required approximately 1,080 hours of CPU time on a Cray-YMP supercomputer at EPA’s Bay City Supercomputing Center.

Given the resource-intensity of many state-of-the-art models, the Project Team was forced to make difficult choices regarding where to invest the limited resources available for air quality modeling. With a mandate to analyze all of the key pollutants affected by historical Clean Air Act programs, to estimate all of the significant endpoints associated with those pollutants, and to do so for a 20 year period covering the entire continental U.S., it was necessary to use simplified approaches for most of the pollutants to be analyzed. In several cases related to primary emissions —particularly sulfur dioxide (SO_2), nitrogen oxides (NO_x), and carbon monoxide (CO)— simple "roll-up model" strategies were adopted based on the expectation that changes in emissions of these pollutants would be highly correlated with subsequent changes in air quality.[29] Significant pollutants involving secondary atmospheric formation, nonlinear formation mechanisms, and/or long-range transport were analyzed using the best air quality model which was affordable given time and resource limitations. These models, discussed in detail in Appendix C, included the Ozone Isopleth Plotting with Optional Mechanism-IV (OZIPM4) model for urban ozone; various forms of the above-referenced RADM model for background ozone, acid deposition, sulfate, nitrate, and visibility effects in the eastern U.S.; and the SJVAQS/AUSPEX Regional Modeling Adaptation Project (SARMAP) Air Quality Model (SAQM) for rural ozone in California agricultural areas. In addition, a linear scaling approach was developed and implemented to estimate visibility changes in large southwestern U.S. urban areas.

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[27] Particles with an aerometric diameter of less than or equal to 10 microns.

[28] For example, ozone concentration-response data exists for effects associated with 1-hour, 2.5-hour, and 6.6-hour exposures.

By adopting simplified approaches for some pollutants, the air quality modeling step adds to the overall uncertainties and limitations of the present analysis. The limited expanse and density of the U.S. air quality monitoring network and the limited coverage by available air quality models of major geographic areas[30] further constrain the achievable scope of the present study. Under these circumstances, it is important to remember the extent and significance of gaps in geographic coverage for key pollutants when considering the overall results of this analysis. Key uncertainties are summarized at the end of this chapter in Table 5. More extensive discussion of the caveats and uncertainties associated with the air quality modeling step is presented in Appendix C. In addition, information regarding the specific air quality models used, the characteristics of the historical monitoring data used as the basis for the control scenario profiles, pollutant-specific modeling strategies and assumptions, references to key supporting documents, and important caveats and uncertainties are also presented in Appendix C.

General Methodology

The general methodological approach taken in this analysis starts with the assumption that actual historical air quality will be taken to represent the control scenario. This may seem somewhat inconsistent with the approach taken in earlier steps of the analysis, which used modeled macroeconomic conditions as the basis for estimating macroeconomic effects and emissions. However, the central focus of the overall analysis is to estimate the difference in cost and benefit outcomes between the control and no-control scenarios. It is consistent with this central paradigm to use actual historical air quality data as the basis for estimating how air quality might have changed in the absence of the Clean Air Act.

The initial step, then, for each of the five non-lead (Pb) criteria pollutants[31] was to compile comprehensive air quality profiles covering the entire analytical period from 1970 to 1990. The source for these data was EPA's Aerometric Information Retrieval System (AIRS), which is a publicly accessible database of historical air quality data. The vast number of air quality observations occurring over this twenty year period from the thousands of monitors in the U.S. indicates the need to represent these observations by statistical distributions. As documented in detail in the supporting documents covering SO_2, NO_x, CO, and ozone,[32] both lognormal and gamma distributional forms were tested against actual data to determine the form which provided the best fit to the historical data.[33] Based on these tests, one or the other statistical distribution was adopted for the air quality profiles developed for each pollutant. In addition to reducing the air quality data to a manageable form, this approach facilitated transformations of air quality profiles from one averaging period basis to another.

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[29] It is important to emphasize that the correlation expected is between changes in emissions and changes in air quality. Direct correlations between the absolute emissions estimates and empirical air quality measurements used in the present analysis may not be strong due to expected inconsistencies between the geographically local, monitor-proximate emissions densities affecting air quality data.

[30] For example, the regional oxidant models available for the present study do not cover some key Midwestern states, where human health, agricultural crop, and other effects from ozone may be significant.

[31] Lead (Pb), the sixth criteria pollutant, is analyzed separately. The ability to correlate emissions directly with blood lead levels obviates the need for using air quality modeling as an intermediate step toward estimation of exposure.

[32] See SAI SO_2, NO_x, and CO Report (1994) and SAI Ozone Report (1995).

Once the control scenario profiles based on historical data were developed, no-control scenarios were derived based on the results of the various air quality modeling efforts. Again, the focus of the overall analysis is to isolate the difference in outcomes between the control and no-control scenarios. The no-control scenario air quality profiles were therefore derived by adjusting the control scenario profiles upward (or downward) based on an appropriate measure of the difference in modeled air quality outcomes. To illustrate this approach, consider a simplified example where the modeled concentration of Pollutant A under the no-control scenario is 0.12 ppm, compared to a modeled concentration under the control scenario of 0.10 ppm. An appropriate measure of the difference between these outcomes, whether it is the 0.02 ppm difference in concentration or the 20 percent percentage differential, is then used to ratchet up the control case profile to derive the no-control case profile. Generally, the modeled differential is applied across the entire control case profile to derive the no-control case profile. As described below in the individual sections covering particulate matter and ozone, however, more refined approaches are used where necessary to take account of differential outcomes for component species (i.e., particulate matter), long-range transport, and background levels of pollutants.

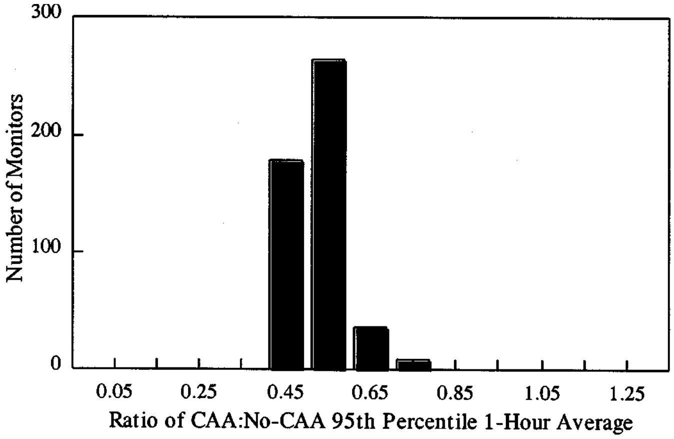
Sample Results

The results of the air quality modeling effort include a vast array of monitor-specific air quality profiles for particulate matter (PM_{10} and TSP),[34] SO_2, NO_2, NO, CO, and ozone; RADM grid cell-based estimates of sulfur and nitrogen deposition; and estimates of visibility degradation for eastern U.S. RADM grid cells and southwestern U.S. urban areas. All of these data were transferred to the effects modelers for use in configuring the human health, welfare, and ecosystem physical effects models. Given the massive quantity and intermediate nature of the air quality data, they are not exhaustively reported herein.[35] To provide the reader with some sense of the magnitude of the difference in modeled air quality conditions under the control and no-control scenarios, some illustrative results for 1990 are presented in this chapter and in Appendix C. In addition, maps depicting absolute levels of control and no-control scenario acid deposition and visibility are presented to avoid potential confusion which might arise through examination of percent change maps alone.[36]

Carbon Monoxide

Figure 8 provides an illustrative comparison of 1990 control versus no-control scenario CO concentrations, expressed as a frequency distribution of the ratios of 1990 control to no-control scenario 95th percentile 1-hour average concentrations at individual CO monitors. Consistent with the emission changes underlying these air quality results, CO concentrations under the control scenario tend to be about half those projected under the no-control scenario, with most individual monitor ratios ranging from about 0.40 to 0.60 percent, and a few with ratios in the 0.60 to 0.80 range.

Figure 8. Frequency Distribution of Estimated Ratios for 1990 Control to No-control Scenario 95th Percentile 1-Hour Average CO Concentrations, by Monitor.



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[33] The statistical tests used to determine goodness of fit are described in the SAI reports.

[34] PM data are reported as county-wide values for counties with PM monitors and a sufficient number of monitor observations.

[35] The actual air quality profiles, however, are available on disk from EPA. See Appendix C for further information.

[36] Large percentage changes can result from even modest absolute changes when they occur in areas with good initial (e.g., control scenario) air quality. Considering percentage changes alone might create false impressions regarding absolute changes in air quality in some areas. For example, Appendix C discusses in detail two such cases: the Upper Great Lakes and Florida-Southeast Atlantic Coast areas, which show high percentage changes in sulfur deposition and visibility.

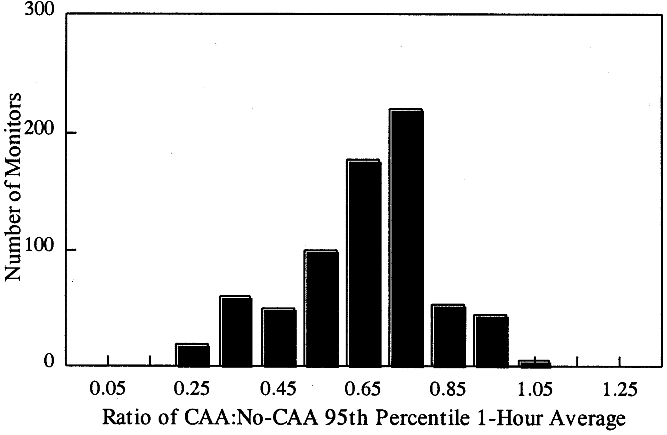
In considering these results, it is important to note that CO is essentially a "hot spot" pollutant, meaning that higher concentrations tend to be observed in localized areas of relatively high emissions. Examples of such areas include major highways, major intersections, and tunnels. Since CO monitors tend to be located in order to monitor the high CO concentrations observed in such locations, one might suspect that using state-wide emissions changes to scale air quality concentration estimates at strategically located monitors might create some bias in the estimates. However, the vast majority of ambient CO is contributed from on-highway vehicles. In addition, the vast majority of the change in CO emissions between the control and no-control scenario occurs due to catalyst controls on highway vehicles. Since CO hot spots result primarily from highway vehicles emissions, controlling such vehicles would mean CO concentrations would be commensurately lowered at CO monitors. While variability in monitor location relative to actual hot spots and other factors raise legitimate concerns about assuming ambient concentrations are correlated with emission changes at any given monitor, the Project Team believes that the results observed provide a reasonable characterization of the aggregate change in ambient CO concentrations between the two scenarios.

Sulfur Dioxide

As for CO, no-control scenario SO_2 concentrations were derived by scaling control scenario air quality profiles based on the difference in emissions predicted under the two scenarios. Unlike CO, SO_2 is predominantly emitted from industrial and utility sources. This means that emissions, and the changes in emissions predicted under the two scenarios, will tend to be concentrated in the vicinity of major point sources. Again, while state-wide emissions changes are used to scale SO_2 concentrations between the scenarios, these state-wide emission changes reflect the controls placed on these individual point sources. Therefore, the Project Team again considers the distribution of control to no-control ratios to be a reasonable characterization of the aggregate results despite the uncertainties associated with estimation of changes at individual monitors.

Figure 9 provides a histogram of the predicted control to no-control ratios for SO_2 which is similar to the one presented for CO. The results indicate that, on an overall basis, SO_2 concentrations were reduced by about one-third. The histogram also shows a much wider distribution of control to no-control ratios for individual monitors than was projected for CO. This result reflects the greater state to state variability in SO_2 emission changes projected in this analysis. This greater state to state variability in turn is a function of the variable responses of SO_2 point sources to historical C control requirements.[37] This source-specific variability was not observed for CO because controls were applied relatively uniformly on highway vehicles.

Figure 9. Frequency Distribution of Estimated Ratios for 1990 Control to No-control Scenario 95th Percentile 1-Hour Average SO_2 Concentrations, by Monitor.



Nitrogen Dioxide

Results for NO_2 are presented in Figure 10. These results are similar to the results observed for CO, and for a similar reason: the vast majority of change in NO_2 emissions between the two scenarios is related to control of highway vehicle emissions. While baseline emissions of NO_2 from stationary sources may be significant, these sources were subject to minimal controls during the historical period of this analysis. On an aggregated basis, overall NO_2 concentrations are estimated to be roughly one-third lower under the control scenario than under the no-control scenario.

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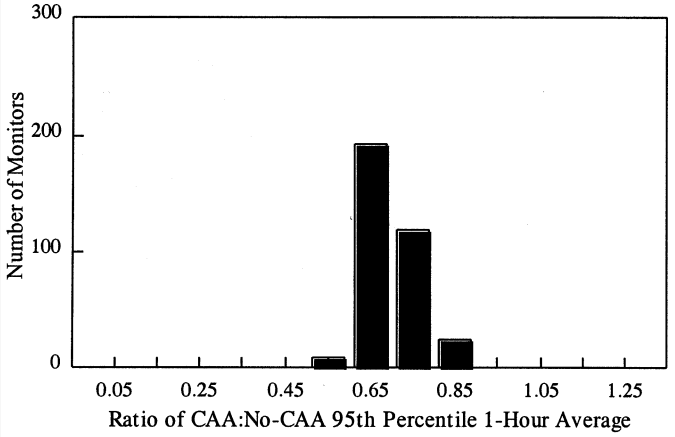
[37] Figure 9 indicates that six monitors were projected to have higher SO_2 concentrations for 1990 under the control scenario than under the no-control scenario. All six of these monitors are located in Georgia, a state for which higher 1990 utility SO_2 emissions are projected in the control scenario due to increased use of higher-sulfur coal. The projected increase in overall Georgia utility consumption of higher sulfur coal under the control case is a result of increased competition for the low-sulfur southern Appalachian coal projected to occur under the control scenario.

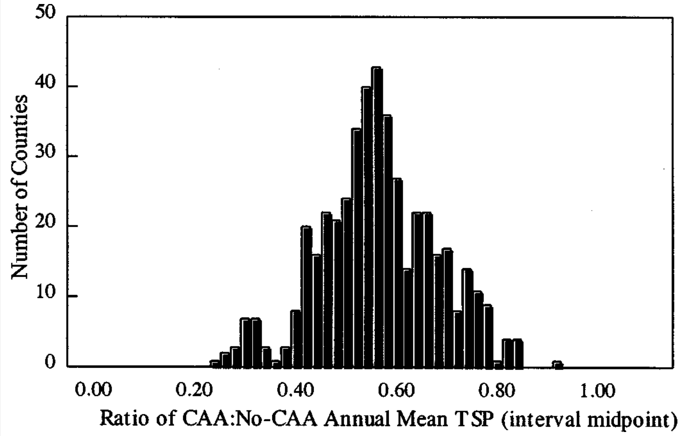
Figure 10. Frequency Distribution of Estimated Ratios for 1990 Control to No-control Scenario 95th Percentile 1-Hour Average NO_2 Concentrations, by Monitor.



Particulate Matter

An indication of the difference in outcomes for particulate matter between the two scenarios is provided by Figure 11. This graph shows the distribution of control to no-control ratios for annual mean TSP in 1990 for those counties which both had particulate monitors and a sufficient number of observations from those monitors.[38] While the distribution of results is relatively wide, reflecting significant county to county variability in ambient concentration, on a national aggregate basis particulate matter concentrations under the control scenario were just over half the level projected under the no-control scenario. The significant county to county variability observed in this case reflects point source-specific controls on particulate matter precursors, especially SO_2, and the effects of long-range transport and transformation.

Figure 11. Frequency Distribution of Estimated Ratios for 1990 Control to No-control Annual Mean TSP Concentrations, by Monitored County.

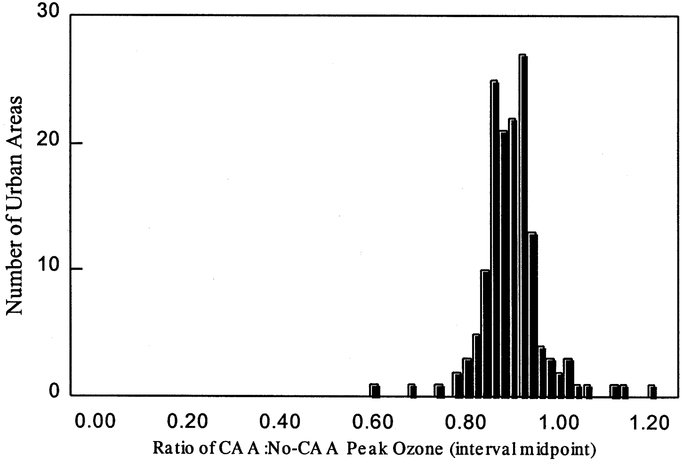


Ozone

Urban Ozone

Figure 12 presents a summary of the results of the 1990 OZIPM4 ozone results for all 147 of the modeled urban areas. In this case, the graph depicts the distribution of ratios of peak ozone concentrations estimated for the control and no-control scenarios. While the vast majority of simulated peak ozone concentration ratios fall below 1.00, eight urban areas show lower simulated peak ozone for the no-control scenario than for the control scenario. For these eight urban areas, emissions of precursors were higher under the no-control scenario; however, the high proportion of ambient NO_x compared to ambient non-methane organic compounds (NMOCs) in these areas results in a decrease in net ozone production in the vicinity of the monitor when NO_x emissions increase.[39]

Figure 12. Distribution of Estimated Ratios for 1990 Control to No-control OZIPM4 Simulated 1-Hour Peak Ozone Concentrations, by Urban Area.



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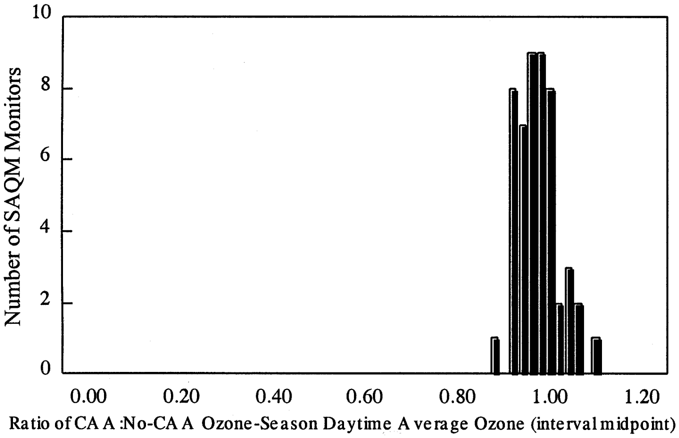
[38] Given the relative importance of particulate matter changes to the bottom line estimate of CAA benefits, and the fact that a substantial portion of the population lives in unmonitored counties, a methodology was developed to allow estimation of particulate matter benefits for these unmonitored counties. This methodology was based on the use of regional air quality modeling to interpolate between monitored counties. It is summarized in Appendix C and described in detail in the SAI PM Interpolation Report (1996).

[39] Over an unbounded geographic area, NO_x reductions generally decrease net ozone production.

Rural Ozone

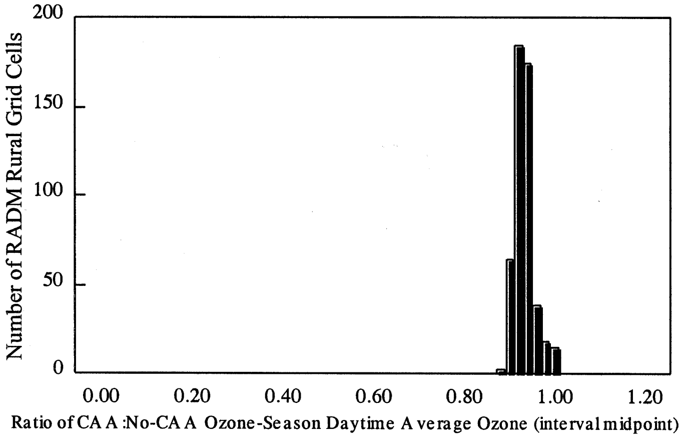
Figures 13 and 14 present frequency distributions for control to no-control ratios of average ozone-season daytime ozone concentrations at rural monitors as simulated by SAQM and RADM, respectively.

Figure 13. Distribution of Estimated Ratios for 1990 Control to No-control SAQM Simulated Daytime Average Ozone Concentrations, by SAQM Monitor.



Both the RADM and SAQM results indicate relatively little overall change in rural ozone concentrations. This is primarily because reductions in ozone precursor emissions were concentrated in populated areas.

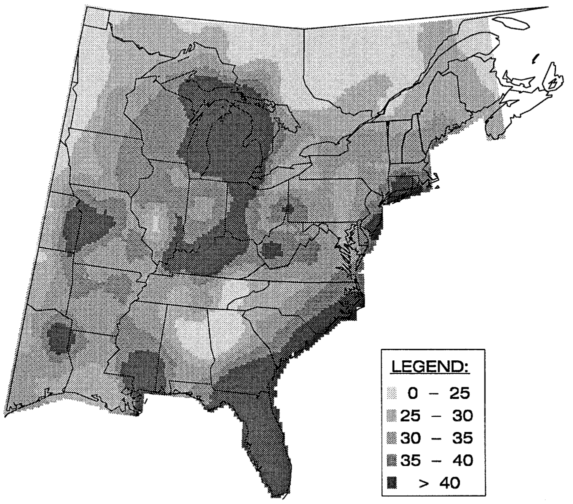
Figure 14. Distribution of Estimated Ratios for 1990 Control to No-control RADM Simulated Daytime Average Ozone Concentrations, by RADM Grid Cell.



Acid Deposition

Figure 15 is a contour map showing the estimated percent increase in sulfur deposition under the no-control scenario relative to the control scenario for 1990. Figure 16 provides comparable information for nitrogen deposition.

Figure 15. RADM-Predicted Percent Increase in Total Sulfur Deposition (Wet + Dry) Under the No-control Scenario.



These results show that acid deposition rates increase significantly under the no-control scenario, particularly in the Atlantic Coast area and in the vicinity of states for which relatively large increases in emissions are projected under the no-control scenario (i.e., Kentucky, Florida, Michigan, Mississippi, Connecticut, and Florida).

In the areas associated with large increases in sulfur dioxide emissions, rates of sulfur deposition increase to greater than or equal to 40 percent. The high proportional increase in these areas reflects both the significant increase in acid deposition precursor emissions in upwind areas and the relatively low deposition rates observed under the control scenario.[40]

Along the Atlantic Coast, 1990 nitrogen deposition rates increase by greater than or equal to 25 percent under the no-control scenario. This is primarily due to the significant increase in mobile source nitrogen oxide emissions along the major urban corridors of the eastern seaboard.

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[40] Even small changes in absolute deposition can yield large percentage changes when initial absolute deposition is low. See Appendix C for further discussion of this issue.

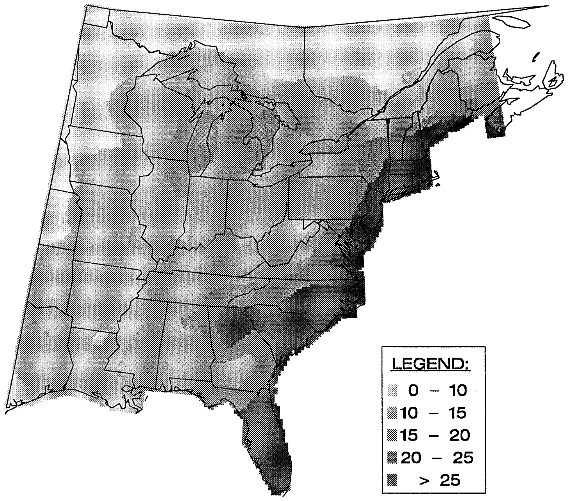

Figure 16. RADM-Predicted Percent Increase in Total Nitrogen Deposition (Wet + Dry) Under the No-control Scenario.

Visibility

The difference in modeled 1990 control and no-control scenario visibility conditions projected by the RADM/EM for the eastern U.S. is depicted by the contour map presented in Figure 17. This figure shows the increase in modeled annual average visibility degradation, in DeciView[41] terms, for 1990 when moving from the control to the no-control scenario. Since the DeciView metric is based on perceptible changes in visibility, these results indicate noticeable deterioration of visibility in the eastern U.S. underthe no-control scenario.

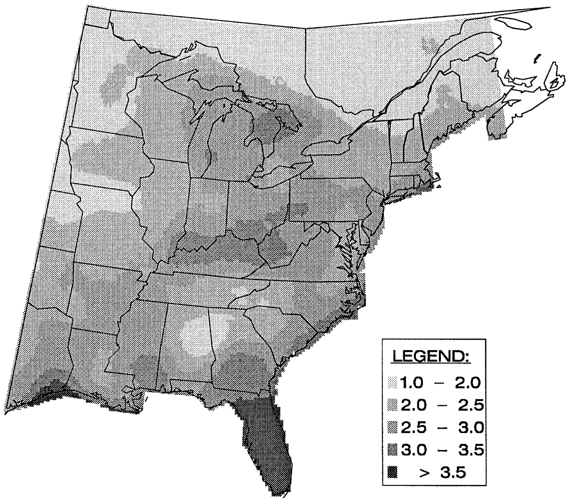

Figure 17. RADM-Predicted Percent Increase in Visibility Degradation, Expressed in DeciViews, for Poor Visibility Conditions (90th Percentile) Under the No-control Scenario.

Visibility changes in 30 southwestern U.S. urban areas were also estimated using emissions scaling techniques. This analysis also found significant, perceptible changes in visibility between the two scenarios. Details of this analysis, including the specific outcomes for the 30 individual urban areas, are presented in Appendix C.

Uncertainty in the Air Quality Estimates

Uncertainty prevades the projected changes in air quality presented in this study. These uncertainties arise due to potential inaccuracies in the emissions inventories used as air quality modeling inputs and due to potential errors in the structure and parameterization of the air quality models themselves. In addition, an important limitation of the present study is the lack of available data and/or modeling results for some pollutants in some regions of the country (e.g., visibility changes in western U.S. Class I areas such as the Grand Canyon). The inability to provide comprehensive estimates of changes in air quality due to the Clean Air Act creates a downward bias in the monetary benefit estimates.

The most important specific sources of uncertainty are presented in Table 5, and are described further in Appendix C. While the list of potential errors presented in Table 5 is not exhaustive, it incorporates the uncertainties with the greatest potential for contributing to error in the monetary benefit estimates. Overall, the uncertainties in the estimated change in air quality are considered small relative to uncertainties contributed by other components of the analysis.

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[41] The DeciView Haze Index (dV) is a relatively new visibility indicator aimed at measuring visibility changes in terms of human perception. It is described in detail in Appendix C.

Table 5. Key Uncertainties Associated with Air Quality Modeling.

Potential Source of Error	**Direction of Potential Bias in Estimate of Air Quality Benefits**	**Significance Relative to Key Uncertainties in Overall Monetary Benefit Estimate**
Use of OZIPM4 model, which does not capture long-range and night-time transport of ozone. Use of a regional oxidant model, such as UAM-V, would mitigate errors associated with neglecting transport.	Underestimate.	Significant, but probably not major. Overall average ozone response of 15% to NO_x and VOC reductions of approximately 30% and 45%, respectively. Even if ozone response doubled to 30%, estimate of monetized benefits of CAA will not change very much. Significant benefits of ozone reduction, however, could not be monetized.
Use of early biogenic emission estimates in RADM to estimate rural ozone changes in the eastern 31 states.	Underestimate.	Probably minor. Errors are estimated to be within -15% to +25% of the ozone predictions.
Use of proxy pollutants to scale up some particulate species in some areas. Uncertainty is created to the extent species of concern are not perfectly correlated with the proxy pollutants.	Unknown.	Potentially significant. Given the relative importance of the estimated changes in fine particle concentrations to the monetized benefit estimate, any uncertainty associated with fine particles is potentially significant. However, the potential error is mitigated to some extent since proxy pollutant measures are applied to both scenarios.
Use of state-wide average emission reductions to configure air quality models. In some cases, control programs may have been targeted to problem areas, so using state-wide averages would miss relatively large reductions in populated areas.	Underestimate.	Probably minor.
Exclusion of visibility benefits in Class I areas in the Southwestern U.S.	Underestimate.	Probably minor. No sensitivity analysis has been performed; however, monetized benefits of reduced visibility impairment in the Southwest would probably not significantly alter the estimate of monetized benefits.

Table 5 (con't). Key Uncertainties Associated with Air Quality Modeling.

Potential Source of Error	**Direction of Potential Bias in Estimate of Air Quality Benefits**	**Significance Relative to Key Uncertainties in Overall Monetary Benefit Estimate**
Lack of model coverage in western 17 states for acid deposition.	Underestimate.	Probably minor. No sensitivity analysis has been performed; however, monetized benefits of reduced acid deposition in the 17 western states would probably not significantly alter the estimate of monetized benefits.
Use of spatially and geographically aggregated emissions data to configure RADM. Lack of available day-specific meteorological data results in inability to account for temperature effects on VOCs and effect of localized meteorology around major point sources.	Unknown.	Potentially significant. Any effect which might influence the direction of long-range transport of fine particulates such as sulfates and nitrates could significantly influence the estimates of total monetized benefits of the CAA.
Use of constant concentration for organic aerosols between the two scenarios. Holding organic aerosol concentrations fixed omits the effect of changes in this constituent of fine particulate matter.	Underestimate.	Probably minor, because (a) nitrates were also held fixed and nitrates and organic aerosols move in opposite directions so the exclusion of both mitigates the effect of omitting either, (b) sulfates are by far the dominant species in the eastern U.S., and (c) larger errors would be introduced by using emissions scaling to estimate changes in organic aerosols since a significant fraction of organic aerosols are caused by biogenic gas-phase VOC emissions which do not change between the scenarios.
Unavailability of ozone models for rural areas outside the RADM and SAQM domains.	Underestimate.	Probably minor. Misses potential human health, welfare, and ecological benefits of reducing rural ozone in agricultural and other rural areas; however, ozone changes are likely to be small given limited precursor reductions in rural areas. RADM control:no-control ratios are in fact, relatively small.
Use of peak episode changes to estimate changes in annual distribution of ozone concentration.	Unknown.	Probably minor, particularly since relative changes in ozone concentration between the scenarios were small.

5

Physical Effects

Human Health and Welfare Effects Modeling Approach

This chapter identifies and, where possible, estimates the principal health and welfare benefits enjoyed by Americans due to improved air quality resulting from the CAA. Health benefits have resulted from avoidance of air pollution-related health effects, such as premature mortality, respiratory illness, and heart disease. Welfare benefits accrued where improved air quality averted damage to measurable resources, including agricultural production and visibility. The analysis of physical effects required a combination of three components: air quality, population, and health or welfare effects. As structured in this study, the 3-step process involved (1) estimating changes in air quality between the control and no-control scenarios, (2) estimating the human populations and natural resources exposed to these changed air quality conditions, and (3) applying a series of concentration-response equations which translated changes in air quality to changes in physical health and welfare outcomes for the affected populations.

Air Quality

The Project Team first estimated changes in concentrations of criteria air pollutants between the control scenario, which at this step was based on historical air quality, and the no-control scenario. Air quality improvements resulting from the Act were evaluated in terms of both their temporal distribution from 1970 to 1990 and their spatial distribution across the 48 conterminous United States. Generally, air pollution monitoring data provided baseline ambient air quality levels for the control scenario. Air quality modeling was used to generate estimated ambient concentrations for the no-control scenario. A variety of modeling techniques was applied, depending on the pollutant modeled. These modeling approaches and results are summarized in Chapter 4 and presented in detail in Appendix C.

Population

Health and some welfare benefits resulting from air quality improvements were distributed to individuals in proportion to the reduction in exposure. Predicting individual exposures, then, was a necessary step in estimating health effects. Evaluating exposure changes for the present analysis required not only an understanding of where air quality improved as a result of the CAA, but also how many individuals were affected by varying levels of air quality improvements. Thus, a critical component of the benefits analysis required that the distribution of the U.S. population nationwide be established.

Three years of U.S. Census data were used to represent the geographical distribution of U.S. residents: 1970, 1980, and 1990. Population data was supplied at the census block group level, with approximately 290,000 block groups nationwide. Allocating air quality improvements to the population for the other target years of this study – 1975 and 1985 – necessitated interpolation of the three years of population data. Linear interpolation was accomplished for each block group in order to maintain the variability in growth rates throughout the country.

Health and Welfare Effects

Benefits attributable to the CAA were measured in terms of the avoided incidence of physical health effects and measured welfare effects. To quantify such benefits, it was necessary to identify concentration-response relationships for each effect being considered. As detailed in Appendix D, such relationships were derived from the published science literature. In the case of health effects, concentration-response functions combined the air quality improvement and population distribution data with estimates of the number of fewer individuals that suffer an adverse health effect per unit change in air quality. By evaluating each concentration-response function for every monitored location throughout the country, and aggregating the resulting incidence estimates, it was possible to generate national estimates of incidence under the control and no-control scenarios.

In performing this step of the analysis, the Project Team discovered that it was impossible to estimate all of the health and welfare benefits which have resulted from the Clean Air Act. While scientific information was available to support estimation of some effects, many other important health and welfare effects could not be estimated. Furthermore, even though some physical effects could be quantified, the state of the science did not support assessment of the economic value of all of these effects. Table 6 shows the health effects for which quantitative analysis was prepared, as well as some of the health effects which could not be quantified in the analysis. Table 7 provides similar information for selected welfare effects.

While the 3-step analytical process described above was applied for most pollutants, health effects for lead were evaluated using a different methodology. Gasoline as a source of lead exposure was addressed separately from conventional point sources. Instead of using ambient concentrations of lead resulting from use of leaded gasoline, the concentration-response functions linked changes in lead releases directly to changes in the population's mean blood lead level. The amount of leaded gasoline used each year was directly related to mean blood lead levels using a relationship described in the 1985 Lead Regulatory Impact Analysis (U.S. EPA, 1985). Health effects resulting from exposure to point sources of atmospheric lead, such as industrial facilities, were considered using the air concentration distributions modeled around these point sources. Concentration-response functions were then used to estimate changes in blood lead levels in nearby populations.

Most welfare effects were analyzed using the same basic 3-step process used to analyze health effects, with one major difference in the concentration-response functions used. Instead of quantifying the relationship between a given air quality change and the number of cases of a physical outcome, welfare effects were measured in terms of the avoided resource losses. An example is the reduction in agricultural crop losses resulting from lower ambient ozone concentrations under the control scenario. These agricultural benefits were measured in terms of net economic surplus.

Another important welfare effect is the benefit accruing from improvements in visibility under the control scenario. Again, a slightly different methodological approach was used to evaluate visibility improvements. Visibility changes were a direct output of the models used to estimate changes in air quality.[42] The models provided estimates of changes in light extinction, which were then translated mathematically into various specific measures of perceived visibility change.[43] These visibility change measures were then combined with population data to estimate the economic value of the visibility changes. Other welfare effects quantified in terms of avoided resource losses include household soiling damage by PM_{10} and decreased worker productivity due to ozone exposure. The results of the welfare effects analysis are found in Chapter 6 and in Appendices D and F.

Because of a lack of available concentration-response functions (or a lack of information concerning affected populations), ecological effects were not quantified for this analysis. However, Appendix E provides discussion of many of the important ecological benefits which may have accrued due to historical implementation of the CAA.

Key Analytical Assumptions

Several important analytical assumptions affect the confidence which can be placed in the results of the physical effects analysis. The most important of these assumptions relate to (a) mapping of potentially exposed populations to the ambient air quality monitoring network, (b) choosing among competing scientific studies in developing quantitative estimates of physical effects, (c) quantifying the contribution to analytical uncertainty of within-study variances in effects estimates and, perhaps most important in the context of the present study, (d) estimating particulate matter-related mortality based on the currently available scientific literature.

Because these resultant uncertainties were caused by the inadequacy of currently available scientific information, there is no compelling reason to believe

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[42] These models, and the specific visibility changes estimated by these models, are described in summary fashion in the previous chapter and are discussed in detail in Appendix C.

[43] These visibility measures are described in Appendix C.

Table 6. Human Health Effects of Criteria Pollutants.

Pollutant	Quantified Health Effects	Unquantified Health Effects	Other Possible Effects
Ozone	Respiratory symptoms Minor restricted activity days Respiratory restricted activity days Hospital admissions Emergency room visits Asthma attacks Changes in pulmonary function Chronic Sinusitis & Hay Fever	Increased airway responsiveness to stimuli Centroacinar fibrosis Inflammation in the lung	Immunologic changes Chronic respiratory diseases Extrapulmonary effects (e.g., changes in structure, function of other organs) Reduced UV-B exposure attenuation
Particulate Matter/ TSP/ Sulfates	Mortality* Bronchitis - Chronic and Acute Hospital admissions Lower respiratory illness Upper respiratory illness Chest illness Respiratory symptoms Minor restricted activity days All restricted activity days Days of work loss Moderate or worse asthma status (asthmatics)	Changes in pulmonary function	Chronic respiratory diseases other than chronic bronchitis Inflammation in the lung
Carbon Monoxide	Hospital Admissions - congestive heart failure Decreased time to onset of angina	Behavioral effects Other hospital admissions	Other cardiovascular effects Developmental effects
Nitrogen Oxides	Respiratory illness	Increased airway responsiveness	Decreased pulmonary function Inflammation in the lung Immunological changes
Sulfur Dioxide	In exercising asthmatics: Changes in pulmonary function Respiratory symptoms Combined responses of respiratory symptoms and pulmonary function changes		Respiratory symptoms in non-asthmatics Hospital admissions
Lead	Mortality Hypertension Non-fatal coronary heart disease Non-fatal strokes IQ loss effect on lifetime earnings IQ loss effects on special education needs	Health effects for individuals in age ranges other than those studied Neurobehavioral function Other cardiovascular diseases Reproductive effects Fetal effects from maternal exposure Delinquent and anti-social behavior in children	

* This analysis estimates excess mortality using PM as an indicator of the pollutant mix to which individuals were exposed.

Table 7. Selected Welfare Effects of Criteria Pollutants.

Pollutant	**Quantified Welfare Effects**	**Unquantified Welfare Effects**
Ozone	Changes in crop yields (for 7 crops) Decreased worker productivity	Changes in other crop yields Materials damage Effects on forests Effects on wildlife
Particulate Matter/ TSP/ Sulfates	Household soiling Visibility	Other materials damage Effects on wildlife
Nitrogen Oxides	Visibility	Crop losses due to acid deposition Materials damage due to acid deposition Effects on fisheries due to acidic deposition Effects on forests
Sulfur Dioxide	Visibility	Crop losses due to acid deposition Materials damage due to acid deposition Effects on fisheries due to acidic deposition Effects on forests

that the results of the present analysis are biased in a particular direction. Some significant uncertainties, however, may have arisen from interpretation of model results, underlying data, and supporting scientific studies. These assumptions and uncertainties are characterized in this report to allow the reader to understand the degree of uncertainty and the potential for misestimation of results. In addition, the overall results are presented in ranges to reflect the aggregate effect of uncertainty in key variables. A quantitative assessment of some of the uncertainties in the present study is presented in Chapter 7. In addition, the key uncertainties in the physical effects modeling step of this analysis are summarized in Table 12 at the end of this chapter. The remainder of this section discusses each of the four critical modeling procedures and associated assumptions.

Mapping Populations to Monitors

The Project Team's method of calculating benefits of air pollution reductions required a correlation of air quality data changes to exposed populations. For pollutants with monitor-level data (i.e., SO_2, O_3, NO_2, CO), it was assumed that all individuals were exposed to air quality changes estimated at the nearest monitor. For PM_{10}, historical air quality data were available at the county level. All individuals residing in a county were assumed to be exposed to that county's PM_{10} air quality.[44]

Many counties did not contain particulate matter air quality monitors or did not have a sufficient number of monitor observations to provide reliable estimates of air quality. For those counties, the Project Team conducted additional analyses to estimate PM_{10} air quality changes during the study period. For counties in the eastern 31 states, the grid cell-specific sulfate particle concentrations predicted by the RADM model were used to provide a scaled interpolation between monitored counties.[45] For counties outside the RADM domain, an alternative method based on state-wide average concentrations was used. With this supplemental analysis, estimates were developed of the health effects of the CAA on almost the entire continental U.S. population.[46] Compliance costs incurred in Alaska and Hawaii were included in this study, but the benefits of historical air pollution reductions were not. In addition, the CAA yielded benefits to Mexico and Canada that were not captured in this study.

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[44] In some counties and in the early years of the study period, particulate matter was monitored as TSP rather than as PM_{10}. In these cases, PM_{10} was estimated by applying TSP:PM_{10} ratios derived from historical data. This methodology is described in Appendix C.

[45] The specific methodology is described in detail in Appendix C.

[46] While this modeling approach captures the vast majority of the U.S. population, it does not model exposure for everyone. To improve computational efficiency, those grid cells with populations less than 500 were not modeled; thus, the analysis covered somewhat more than 97 percent of the population.

Air quality monitors are more likely to be found in high pollution areas rather than low-pollution areas. Consequently, mapping population to the nearest monitor regardless of the distance to that monitor almost certainly results in an overstatement of health impacts due to air quality changes for those populations. The Project Team conducted a sensitivity analysis to illustrate the importance of the "mapping to nearest monitor" assumption. For comparison to the base case, which modeled exposure for the 48 state population, Table 8 presents the percentage of the total 48-state population covered in the "50 km" sensitivity scenario. For most pollutants in most years, 25 percent or more of the population resided more than 50 km from an air quality monitor (or in a county without PM_{10} monitors). Estimated health benefits are approximately linear to population covered — that is, if the population modeled for a pollutant in a given year in the sensitivity analysis is 25 percent smaller than the corresponding population modeled in the base case, then estimated health benefits are reduced by roughly 25 percent in the sensitivity case. This sensitivity analysis demonstrates that limiting the benefits analysis to reflect only those living within 50 km of a monitor or within a PM-monitored county would lead to a substantial underestimate of the historical benefits of the CAA. Since these alternative results may have led to severely misleading comparisons of the costs and benefits of the Act, the Project Team decided to adopt the full 48-state population estimate as the central case for this analysis despite the greater uncertainties and potential biases associated with estimating exposures from distant monitoring sites.

Table 8. Percent of Population (of the Continental US) within 50km of a monitor (or in a County with PM monitors), 1970-1990.

	Pollutant				
Year	**PM_{10}**	**O_3**	**NO_2**	**SO_2**	**CO**
1975	79%	56%	53%	65%	67%
1980	80%	71%	59%	73%	68%
1985	75%	72%	61%	73%	68%
1990	68%	74%	62%	71%	70%

Choice of Study

The Project Team relied on the most recent available, published scientific literature to ascertain the relationship between air pollution and human health and welfare effects. The choice of studies, and the uncertainties underlying those studies, also created uncertainties in the results. For example, to the extent the published literature may collectively overstate the effects of pollution, EPA's analysis will overstate the effects of the CAA. Such outcomes may occur because scientific research which fails to find significant relationships is less likely to be published than research with positive results. On the other hand, history has shown that it is highly likely that scientific understanding of the effects of air pollution will improve in the future, resulting in discovery of previously unknown effects. Important examples of this phenomenon are the substantial expected health and welfare benefits of reductions in lead and ambient particulate matter, both of which have been shown in recent studies to impose more severe effects than scientists previously believed. To the extent the present analysis misses effects of air pollution that have not yet been subject to adequate scientific inquiry, the analysis may understate the effects of the CAA.

For some health endpoints, the peer-reviewed scientific literature provides multiple, significantly differing alternative CR functions. In fact, it is not unusual for two equally-reputable studies to differ by a factor of three or four in implied health impact. The difference in implied health effects across studies can be considered an indication of the degree of scientific uncertainty associated with measurement of that health effect. Where more than one acceptable study was available, the Project Team used CR functions from all relevant studies to infer health effects. That is, the health effect implied by each study is reported (see Appendix D), and a range of reported results for a particular health endpoint can be interpreted as a measure of the uncertainty of the estimate.

Variance Within Studies

Even where only one CR function was available for use, the uncertainty associated with application of that function to estimate physical outcomes can be evaluated quantitatively. Health effects studies provided "best estimates" of the relationship between air quality changes and health effects, and a measure of the statistical uncertainty of the relationship. In this analysis, the Project Team used simulation modeling techniques to evaluate the overall uncertainty of the results given uncertainties within individual studies, across studies examining a given endpoint, and in the economic valuation coefficients applied to each endpoint. The analysis estimating aggregate quantitative uncertainty is presented in Chapter 7.

PM-Related Mortality

The most serious human health impact of air pollution is an increase in incidences of premature mortality. In the present study, excess premature mortality is principally related to increased exposure to lead (Pb)[47] and to particulate matter (PM) and associated non-Pb criteria pollutants.[48] With respect to PM, a substantial body of published health science literature recognizes a correlation between elevated PM concentrations and increased mortality rates. However, there is a diversity of opinion among scientific experts regarding the reasonableness of applying these studies to derive quantitative estimates of premature mortality associated with exposure to PM. While 19 of 21 members of the Science Advisory Board Clean Air Act Scientific Advisory Committee agree that present evidence warrants concern and implementation of a fine particle ($PM_{2.5}$) standard to supplement the PM_{10} standard, they also point out that the causal mechanism has not been clearly established.

For the purposes of the present study, the Project Team has concluded that the well-established correlation between exposure to elevated PM and premature mortality is sufficiently compelling to warrant an assumption of a causal relationship and derivation of quantitative estimates of a PM-related premature mortality effect. In addition to the assumption of causality, a number of other factors contribute to uncertainty in the quantitative estimates of PM-related mortality.[49] First, although there is uncertainty regarding the shape of the CR functions derived from the epidemiological studies, the present analysis assumes the relationship to be linear throughout the relevant range of exposures. Second, there is significant variability among the underlying studies which may reflect, at least in part, location-specific differences in CR functions. Transferring CR functions derived from one or more specific locations to all other locations may contribute significantly to uncertainty in the effect estimate. Third, a number of potentially significant biases and uncertainties specifically associated with each of the two types of PM-related mortality study further contribute to uncertainty. The remainder of this section discusses these two groups of studies and their attendant uncertainties and potential biases. (See Appendix D for a more complete discussion of these studies and their associated uncertainties.)

Short-Term Exposure Studies

Many of the studies examining the relationship between PM exposure and mortality evaluate changes in mortality rates several days after a period of elevated PM concentrations. In general, significant correlations have been found. These "short-term exposure" or "episodic" studies are unable to address two important issues: (1) the degree to which the observed excess mortalities are "premature," and (2) the degree to which daily mortality rates are correlated with long-term exposure to elevated PM concentrations (i.e., exposures over many years rather than a few days).

Because the episodic mortality studies evaluate the mortality rate impact only a few days after a high-pollution event, it is likely that many of the "excess mortality" cases represented individuals who were already suffering impaired health, and for whom the high-pollution event represented an exacerbation of an already serious condition. Based on the episodic studies only, however, it is unknown how many of the victims would have otherwise lived only a few more days or weeks, or how many would have recovered to enjoy many years of a healthy life in the absence of the high-pollution event. For the purpose of cost-benefit analysis, it can be important to determine whether a pollution event reduces the average lifespan by several days or by many years. Although the episodic mortality studies do not provide an estimate of the expected life years lost (nor do they address the health status of victims), some have evaluated the age of the excess premature mortality cases, and have estimated that 80 to 85 percent of the victims are age 65 or older.

In addition to causing short-term health problems, air pollution (measured by elevated annual PM concentrations) can cause longer-term health problems that may lead to premature mortality. Such long-term changes in susceptibility to premature mortality in the future will be missed by efforts to correlate premature mortalities with near-term episodes of elevated pollution concentrations. Consequently, excess premature mortality estimates based on the results of the "episodic" mortality studies will underestimate the effect of long-term elevated pollution concentrations on mortality rates.

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[47] Detailed information on methods, sources, and results of the Pb mortality analysis are presented in Appendix G.

[48] PM concentrations are highly correlated with concentrations of other criteria pollutants. It is difficult to determine which pollutant is the causative factor in elevated mortality rates. In this study, the Project Team has used PM as a surrogate for a mix of criteria pollutants.

[49] It should also be noted that some of the morbidity studies, most notably the PM/chronic bronchitis epidemiological studies, involve many of the same uncertainties.

Long-Term Exposure Studies

The other type of PM-related mortality study involves examination of the potential relationship between long-term exposure to PM and annual mortality rates. These studies are able to avoid some of the weaknesses of the episodic studies. In particular, by investigating changes in annual (rather than daily) mortality rates, the long-term studies do not predict most cases of excess premature mortality where mortality is deferred for only a few days; also, the long-term studies are able to discern changes in mortality rates due to long-term exposure to elevated air pollution concentrations. Additionally, the long-term exposure studies are not limited to measuring mortalities that occur within a few days of a high-pollution event. Consequently, use of the results of the long-term studies is likely to result in a more complete assessment of the effect of air pollution on mortality risk.

The long-term exposure studies, however, have some significant limitations and potential biases. Although studies that are well-executed attempt to control for those factors that may confound the results of the study, there is always the possibility of insufficient or inappropriate adjustment for those factors that affect long-term mortality rates and may be confounded with the factor of interest (e.g., PM concentrations). Prospective cohort studies have an advantage over ecologic, or population-based, studies in that they gather individual-specific information on such important risk factors as smoking. It is always possible, however, that a relevant, individual-specific risk factor may not have been controlled for or that some factor that is not individual-specific (e.g., climate) was not adequately controlled for. It is therefore possible that differences in mortality rates that have been ascribed to differences in average PM levels may be due, in part, to some other factor or factors (e.g., differences among communities in diet, exercise, ethnicity, climate, industrial effluents, etc.) that have not been adequately controlled for.

Another source of uncertainty surrounding the prospective cohort studies concerns possible historical trends in PM concentrations and the relevant period of exposure, which is as yet unknown. TSP concentrations were substantially higher in many locations for several years prior to the cohort studies and had declined substantially by the time these studies were conducted. If this is also true for $PM_{2.5}$ and PM_{10}, it is possible that the larger PM coefficients reported by the long-term exposure studies (as opposed to the short-term exposure studies) reflect an upward bias. If the relevant exposure period extends over a decade or more, then a coefficient based on PM concentrations at the beginning of the study or in those years immediately prior to the study could be biased upward if pollution levels had been decreasing markedly for a decade or longer prior to the study.

On the other hand, if a downward trend in PM concentrations continued throughout the period of the study, and if a much shorter exposure period is relevant (e.g., contained within the study period itself), then characterizing PM levels throughout the study by those levels just prior to the study would tend to bias the PM coefficient downward. Suppose, for example, that PM levels were converging across the different study locations over time, and in particular, into the study period. (That is, suppose PM levels were decreasing over time, but decreasing faster in the high-PM locations than in the low-PM locations, so that at the beginning of the study period the interlocational differences in PM concentrations were smaller than they were a decade earlier.) Suppose also that the relevant exposure period is about one year, rather than many years. The Pope study characterizes the long-term PM concentration in each of the study locations by the median PM concentration in the location during the five year period 1979-1983. Study subjects were followed, however, from 1982 through 1989. If the difference in median PM concentrations across the 50 study locations during the period 1979-1983 was greater than the difference during the period 1983-1988, and if it is PM levels during the period 1983-1988 that most affect premature mortality during the study period (rather than PM levels during the period 1979-1983), then the study would have attributed interlocational differences in mortality to larger interlocational differences in PM concentrations than were actually relevant. This would result in a downward bias of the PM coefficient estimated in the study.

The relevant exposure period is one of a cluster of characteristics of the mortality-PM relationship that are as yet unknown and potentially important. It is also unknown whether there is a time lag in the PM effect. Finally, it is unknown whether there may be cumulative effects of chronic exposure — that is, whether the relative risk of mortality actually increases as the period of exposure increases.

Three recent studies have examined the relationship between mortality and long-term exposure to PM: Pope et al. (1995), Dockery et al. (1993), and Abbey et al. (1991). The Pope et al. study is considered a better choice of long-term exposure study than either of the other two studies. Pope et al. examined a much larger population and many more locations than either the Dockery study or the Abbey study. The Dockery study covered only six cities. The Abbey study covered a cohort of only 6,000 people in California. In particular, the cohort in the Abbey study was considered substantially too small and too young to enable the detection of small increases in mortality risk. The study was therefore omitted from consideration in this analysis. Even though Pope et al. (1995) reports a smaller premature mortality response to elevated PM than Dockery et al. (1993), the results of the Pope study are nevertheless consistent with those of the Dockery study.

Pope et al., (1995) is also unique in that it followed a largely white and middle class population, decreasing the likelihood that interlocational differences in premature mortality were attributable to differences in socioeconomic status or related factors. Furthermore, the generally lower mortality rates and possibly lower exposures to pollution among this group, in comparison to poorer minority populations, would tend to bias the PM coefficient from this study downward, counteracting a possible upward bias associated with historical air quality trends discussed above.

Another source of downward bias in the PM coefficient in Pope et al., (1995) is that intercity movement of cohort members was not considered. Migration across study cities would result in exposures of cohort members being more similar than would be indicated by assigning city-specific annual average pollution levels to each member of the cohort. The more intercity migration there is, the more exposure will tend toward an intercity mean. If this is ignored, differences in exposure levels, proxied by differences in city-specific annual average PM levels, will be exaggerated, resulting in a downward bias of the PM coefficient. This is because a given difference in mortality rates is being associated with a larger difference in PM levels than is actually the case.

An additional source of uncertainty in the Pope et al., study arises from the PM indicator used in the study. The Pope et al. study examined the health effects associated with two indices of PM exposure; sulfate particles and fine particles ($PM_{2.5}$). The $PM_{2.5}$ relationship is used in this analysis because it is more consistent with the air quality data selected for this analysis (PM_{10}). Because we use a $PM_{2.5}$ mortality relationship, air quality profiles were developed from the PM_{10} profiles generated for the entire 20 year period. The same regional information about the PM_{10} components (sulfate, nitrate, organic particulate and primary particulate) used to develop the PM_{10} profiles was used to develop regional $PM_{2.5}/PM_{10}$ ratios. Although both urban and rural ratios are available, for computational simplicity, only the regional urban ratios were used to estimate the $PM_{2.5}$ profiles from the PM_{10} profiles used in the analysis. This reflects the exposure of the majority of the modeled population (i.e., the urban population), while introducing some error in the exposure changes for the rural population. In the east and west, where the rural ratio is larger than the urban ratio, the change in $PM_{2.5}$ exposure will be underestimated for the rural population. In the central region the $PM_{2.5}$ change will be overestimated. These ratios were used in each year during 1970-1990, introducing another source of uncertainty in the analysis.

After considering the relative advantages and disadvantages of the various alternative studies available in the peer-reviewed literature, the Project Team decided that the long-term exposure studies were preferable for the purposes of the present study, primarily because the long-term exposure studies appear to provide a more comprehensive estimate of the premature mortality incidences attributable to PM exposure. Among the long-term exposure studies, the Pope et al., (1995) study appears more likely to mitigate a key source of potential confounding. For these reasons, the CR function estimated in Pope et al., (1995) is considered the most reasonable choice for this analysis and is utilized in spite of the several important residual uncertainties and potential biases which are subsequently reflected in the PM-related mortality effect estimate.

Health Effects Modeling Results

This section provides a summary of the differences in health effects estimated under the control and no-control scenarios. Because the differences in air quality between the two scenarios generally increased from 1970 to 1990, and the affected population grew larger during that period, the beneficial health effects of the CAA increased steadily during the 1970 to 1990 period. More detailed results are presented in Appendix D.

Avoided Premature Mortality Estimates

The Project Team determined that, despite their limitations, the long-term particulate matter exposure studies provided the superior basis for estimating mortality effects for the purpose of benefit-cost analysis. Three prospective cohort studies were identified (Pope et al. (1995), Dockery et al. (1993), and Abbey et al. (1991)), although the Abbey study was omitted from consideration because the cohort in that study was considered insufficient to allow the detection of small increases in mortality risk. Exposure-response relationships inferred from the Pope et al. study were used in the health benefits model to estimate avoided mortality impacts of the CAA. The Pope et al. study was selected because it is based on a much larger population and a greater number of communities (50) than is the six-city Dockery et al. Study. The results of the Pope et al. are consistent with those of the other study, and are consistent with earlier ecological population mortality studies. See Appendix D for additional discussion of the selection of mortality effects studies.

Table 9 presents estimated avoided excess premature mortalities for 1990 only, with the mean estimate and 90 percent confidence interval. See Appendix D for more detail on results implied by individual epidemiological studies, and on the temporal pattern of impacts.[50] The model reports a range of results for each health endpoint. Here, the fifth percentile, mean, and ninety-fifth percentile estimates are used to characterize the distribution. The total number of avoided cases of premature mortality due to reduced exposure to lead (Pb) and particulate matter are presented. Additionally, avoided mortality cases are listed by age cohort of those who have avoided premature mortality in 1990, along with the expected remaining lifespan (in years) for the average person in each age cohort. The average expected remaining lifespan across all age groups is also indicated. These averages might be higher if data were available for PM-related mortality in the under 30 age group and for Pb-related mortality in the 5-39 age group.

Table 9. Criteria Pollutants Health Benefits -- Distributions of 1990 Avoided Premature Mortalities (thousands of cases reduced) for 48 State Population.

Pollutant	Age group	Remaining Life Expectancy (yrs)	Annual Cases Avoided (thousands)		
			5th %ile	Mean	95th %ile
$PM_{2.5}$	30 and over		112	184	257
	30-34	48	2	3	5
	35-44	38	5	8	11
	45-54	29	7	11	15
	55-64	21	14	23	33
	65-74	14	26	43	62
	75-84	9	32	54	76
	>84	6	24	41	59
		Avg.: 14*			
Lead	all ages		7	22	54
	infants	75	5	5	5
	40-44	38	0	2	13
	45-54	29	0	4	20
	55-64	21	0	6	18
	65-74	14	0	4	15
		Avg.: 38*			
TOTAL			166	205	252

*Averages calculated from proportions of premature mortalities by age group, from Table D-14.

Non-Fatal Health Impacts

The health benefits model reports non-fatal health effects estimates similarly to estimates of premature mortalities: as a range of estimates for each quantified health endpoint, with the range dependent on the quantified uncertainties in the underlying concentration-response functions. The range of results for 1990 only is characterized in Table 10 with fifth percentile, mean, and ninety-fifth percentile estimates. All estimates are expressed as thousands of new cases avoided in 1990. "Lost IQ Points" represent the aggregate number of points (in thousands) across the population affected by lead concentrations in 1990. All "Hospital Admissions" estimates are in thousands of admissions, regardless of the length of time spent in the hospital. "Shortness of breath" is expressed as thousands of

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[50] Earlier years are estimated to have had fewer excess premature mortalities.

Table 10. Criteria Pollutants Health Benefits -- Distributions of 1990 Non-Fatal Avoided Incidence (thousands of cases reduced) for 48 State Population.

Endpoint	Pollutant(s)	Affected Population (age group)	Annual Effects Avoided (thousands)			Unit
			5th %ile	Mean	95th %ile	
Chronic Bronchitis	PM	all	493	674	886	cases
Lost IQ Points	Lead	children	7,440	10,400	13,000	points
IQ < 70	Lead	children	31	45	60	cases
Hypertension	Lead	men 20-74	9,740	12,600	15,600	cases
Chronic Heart Disease	Lead	40-74	0	22	64	cases
Atherothrombotic brain infarction	Lead	40-74	0	4	15	cases
Initial cerebrovascular accident	Lead	40-47	0	6	19	cases
Hospital Admissions						
All Respiratory	PM & O3	all	75	89	103	cases
COPD + Pneumonia	PM & O3	over 65	52	62	72	cases
Ischemic Heart Disease	PM	over 65	7	19	31	cases
Congestive Heart Failure	PM & CO	65 and over	28	39	50	cases
Other Respiratory-Related Ailments						
Shortness of breath, days	PM	children	14,800	68,800	133,000	days
Acute Bronchitis	PM	children	0	8,700	21,600	cases
Upper & Lower Resp. Symptoms	PM	children	5,400	9,500	13,400	cases
Any of 19 Acute Symptoms	PM & O3	18-65	15,400	130,000	244,000	cases
Asthma Attacks	PM & O3	asthmatics	170	850	1,520	cases
Increase in Respiratory Illness	NO2	all	4,840	9,800	14,000	cases
Any Symptom	SO2	asthmatics	26	264	706	cases
Restricted Activity and Work Loss Days						
MRAD	PM & O3	18-65	107,000	125,000	143,000	days
Work Loss Days (WLD)	PM	18-65	19,400	22,600	25,600	days

The following additional welfare benefits were quantified directly in economic terms: household soiling damage, visibility, decreased worker productivity, and agricultural benefits (measured in terms of net surplus).

days: that is, one "case" represents one child experiencing shortness of breath for one day. Likewise, "Restricted Activity Days" and "Work Loss Days" are expressed in person-days.

Other Physical Effects

Human health impacts of criteria pollutants dominate quantitative analyses of the effects of the CAA, in part because the scientific bases for quantifying air quality and physical effect relationships are most advanced for health effects. The CAA yielded other benefits, however, which are important even though they were sometimes difficult or impossible to quantify fully given currently available scientific and applied economic information.

Ecological Effects

The CAA yielded important benefits in the form of healthier ecological resources, including: stream, river, lake and estuarine ecosystems; forest and wetland ecosystems; and agricultural ecosystems. These benefits are important because of both the intrinsic value of these ecological resources and the intimate linkage between human health and the health and vitality of our sustaining ecosystems. Given the complexity of natural and agricultural ecosystems and the large spatial and temporal dimensions involved, it has been difficult or impossible to quantify benefits fully given currently available scientific and applied economic information.

Aquatic and Forest Effects

Beyond the intrinsic value of preserving natural aquatic (i.e., lakes, streams, rivers, and estuaries), terrestrial (i.e., forest and grassland), and wetland ecosystems and the life they support, protection of ecosystems from the adverse effects of air pollution can yield significant benefits to human welfare. The historical reductions in air pollution achieved under the CAA probably led to significant improvements in the health of ecosystems and the myriad ecological services they provide. Reductions in acid deposition (SO_x and NO_x) and mercury may have reduced adverse effects on aquatic ecosystems, including finfish, shellfish, and amphibian mortality and morbidity, reduced acidification of poorly buffered systems, and reduced eutrophication of estuarine systems. Ecological protection, in turn, can enhance human welfare through improvements in commercial and recreational fishing, wildlife viewing, maintenance of biodiversity, improvements in drinking water quality, and improvements in visibility.

Wetlands ecosystems are broadly characterized as transitional areas between terrestrial and aquatic systems in which the water table is at or near the surface or the land is periodically covered by shallow water. Valuable products and services of wetlands include: flood control, water quality protection and improvement, fish and wildlife habitat, and landscape and biological diversity. High levels of air pollutants have the potential to adversely impact wetlands. Reductions of these pollutants due to compliance with the CAA have reduced the adverse effects of acidification and eutrophication of wetlands, which in turn has protected habitat and drinking water quality.

Forest ecosystems, which cover 33 percent of the land in the United States, provide an extensive array of products and services to humans. Products include lumber, plywood, paper, fuelwood, mulch, wildlife (game), water (quality), seeds, edible products (e.g., nuts, syrup), drugs, and pesticides. Forest services include recreation, biological and landscape diversity, amenity functions (e.g., urban forest), reduced runoff and erosion, increased soil and nutrient conservation, pollutant sequestration (e.g., CO_2, heavy metals) and pollutant detoxification (e.g., organochlorines). The greatest adverse effect on forest systems are imposed by ozone. No studies have attempted to quantify the economic benefits associated with all product and service functions from any U.S. forest. Some studies have attempted to estimate the net economic damage from forest exposure to air pollutants by calculating hypothetical or assumed reductions in growth rates of commercial species. While quantification of forest damages remains incomplete, available evidence suggests that recreational, service, and non-use benefits may be substantial.

For a more comprehensive discussion of the possible ecological effects of the CAA, see Appendix E.

Quantified Agricultural Effects

Quantification of the effects of the CAA on agriculture was limited to the major agronomic crop species including barley, corn, soybeans, peanuts, cotton, wheat, and sorghum. These species account for 70 percent of all cropland in the U.S., and 73 percent of the nation's agricultural receipts. Ozone is the primary pollutant affecting agricultural production. Nationwide crop damages were estimated under the control and no-control scenarios. Net changes in economic surplus (in 1990 dollars) annually and as a cumulative present value (discounted at 5%) over the period 1976-1990 were estimated. Positive surpluses were exhibited in almost all years and were the result of the increase in yields associated with decreased ozone concentrations under the control scenario. The present value (in 1990) of the estimated agricultural benefits of the CAA ranges from $7.8 billion in the minimum response case to approximately $37 billion in the maximum response case[51] (note that discounting 1976-1990 benefits to 1990 amounts to a compounding of benefits). Exposure-response relationships and cultivar mix reflect historical patterns and do not account for possible substitution of more ozone-resistant cultivars in the no-control scenario. Thus, the upper end of the range of benefit calculations may overestimate the actual agricultural benefits of the CAA with respect to these crops. Because numerous crops are excluded from the analysis, including high value crops that may be sensitive to ozone, the lower end of the range is not likely to fully capture the agricultural benefits of reductions in ozone.

Effects of Air Toxics

In addition to control of criteria pollutants, the Clean Air Act resulted in control of some air toxics — defined as non-criteria pollutants which can cause adverse effects to human health and to ecological resources. Control of these pollutants resulted both from incidental control due to criteria pollutant programs and specific controls targeted at air toxics through the National Emission Standards for Hazardous Air Pollutants (NESHAPs) under Section 112 of the Act.

Air toxics are capable of producing a wide variety of effects. Table 11 presents the range of potential human health and ecological effects which can occur due to air toxics exposure. For several years, the primary focus of risk assessments and control programs designed to reduce air toxics has been cancer. Accord-

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[51] Ranges reflect usage of alternate exposure-response functions.

Table 11. Health and Welfare Effects of Hazardous Air Pollutants.

Effect Category	Quantified Effects	Unquantified Effects	Other Possible Effects
Human Health	Cancer Mortality - nonutility stationary source - mobile source	Cancer Mortality - utility source - area source Noncancer effects - neurological - respiratory - reproductive - hematopoietic - developmental - immunological - organ toxicity	
Human Welfare		Decreased income and recreation opportunities due to fish advisories Odors	Decreased income resulting from decreased physical performance
Ecological		Effects on wildlife Effects on plants Ecosystem effects Loss of biological diversity	Effects on global climate
Other Welfare		Visibility Building Deterioration	Loss of biological diversity

ing to present EPA criteria, there are over 100 known or suspected carcinogens. EPA's 1990 Cancer Risk study indicated that as many as 1,000 to 3,000 cancers annually may be attributable to the air toxics for which assessments were available (virtually all of this estimate came from assessments of about a dozen well-studied pollutants).[52]

In addition to cancer, these pollutants can cause a wide variety of health effects, ranging from respiratory problems to reproductive and developmental effects. There has been considerably less work done to assess the magnitude of non-cancer effects from air toxics, but one survey study has shown that some pollutants are present in the atmosphere at reference levels that have caused adverse effects in animals.[53]

Emissions of air toxics can also cause adverse health effects via non-inhalation exposure routes. Persistent bioaccumulating pollutants, such as mercury and dioxins, can be deposited into water or soil and subsequently taken up by living organisms. The pollutants can biomagnify through the food chain and exist in high concentrations when consumed by humans in foods such as fish or beef. The resulting exposures can cause adverse effects in humans, and can also disrupt ecosystems by affecting top food chain species.

Finally, there are a host of other potential ecological and welfare effects associated with air toxics, for which very little exists in the way of quantitative analysis. Toxic effects of these pollutants have the potential to disrupt both terrestrial and aquatic ecosystems and contribute to adverse welfare effects such as fish consumption advisories in the Great Lakes.[54]

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[52] U.S. EPA, Cancer Risk from Outdoor Exposure to Air Toxics. EPA-450/1-90-004f. Prepared by EPA/OAR/OAQPS.

[53] U.S. EPA, "Toxic Air Pollutants and Noncancer Risks: Screening Studies," External Review Draft, September, 1990.

[54] U.S. EPA, Office of Air Quality Planning and Standards. "Deposition of Air Pollutants to the Great Waters, First Report to Congress," May 1994. EPA-453/R-93-055.

Unfortunately, the effects of air toxics emissions reductions could not be quantified for the present study. Unlike criteria pollutants, there was relatively little monitoring data available for air toxics, and that which exists covered only a handful of pollutants. Emissions inventories were very limited and inconsistent, and air quality modeling has only been done for a few source categories. In addition, the scientific literature on the effects of air toxics was generally much weaker than that available for criteria pollutants.

Limitations in the underlying data and analyses of air toxics led the Project Team to exclude the available quantitative results from the primary analysis of CAA costs and benefits. The estimates of cancer incidence benefits of CAA air toxics control which were developed, but ultimately rejected, are presented in Appendix H. Also found in Appendix H is a list of research needs identified by the Project Team which, if met, would enable at least a partial assessment of air toxics benefits in future section 812 studies.

Uncertainty In The Physical Effects Estimates

As discussed above, and in greater detail in Appendix D, a number of important assumptions and uncertainties in the physical effects analysis may influence the estimate of monetary benefits presented in this study. Several of these key uncertainties, their potential directional bias, and the potential significance of this uncertainty for the overall results of the analysis are summarized in Table 12.

Table 12. Uncertainties Associated with Physical Effects Modeling.

Potential Source of Error	**Direction of Potential Bias in Physical Effects Estimate**	**Significance Relative to Key Uncertainties in Overall Monetary Benefit Estimate**
Estimation of $PM_{2.5}$ from modeled PM_{10} and TSP data (to support mortality estimation)	Unknown	Significant. Estimated $PM_{2.5}$ profiles are used to calculate most of the premature mortality. There is significant uncertainty about how the fine particle share of overall PM levels varies temporally and spatially throughout the 20 year period.
Extrapolation of health effects to populations distant from monitors (or monitored counties in the case of PM).	Probable overestimate.	Probably minor. In addition, this adjustment avoids the underestimation which would result by estimating effects for only those people living near monitors. Potential overestimate may result to the extent air quality in areas distant from monitors is significantly better than in monitored areas. This disparity should be quite minor for regional pollutants, such as ozone and fine particulates.
Estimation of degree of life-shortening associated with PM-related premature mortality.	Unknown.	Unknown, possibly significant when using a value of life-years approach. Varying the estimate of degree of prematurity has no effect on the aggregate benefit estimate when a value of statistical life approach is used since all incidences of premature mortality are valued equally. Under the alternative approach based on valuing individual life-years, the influence of alternative values for numbers of average life-years lost may be significant.
Assumption of zero lag between exposure and incidence of PM-related premature mortality.	Overestimate.	Probably minor. The short-term mortality studies indicate that a significant portion of the premature mortality associated with exposure to elevated PM concentrations is very short-term (i.e., a matter of a few days). In addition, the available epidemiological studies do not provide evidence of a significant lag between exposure and incidence. The lag is therefore likely to be a few years at most and application of reasonable discount rates over a few years would not alter the monetized benefit estimate significantly.
Choice of CR function (i.e., “across-study” uncertainties)	Unknown.	Significant. The differences in implied physical outcomes estimated by different underlying studies are large.
Uncertainty associated with CR functions derived from each individual study (i.e., “within study” uncertainty)	Unknown.	Probably minor.
Exclusion of potential UV-B attenuation benefits associated with higher concentrations of tropospheric ozone under the no-control case.	Overestimate.	Insignificant. In addition to the incomplete scientific evidence that there is a UV-B exposure disbenefit associated specifically with tropospheric ozone reductions, the potential contribution toward total ozone column attenuation from the tropospheric layer is probably very small.
Exclusion of potential substitution of ozone-resistant cultivars in agriculture analysis.	Overestimate.	Insignificant, given small relative contribution of quantified agricultural effects to overall quantified benefit estimate.
Exclusion of other agricultural effects (crops, pollutants)	Underestimate.	Unknown, possibly significant.
Exclusion of effects on terrestrial, wetland, and aquatic ecosystems, and forests.	Underestimate.	Unknown, possibly significant.
No quantification of materials damage	Underestimate	Unknown, possibly significant.

6

Economic Valuation

Estimating the reduced incidence of physical effects represents a valuable measure of health benefits for individual endpoints; however, to compare or aggregate benefits across endpoints, the benefits must be monetized. Assigning a monetary value to avoided incidences of each effect permits a summation, in terms of dollars, of monetized benefits realized as a result of the CAA, and allows that summation to be compared to the cost of the CAA.

For the present analysis of health and welfare benefits, valuation estimates were obtained from the economic literature, and are reported in dollars per case reduced for health effects and dollars per unit of avoided damage for welfare effects.[55] Similar to estimates of physical effects provided by health studies, each of the monetary values of benefits applied in this analysis is reported in terms of a mean value and a probability distribution around the mean estimate. The statistical form of the probability distribution used for the valuation measures varies by endpoint. For example, while the estimate of the dollar value of an avoided premature mortality is described by the Weibull distribution, the estimate for the value of a reduced case of acute bronchitis is assumed to be uniformly distributed between a minimum and maximum value.

Methods for Valuation of Health and Welfare Effects

In environmental benefit-cost analysis, the dollar value of an environmental benefit (e.g., a health-related improvement in environmental quality) conferred on a person is the dollar amount such that the person would be indifferent between having the environmental benefit and having the money. In some cases, this value is measured by studies which estimate the dollar amount required to compensate a person for new or additional exposure to an adverse effect. Estimates derived in this manner are referred to as "willingness-to-accept" (WTA) estimates. In other cases, the value of a welfare change is measured by estimating the amount of money a person is willing to pay to eliminate or reduce a current hazard. This welfare change concept is referred to as "willingness-to-pay" (WTP). For small changes in risk, WTP and WTA are virtually identical, primarily because the budget constraints normally associated with expressions of WTP are not significant enough to drive a wedge between the estimates. For larger risk changes, however, the WTP and WTA values may diverge, with WTP normally being less than WTA because of the budget constraint effect. While the underlying economic valuation literature is based on studies which elicited expressions of WTP and/or WTA, the remainder of this report refers to all valuation coefficients as WTP estimates. In some cases (e.g., stroke-related hospital admissions), neither WTA nor WTP estimates are available and WTP is approximated by cost of illness (COI) estimates, a clear underestimate of the true welfare change since important value components (e.g., pain and suffering associated with the stroke) are not reflected in the out-of-pocket costs for the hospital stay.

For most goods, WTP can be observed by examining actual market transactions. For example, if a gallon of bottled drinking water sells for one dollar, it can be observed that at least some persons are willing to pay one dollar for such water. For goods that are not exchanged in the market, such as most environmental "goods," valuation is not so straightforward. Nevertheless, value may be inferred from observed behavior, such as through estimation of the WTP for mortality risk reductions based on observed sales and prices of safety devices such as smoke detectors. Alternatively, surveys may be used in an attempt to elicit directly WTP for an environmental improvement.

Wherever possible, this analysis uses estimates of the mean WTP of the U.S. population to avoid an environmental effect as the value of avoiding that effect. In some cases, such estimates are not available, and the cost of mitigating or avoiding the effect is used as a rough estimate of the value of avoiding the effect. For example, if an effect results in hospitalization, the avoided medical costs were considered as a possible estimate of the value of avoiding the effect. Finally, where even the "avoided cost" estimate is not available, the analysis relies on other available methods to provide a rough approximation of WTP. As noted above, this analysis uses a range of values for most environmental effects, or endpoints. Table 13

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[55] The literature reviews and valuation estimate development process is described in detail in Appendix I and in the referenced supporting reports.

Table 13. Health and Welfare Effects Unit Valuation (1990 dollars).

Endpoint	Pollutant	Valuation (mean est.)	
Mortality	PM & Pb	$4,800,000	per case
Chronic Bronchitis	PM	$260,000	per case
IQ Changes			
Lost IQ Points	Pb	$3,000	per IQ point
IQ < 70	Pb	$42,000	per case
Hypertension	Pb	$680	per case
Strokes*	Pb	$200,000 $150,000	per case-males per case-females
Coronary Heart Disease	Pb	$52,000	per case
Hospital Admissions			
Ischemic Heart Disease	PM	$10,300	per case
Congestive Heart Failure	PM	$8,300	per case
COPD	PM & O_3	$8,100	per case
Pneumonia	PM & O3	$7,900	per case
All Respiratory	PM & O_3	$6,100	per case
Respiratory Illness and Symptoms			
Acute Bronchitis	PM	$45	per case
Acute Asthma	PM & O_3	$32	per case
Acute Respiratory Symptoms	PM, O_3, NO_2, SO_2	$18	per case
Upper Respiratory Symptoms	PM	$19	per case
Lower Respiratory Symptoms	PM	$12	per case
Shortness of Breath	PM	$5.30	per day
Work Loss Days	PM	$83	per day
Mild Restricted Activity Days	PM & O_3	$38	per day
Welfare Benefits			
Visibility	DeciView	$14	per unit change in DeciView
Household Soiling	PM	$2.50	per household per PM_{10} change
Decreased Worker Productivity	O_3	$1	**
Agriculture (Net Surplus)	O_3	Estimated Change In Economic Surplus	

* Strokes are comprised of atherothrombotic brain infarctions and cerebrovascular accidents; both are estimated to have the same monetary value.

** Decreased productivity valued as change in daily wages: $1 per worker per 10% decrease in O_3.

provides a summary of the mean unit value estimates used in the analysis. The full range of values can be found in Appendix I.

Mortality

Some forms of air pollution increase the probability that individuals will die prematurely. The concentration-response functions for mortality used in this analysis express this increase in mortality risk as cases of "excess premature mortality" per time period (e.g., per year).

The benefit, however, is the avoidance of small increases in the risk of mortality. If individuals' WTP to avoid small increases in risk is summed over enough individuals, the value of a statistical premature death avoided can be inferred.[56] For expository purposes, this valuation is expressed as "dollars per mortality avoided," or "value of a statistical life" (VSL), even though the actual valuation is of small changes in mortality risk.

The mortality risk valuation estimate used in this study is based on an analysis of 26 policy-relevant value-of-life studies (see Table 14). 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 Project Team used the best estimate from each of the 26 studies to construct a distribution of mortality risk valuation estimates for the section 812 study. A Weibull distribution, with a mean of $4.8 million and standard deviation of $3.24 million, provided the best fit to the 26 estimates. There is considerable uncertainty associated with this approach, however, which is discussed in detail later in this chapter and in Appendix I.

In addition, the Project Team developed alternative calculations based on a life-years lost approach. To employ the value of statistical life-year (VSLY) approach, the Project Team had to first estimate the age distribution of those lives which would be saved by reducing air pollution. Based on life expectancy tables, the life-years saved from each statistical life saved within each age and sex cohort were calculated. To value these statistical life-years, a conceptual model was hypothesized which depicted the relationship between the value of life and the value of life-years. As noted earlier in Table 9, the average number of life-years saved across all age groups for which data were available are 14 for PM-related mortality and 38 for Pb-related mortality. The

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[56] Because people are valuing small decreases in the risk of premature mortality, it is expected deaths that are inferred. For example, suppose that a given reduction in pollution confers on each exposed individual a decrease in mortal risk of 1/100,000. Then among 100,000 such individuals, one fewer individual can be expected to die prematurely . If each individual's WTP for that risk reduction is $50, then the implied value of a statistical premature death avoided is $50 x 100,000 = $5 million.

Table 14. Summary of Mortality Valuation Estimates (millions of $1990)

Study	Type of Estimate	Valuation (millions 1990$)
Kneisner and Leeth (1991) (US)	Labor Market	0.6
Smith and Gilbert (1984)	Labor Market	0.7
Dillingham (1985)	Labor Market	0.9
Butler (1983)	Labor Market	1.1
Miller and Guria (1991)	Cont. Value	1.2
Moore and Viscusi (1988a)	Labor Market	2.5
Viscusi, Magat, and Huber (1991b)	Cont. Value	2.7
Gegax et al. (1985)	Cont. Value	3.3
Marin and Psacharopoulos (1982)	Labor Market	2.8
Kneisner and Leeth (1991) (Australia)	Labor Market	3.3
Gerking, de Haan, and Schulze (1988)	Cont. Value	3.4
Cousineau, Lacroix, and Girard (1988)	Labor Market	3.6
Jones-Lee (1989)	Cont. Value	3.8
Dillingham (1985)	Labor Market	3.9
Viscusi (1978, 1979)	Labor Market	4.1
R.S. Smith (1976)	Labor Market	4.6
V.K. Smith (1976)	Labor Market	4.7
Olson (1981)	Labor Market	5.2
Viscusi (1981)	Labor Market	6.5
R.S. Smith (1974)	Labor Market	7.2
Moore and Viscusi (1988a)	Labor Market	7.3
Kneisner and Leeth (1991) (Japan)	Labor Market	7.6
Herzog and Schlottman (1987)	Labor Market	9.1
Leigh and Folson (1984)	Labor Market	9.7
Leigh (1987)	Labor Market	10.4
Gaten (1988)	Labor Market	13.5
SOURCE: Viscusi, 1992		

average for PM, in particular, differs from the 35-year expected remaining lifespan derived from existing wage-risk studies.[57]

Using the same distribution of value of life estimates used above (i.e. the Weibull distribution with a mean estimate of $4.8 million), a distribution for the value of a life-year was then estimated and combined with the total number of estimated life-years lost. The details of these calculations are presented in Appendix I.

Survey-Based Values

Willingness-to pay for environmental improvement is often elicited through survey methods (such as the "contingent valuation" method). Use of such methods in this context is controversial within the economics profession. In general, economists prefer to infer WTP from observed behavior. There are times when such inferences are impossible, however, and some type of survey technique may be the only means of eliciting WTP. Economists' beliefs regarding the reliability of such survey-based data cover a broad spectrum, from unqualified acceptances of the results of properly-conducted surveys to outright rejections of all survey-based valuations.

In this analysis, unit valuations which rely exclusively on the contingent valuation method are chronic bronchitis, respiratory-related ailments, minor restricted activity days, and visibility. As indicated above, the value derived for excess premature mortality stems from 26 studies, of which five use the contingent valuation method. These five studies are within the range of the remaining 21 labor market studies. All five report mortality valuations lower than the central estimate used in this analysis. Excluding the contingent valuation studies from the mortality valuation estimate would yield a central estimate approximately ten percent higher than the 4.8 million dollar value reported above. The endpoints with unit valuations based exclusively on contingent valuation account for approximately 30 percent of the present value of total monetized benefits. Most of the CV-based benefits are attributable to avoided cases of chronic bronchitis.

Chronic Bronchitis

The best available estimate of WTP to avoid a case of chronic bronchitis (CB) comes from Viscusi et al.(1991). The case of CB described to the respondents in the Viscusi study, however, was described by the authors as a severe case. The Project Team employed an estimate of WTP to avoid a pollution-related case of CB that was based on adjusting the WTP to avoid a severe case, estimated by Viscusi et al. (1991), to account for the likelihood that an average case of pollution-related CB is not as severe as the case described in the Viscusi study.

The central tendency estimate of WTP to avoid a pollution-related case of chronic bronchitis (CB) used in this analysis is the mean of a distribution of WTP estimates. This distribution incorporates the uncertainty from three sources: (1) the WTP to avoid a case of severe CB, as described by Viscusi et al., 1991; (2) the severity level of an average pollution-related case of CB (relative to that of the case described by Viscusi et al.(1991); and (3) the elasticity of WTP with respect to severity of the illness. Based on assumptions about the distributions of each of these 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 $260,000, is taken as the central tendency estimate of WTP to avoid a pollution-related case of CB. The three underlying distributions, and the generation of the resulting distribution of WTP, are described in Appendix I.

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[57] See, for example, Moore and Viscusi (1988) or Viscusi (1992).

Respiratory-Related Ailments

In general, the valuations assigned to the respiratory-related ailments listed in Table 14 represent a combination of willingness to pay estimates for individual symptoms which comprise each ailment. For example, a willingness to pay estimate to avoid the combination of specific upper respiratory symptoms defined in the concentration-response relationship measured by Pope et al. (1991) is not available. However, while that study defined upper respiratory symptoms as one suite of ailments (runny or stuffy nose; wet cough; and burning, aching, or red eyes), the valuation literature reported individual WTP estimates for three closely matching symptoms (head/sinus congestion, cough, and eye irritation). The available WTP estimates were therefore used as a surrogate to the values for the precise symptoms defined in the concentration-response study.

To capture the uncertainty associated with the valuation of respiratory-related ailments, this analysis incorporated a range of values reflecting the fact that an ailment, as defined in the concentration-response relationship, could be comprised of just one symptom or several. At the high end of the range, the valuation represents an aggregate of WTP estimates for several individual symptoms. The low end represents the value of avoiding a single mild symptom.

Minor Restricted Activity Days

An individual suffering from a single severe or a combination of pollution-related symptoms may experience a Minor Restricted Activity Day (MRAD). Krupnick and Kopp (1988) argue that mild symptoms will not be sufficient to result in a MRAD, so that WTP to avoid a MRAD should exceed WTP to avoid any single mild symptom. On the other hand, WTP to avoid a MRAD should not exceed the WTP to avoid a work loss day (which results when the individual experiences more severe symptoms). No studies are reported to have estimated WTP to avoid a day of minor restricted activity. Instead, this analysis uses an estimate derived from WTP estimates for avoiding combinations of symptoms which may result in a day of minor restricted activity ($38 per day). The uncertainty range associated with this value extends from the highest value for a single symptom to the value for a work loss day. Furthermore, the distribution acknowledges that the actual value is likely to be closer to the central estimate than either extreme.

Visibility

The value of avoided visibility impairment was derived from existing contingent valuation studies of the household WTP to improve visibility, as reported in the economics literature. These studies were used to define a single, consistent basis for the valuation of visibility benefits nationwide. The central tendency of the benefits estimate is based on an annual WTP of $14 per household per unit improvement in the DeciView index, with upper and lower bounds of $21 and $8, respectively, on the uncertainty range of the estimate.

Avoided Cost Estimates

For some health effects, WTP estimates are not available, and the Project Team instead used “costs avoided” as a substitute for WTP. Avoided costs were used to value the following endpoints: hypertension, hospital admissions, and household soiling.

Hypertension and Hospital Admissions

Avoided medical costs and the avoided cost of lost work time were used to value hypertension (high blood pressure) and hospital admissions (this includes hospital admissions for respiratory ailments as well as heart disease, heart attacks, and strokes) .

For those hospital admissions which were specified to be the initial hospital admission (in particular, hospital admissions for coronary heart disease (CHD) events and stroke), avoided cost estimates should consist of the present discounted value of the stream of medical expenditures related to the illness, as well as the present discounted value of the stream of lost earnings related to the illness. While an estimate of present discounted value of both medical expenditures and lost earnings was available for stroke ($200,000 for males and $150,000 for females), the best available estimate for CHD ($52,000) did not include lost earnings. Although no published estimates of the value of lost earnings due to CHD events are available, one unpublished study suggests that this value could be substantial, possibly exceeding the value of medical expenditures. The estimate of $52,000 for CHD may therefore be a substantial underestimate. The derivations of the avoided cost estimates for CHD and stroke are discussed in Appendix G.

In those cases for which it is unspecified whether the hospital admission is the initial one or not (that is, for all hospital admissions endpoints other than CHD and stroke), it is unclear what portion of medical expenditures and lost earnings after hospital discharge can reasonably be attributed to pollution exposure and what portion might have resulted from an individual's pre-existing condition even in the absence of a particular pollution-related hospital admission. In such cases, the estimates of avoided cost include only those costs associated with the hospital stay, including the hospital charge, the associated physician charge, and the lost earnings while in the hospital ($6,100 to $10,300, depending on the ailment for which hospitalization is required).

The estimate of avoided cost for hypertension included physician charges, medication costs, and hospitalization costs, as well as the cost of lost work time, valued at the rate estimated for a work loss day (see discussion below). Based on this approach, the value per year of avoiding a case of hypertension is taken to equal the sum of medical costs per year plus work loss costs per year; the resulting value is $680 per case per year.

Presumably, willingness-to-pay to avoid the effects (and treatment) of hypertension would reflect the value of avoiding any associated pain and suffering, and the value placed on dietary changes, etc. Likewise, the value of avoiding a health effect that would require hospitalization or doctor's care would include the value of avoiding the pain and suffering caused by the health effect as well as lost leisure time, in addition to medical costs and lost work time. Consequently, the valuations for these endpoints used in this analysis likely represent lower-bound estimates of the true social values for avoiding such health effects.

Household Soiling

This analysis values benefits for this welfare effect by considering the avoided costs of cleaning houses due to particulate matter soiling. The Project Team's estimate reflects the average household's annual cost of cleaning per $\mu g/m^3$ particulate matter ($2.50). Considered in this valuation are issues such as the nature of the particulate matter, and the proportion of households likely to do the cleaning themselves. Since the avoided costs of cleaning used herein do not reflect the loss of leisure time (and perhaps work time) incurred by those who do their own cleaning, the valuation function likely underestimates true WTP to avoid additional soiling.

Other Valuation Estimates

Changes in Children's IQ

One of the major effects of lead exposure is permanently impaired cognitive development in children. No ready estimates of society's WTP for improved cognitive ability are currently available. Two effects of IQ decrements can be monetized, however: reductions in expected lifetime income, and increases in societal expenditures for compensatory education. These two effects almost certainly understate the WTP to avoid impaired cognitive development in children, and probably should be considered lower bound estimates. In the absence of better estimates, however, the Project Team has assumed that the two monetized effects represent a useful approximation of WTP.

The effect of IQ on expected lifetime income comprises a direct and an indirect effect. The direct effect is drawn from studies that estimate, all else being equal, the effect of IQ on income. The indirect effect occurs as a result of the influence of IQ on educational attainment: higher IQ leads to more years of education, and more education leads in turn to higher expected future income. However, this indirect benefit is mitigated, but not eliminated, by the added costs of the additional education and by the potential earnings forgone by the student while enrolled in school.[58] Combining the direct and indirect influences, the net effect of higher IQ on expected lifetime income (discounted to the present at five percent) is estimated to be $3,000 per additional IQ point.

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[58] Theoretically, the indirect effect should be small relative to the direct effect of IQ on future earnings. The empirical research used to derive values for this analysis, however, implies that the indirect effect is roughly equal in magnitude to the direct effect. One can infer from this information that there is a market distortion of some sort present (such as imperfect knowledge of the returns to education), or, perhaps, that individuals make their education "investments" for purposes other than (or in addition to) "maximizing lifetime income." See Appendix G for further discussion of this issue.

In this analysis, it is assumed that part-time compensatory education is required for all children with IQ less than 70. The Project Team assumed that the WTP to avoid cases of children with IQ less than 70 can be approximated by the cost ($42,000 per child) of part-time special education in regular classrooms from grades one through twelve (as opposed to independent special education programs), discounted to the present at five percent. See Appendix G for more detail on valuation methods and data sources for IQ effects and other lead-related health impacts.

Work Loss Days and Worker Productivity

For this analysis, it was assumed that the median daily 1990 wage income of 83 dollars was a reasonable approximation of WTP to avoid a day of lost work. Although a work loss day may or may not affect the income of the worker, depending on the terms of employment, it does affect economic output and is thus a cost to society. Conversely, avoiding the work loss day is a benefit.

A decline in worker productivity has been measured in outdoor workers exposed to ozone. Reduced productivity is measured in terms of the reduction in daily income of the average worker engaged in strenuous outdoor labor, estimated at $1 per 10 percent increase in ozone concentration.

Agricultural Benefits

Similar to the other welfare effects, the agricultural benefits analysis estimated benefits in dollars per unit of avoided damage, based on estimated changes in crop yields predicted by an agricultural sector model. This model incorporated agricultural price, farm policy, and other data for each year. Based on expected yields, the model estimated the production levels for each crop, and the economic benefits to consumers, and to producers, associated with these production levels. To the extent that alternative exposure-response relationships were available, a range of potential benefits was calculated (see Appendix F).

Valuation Uncertainties

The Project Team attempted to handle most valuation uncertainties explicitly and quantitatively by expressing values as distributions (see Appendix I for a complete description of distributions employed), using a Monte-Carlo simulation technique to apply the valuations to physical effects (see Chapter 7) with the mean of each valuation distribution equal to the “best estimate” valuation. This approach does not, of course, guarantee that all uncertainties have been adequately characterized, nor that the valuation estimates are unbiased. It is possible that the actual WTP to avoid an air pollution-related impact is outside of the range of estimates used in this analysis. Nevertheless, the Project Team believes that the distributions employed are reasonable approximations of the ranges of uncertainty, and that there is no compelling reason to believe that the mean values employed are systematically biased (except for the IQ-related and avoided cost-based values, both of which probably underestimate WTP).

One particularly important area of uncertainty is valuation of mortality risk reduction. As noted in Chapter 7, changes in mortality risk are a very important component of aggregate benefits, and mortality risk valuation is an extremely large component of the quantified uncertainty. Consequently, any uncertainty concerning mortality risk valuation beyond that addressed by the quantitative uncertainty assessment (i.e., that related to the Weibull distribution with a mean value of $4.8 million) deserves note. One issue merits special attention: uncertainties and possible biases related to the “benefits transfer” from the 26 valuation source studies to valuation of reductions in PM-related mortality rates.

Mortality Risk Benefits Transfer

Although each of the mortality risk valuation source studies (see Table 14) estimated the average WTP for a given reduction in mortality risk, the degree of reduction in risk being valued varied across studies and is not necessarily the same as the degree of mortality risk reduction estimated in this analysis. The transferability of estimates of the value of a statistical life from the 26 studies to the section 812 benefit analysis rests on the assumption that, within a reasonable range, WTP for reductions in mortality risk is linear in risk reduction. For example, suppose a study estimates that the average WTP for a reduction in mortality risk of 1/100,000 is 50 dollars, but that the actual mortality risk reduction resulting from a given pollutant reduction is 1/10,000. If WTP for reductions in mortality risk is linear in risk reduction, then a WTP of 50 dollars for a reduction of 1/100,000 implies a WTP of 500 dollars 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.

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 the benefit 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 average WTP to reduce risk. The appropriateness of the mean of the WTP estimates from the 26 studies for valuing the mortality-related benefits of reductions in pollutant 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 air pollution and (2) the extent to which the risks being valued are similar.

The substantial majority of the 26 studies relied upon are wage-risk (or labor market) studies. Compared with the subjects in these wage-risk studies, the population most affected by air pollution-related mortality risk changes is likely to be, on average, older and probably more risk averse. Some evidence suggests that approximately 85 percent of those identified in short-term ("episodic") studies who die prematurely from PM-related causes are over 65.[59] The average age of subjects in wage-risk studies, in contrast, would be well under 65.

The direction of bias resulting from the age difference is unclear. It could be argued that, because an older person has fewer expected years left to lose, his or her WTP to reduce mortality risk would be less than that of a younger person. This hypothesis is supported by one empirical study, Jones-Lee et al. (1985), which found WTP to avoid mortality risk at age 65 to be about 90 percent of what it is at age 40. On the other hand, there is 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 the list of 26 studies used here 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.

There is substantial evidence that the income elasticity of WTP for health risk reductions is positive (although there is uncertainty about the exact value of this elasticity). Individuals with higher incomes (or greater wealth) should, then, be willing to pay more to reduce risk, all else equal, than individuals with lower incomes or wealth. The comparison between the (actual and potential) income or wealth of the workers in the wage-risk studies versus that of the population of individuals most likely to be affected by changes in pollution concentrations, however, is unclear. One could argue that because the elderly are relatively wealthy, the affected population is also wealthier, on average, than are the wage-risk study subjects, who tend to be middle-aged (on average) blue-collar workers. On the other hand, the workers in the wage-risk studies will have potentially more years remaining in which to acquire streams of income from future earnings. In addition, it is possible 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). On net, the potential income comparison is unclear.

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[60] that people will pay more to reduce involuntarily incurred risks than risks incurred voluntarily. If this is the case, WTP estimates based on wage-risk studies may be downward biased estimates of WTP to reduce involuntarily incurred air pollution-related mortality risks.

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[59] See Schwartz and Dockery (1992), Ostro et al. (1995), and Chestnut (1995).

[60] See, for example, Violette and Chestnut, 1983.

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

The potential sources of bias introduced by relying on wage-risk studies to derive an estimate of the WTP to reduce air pollution-related mortality risk are summarized in Table 15. Among these potential biases, it is disparities in age and income between the subjects of the wage-risk studies and those affected by air pollution which have thus far motivated specific suggestions for quantitative adjustment[61] ; however, the appropriateness and the proper magnitude of such potential adjustments remain unclear given presently available information. These uncertainties are particularly acute given the possibility that age and income biases might offset each other in the case of pollution-related mortality risk aversion. Furthermore, the other potential biases discussed above, and summarized in Table 16, add additional uncertainty regarding the transferability of WTP estimates from wage-risk studies to environmental policy and program assessments.

Table 15. Estimating Mortality Risk Based on Wage-Risk Studies: Potential Sources and Likely Direction of Bias.

Factor	Likely Direction of Bias in WTP Estimate
Age	Uncertain, perhaps upward
Degree of Risk Aversion	Downward
Income	Uncertain
Voluntary vs. Involuntary	Downward
Catastrophic vs. Protracted Death	Uncertain, perhaps downward

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[61] Chestnut, 1995; IEc, 1992.

7

Results and Uncertainty

This chapter presents a summary of the monetized benefits of the CAA from 1970 to 1990, compares these with the corresponding costs, explores some of the major sources of uncertainty in the benefits estimates, and presents alternative results reflecting diverging viewpoints on two key variables: PM-related mortality valuation and the discount rate.

Monetized economic benefits for the 1970 to 1990 period were derived by applying the unit valuations discussed in Chapter 6 to the stream of physical effects estimated by the method documented in Chapter 5. The range of estimates for monetized benefits is based on the quantified uncertainty associated with the health and welfare effects estimates and the quantified uncertainty associated with the unit valuations applied to them. Quantitative estimates of uncertainties in earlier steps of the analysis (i.e., estimation of compliance costs,[62] emissions changes, and air quality changes) could not be adequately developed and are therefore not applied in the present study. As a result, the range of estimates for monetized benefits presented in this chapter is narrower than would be expected with a complete accounting of the uncertainties in all analytical components. However, the uncertainties in the estimates of physical effects and unit values are considered to be large relative to these earlier components. The characterization of the uncertainty surrounding unit valuations is discussed in detail in Appendix I. The characterization of the uncertainty surrounding health and welfare effects estimates, as well as the characterization of overall uncertainty surrounding monetized benefits, is discussed below.

Quantified Uncertainty in the Benefits Analysis

Alternative studies published in the scientific literature which examine the health or welfare consequences of exposure to a given pollutant often obtain different estimates of the concentration-response (CR) relationship between the pollutant and the effect. In some instances the differences among CR functions estimated by, or derived from, the various studies are substantial. In addition to sampling error, these differences may reflect actual variability of the concentration-response relationship across locations. Instead of a single CR coefficient characterizing the relationship between an endpoint and a pollutant in the CR function, there could be a distribution of CR coefficients which reflect geographic differences.[63] Because it is not feasible to estimate the CR coefficient for a given endpoint-pollutant combination in each county in the nation, however, the national benefits analysis applies the mean of the distribution of CR coefficients to each county. This mean is estimated based on the estimates of CR coefficients reported in the available studies and the information about the uncertainty of these estimates, also reported in the studies.

Based on the assumption that for each endpoint-pollutant combination there is a distribution of CR coefficients, the Project team used a Monte Carlo approach to estimate the mean of each distribution and to characterize the uncertainty surrounding each estimate. For most health and welfare effects, only a single study is considered. In this case, the best estimate of the mean of the distribution of CR coefficients is the reported estimate in the study. The uncertainty surrounding the estimate of the mean CR coefficient is best characterized by the standard error of the reported estimate. This yields a normal distribution, centered at the reported estimate of the mean. If two or more studies are considered for a given endpoint-pollutant combination, a normal distribution is derived for each study, centered at the mean estimate reported in the study. On each iteration of a Monte Carlo procedure, a CR coefficient is randomly selected from each of the normal distributions, and the selected values are averaged. This yields an estimate of the mean CR coefficient for that endpoint-pollutant combination. Iterating this procedure many times results in a distribution of estimates of the mean CR coefficient.

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[62] Although compliance cost estimation is primarily of concern to the cost side of this analysis, uncertainty in the estimates for compliance costs does influence the uncertainty in the benefit estimates because compliance cost changes were used to estimate changes in macroeconomic conditions which, in turn, influenced the estimated changes in emissions, air quality, and physical effects.

[63] Geographic variability may result from differences in lifestyle (e.g., time spent indoors vs outdoors), deposition rates, or other localized factors which influence exposure of the population to a given atmospheric concentration of the pollutant.

Each estimate randomly selected from this distribution was evaluated for each county in the nation, and the results were aggregated into an estimate of the national incidence of the health or welfare effect. Through repeated sampling from the distribution of mean CR coefficients, a distribution of the estimated change in effect outcomes due to the change in air quality between the control and no-control scenarios was generated.

Once a distribution of estimated outcomes was generated for each health and welfare effect, Monte Carlo methods were used again to characterize the overall uncertainty surrounding monetized benefits. For each health and welfare effect in a set of non-overlapping effects, an estimated incidence was randomly selected from the distribution of estimated incidences for that endpoint, and a unit value was randomly selected from the corresponding distribution of unit values, on each iteration of the Monte Carlo procedure. The estimated monetized benefit for that endpoint produced on that iteration is the product of these two factors. Repeating the process many times generated a distribution of estimated monetized benefits by endpoint. Combining the results for the individual endpoints using the Monte Carlo procedure yielded a distribution of total estimated monetized benefits for each target year (1975, 1980, 1985 and 1990). This technique enabled a representation of uncertainty in current scientific and economic opinion in these benefits estimates.

Aggregate Monetized Benefits

For each of the target years of the analysis, the monetized benefits associated with the different health and welfare effects for that year must be aggregated. These aggregate benefits by target year must then be aggregated across the entire 1970 to 1990 period of the study to yield a present discounted value of aggregate benefits for the period. The issues involved in each stage of aggregation, as well as the results of aggregation, are presented in this section. (The detailed results for the target years are presented in Appendix I.)

Table 16. Present Value of 1970 to 1990 Monetized Benefits by Endpoint Category for 48 State Population (billions of $1990, discounted to 1990 at 5 percent).

		Present Value		
Endpoint	**Pollutant(s)**	**5th %ile**	**Mean**	**95th %ile**
Mortality	PM	$2,369	$16,632	$40,597
Mortality	Pb	$121	$1,339	$3,910
Chronic Bronchitis	PM	$409	$3,313	$10,401
IQ (Lost IQ Pts. + Children w/ IQ<70)	Pb	$271	$399	$551
Hypertension	Pb	$77	$98	$120
Hospital Admissions	PM, O3, Pb, & CO	$27	$57	$120
Respiratory-Related Symptoms, Restricted Activity, & Decreased Productivity	PM, O3, NO2, & SO2	$123	$182	$261
Soiling Damage	PM	$6	$74	$192
Visibility	particulates	$38	$54	$71
Agriculture (Net Surplus)	O3	$11	$23	$35

Table 16 presents monetized benefits for each quantified and monetized health and welfare endpoint (or group of endpoints), aggregated from 1970 to 1990. The mean estimate resulting from the Monte Carlo simulation is presented, along with the measured credible range (upper and lower fifth percentiles of the distribution). Aggregating the stream of monetized benefits across years involved compounding the stream of monetized benefits estimated for each year to the 1990 present value (using a five percent discount rate).

Since the present value estimates combine streams of benefits from 1970 to 1990, the calculation required monetized estimates for each year. However, Monte Carlo modeling was carried out only for the four target years (1975, 1980, 1985 and 1990). In the intervening years, only a central estimate of benefits was estimated for each health and welfare endpoint (by multiplying the central incidence estimate for the given year by the central estimate of the unit valuation). The resulting annual benefit estimates provided a temporal trend of monetized benefits across the period resulting from the annual changes in air quality. They did not, however, characterize the uncertainty associated with the yearly estimates for intervening years. In an attempt to capture uncertainty associated with these estimates, the Project Team relied on the ratios of the 5th percentile to the mean and the 95th percentile to the mean in the target years. In general, these ratios were fairly constant across the target years, for a given endpoint. The ratios were interpolated between the target years, yielding ratios for the intervening years. Multiplying the ratios for each intervening year by the central estimate generated for that year provided estimates of the 5th and 95th percentiles, which were used to characterize uncertainty about the central estimate. Thus, the present value of the stream of benefits, including the credible range estimates, could be computed.

Table 16 offers a comparison of benefits by health or welfare endpoint. The effect categories listed in the table are mutually exclusive, allowing the monetized benefits associated with them to be added. It should be noted, however, that the listed categories combine estimates that are not mutually exclusive. To avoid double counting, care was taken to treat the benefits associated with overlapping effects as alternative estimates. For example, the "Hospital Admissions" category includes admissions for specific ailments (Pneumonia and COPD) as well as the broader classification of "all respiratory" ailments. Clearly, benefits accruing from the first two represent a subset of the last and adding all three together would result in an overestimate of total monetized benefits. To avoid this, the sum of benefits from Pneumonia and COPD was treated as an alternative to the benefits estimated for all respiratory ailments (the sum of the first two was averaged with the third). This issue of double-counting also arose for two other cases of overlapping health effects, both of which have been combined into the "Respiratory-Related Symptoms, Restricted Activity, & Decreased Productivity" category in Table 16. First, acute bronchitis was treated as an alternative (i.e., averaged with) the combination of upper and lower respiratory symptoms, since their definitions of symptoms overlap. Second, various estimates of restricted activity, with different degrees of severity, were combined into a single benefit category.

Table 17. Total Monetized Benefits for 48 State Population (Present Value in billions of 1990$, discounted to 1990 at 5 percent).

	Present Value		
	5th %ile	Mean	95th %ile
TOTAL (Billions of 1990-value dollars)	$5,600	$22,200	$49,400

Table 17 reports the estimated total national monetized benefits attributed in this analysis to the CAA from 1970 to 1990. The benefits, valued in 1990 dollars, range from $5.6 to $49.4 trillion with a central estimate of $22.2 trillion. The Monte Carlo technique was used to aggregate monetized benefits across endpoints. For each of several thousand iterations, a random draw of the monetized benefits for each endpoint was selected from the distributions summarized in

Table 16 and the individual endpoint estimates were then summed. This resulted in the distribution of total national monetized benefits reported above.[64]

The temporal pattern of benefits during the 1970 to 1990 period is related to the difference in emissions between the control and no-control scenarios and is magnified by population growth during that period. As illustrated by Figure 18, quantified annual benefits increased steadily during the study period, with the greatest increases occurring during the late 1970s. The mean estimate of quantified annual benefits grew from 355 billion dollars in 1975 (expressed as inflation-adjusted 1990 dollars) to 930 billion dollars in 1980, 1,155 billion dollars in 1985, and 1,248 billion dollars in 1990.

Figure 19 depicts the distribution of monetized benefits for 1990 (similar distributions were generated for other years in the analysis period). The solid vertical bars in the figure represent the relative frequency of a given result in the 1990 Monte Carlo analysis. The largest bar, located above the "<$1,000", indicates that more Monte Carlo iterations generated monetized benefits of $900 billion to $1 trillion than in any other $100 billion range bin, making this the modal bin. The expected value of the estimate for total monetized benefit for 1990 (i.e., the mean of the distribution) is $1.25 trillion. The ninety percent confidence interval, a summary description of the spread of a distribution, is also noted in the figure.

Figure 18. Monte Carlo Simulation Model Results for Target Years (in billions of 1990 dollars).

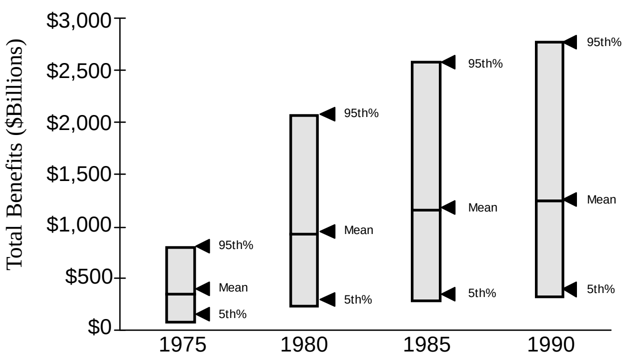
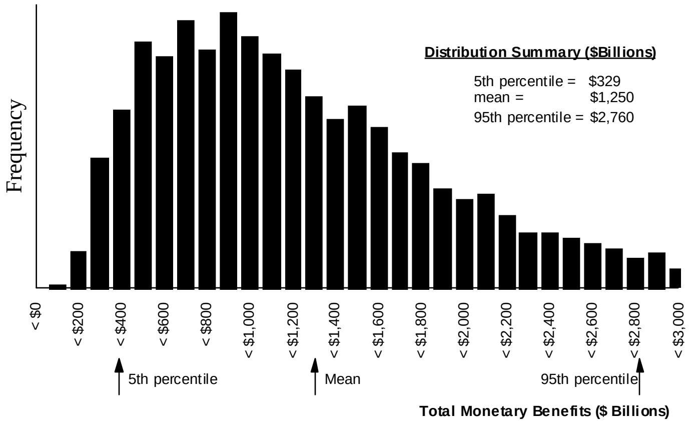


Figure 19. Distribution of 1990 Monetized Benefits of CAA (in billions of 1990 dollars).



On initial inspection, the estimated $1.25 trillion value for monetized benefits in 1990 may seem implausibly large, even though 1990 is the year in which the differences between outcomes under the control and no-control scenarios are at their most extreme. The plausibility of this estimate may seem particularly questionable to some if one considers that the $1.25 trillion value for 1990 is over five percent of the estimated $22.8 trillion value for total 1990 assets of households and nonprofit organizations. Considered from this perspective, $1.25 trillion may seem to represent a large share of total wealth, and some might question whether Americans would really be willing to pay this much money for the reductions in risk achieved by the Clean Air Act and related programs, even if the risk in question involves premature death. However, in the end it is clear that such comparisons are overly simplistic and uninformative because they ignore the magnitude and nature of the welfare change being measured.

First, with respect to the magnitude of the difference in estimated social welfare under the two scenarios, it is important to recognize how severe air quality conditions and health risks would be under the hypothetical no-control scenario. Focusing on ambient particulate matter, the pollutant responsible for the vast majority of the estimated monetary benefits, a comparison of the estimated annual mean concentrations of total suspended particulates (TSP) projected in the U.S. under the no-control scenario with estimated annual mean TSP concentrations in other parts of the world[65] indicates that in 1990—

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[64] Comparing Tables 16 and 17, it can be seen that the sum of benefits across endpoints at a given percentile level does not result in the total monetized benefits estimate at the same percentile level in Table 17. For example, if the fifth percentile benefits of the endpoints shown in Table 16 were added, the resulting total would be substantially less than $5.6 trillion, the fifth percentile value of the distribution of aggregate monetized benefits reported in Table 17. This is because the various health and welfare effects are treated as stochastically independent, so that the probability that the aggregate monetized benefit is less than or equal to the sum of the separate five percentile values is substantially less than five percent.

- 60 metropolitan areas in the U.S. would have had higher TSP concentrations than Moscow, Russia
- 7 metropolitan areas would be worse than Bangkok, Thailand
- 6 metropolitan areas would be worse than Bombay, India
- 2 metropolitan areas would be worse than Manila, Philippines
- One metropolitan area would be worse than Delhi, India (one of the most polluted cities in the world)

Under the control scenario, TSP levels in only 3 metropolitan areas were projected to exceed those in Moscow, and none exceeded levels found in the other foreign cities listed above. The principal reason air quality conditions are so poor under the no-control scenario is that air pollution control requirements remain fixed at their 1970 levels of scope and stringency while total economic activity, including polluting activity, grows by 70 percent and population grows by 22.3 percent between 1970 and 1990. Under the severe air quality conditions projected throughout the U.S. in 1990 under the no-control case, an additional 205,000 people would be projected to die prematurely due to the effects of particulate matter, lead, and other criteria pollutants. This represents a very large increase in the risk of premature mortality. Since the estimate that the average loss of life for those who actually succumb to PM exposure related health effects is approximately 14 years, and life-shortening due to lead exposure is even greater, it is no longer surprising that the estimated value of avoiding these severe conditions is so high.

Second, with respect to the nature of the welfare change reflected in the monetized benefit estimate, the concern about the effects of limited budgets constraining Americans' collective ability to pay to avoid these severe no-control scenario conditions is misplaced. In reality, what society actually had to pay to avoid these conditions is measured on the cost side of the analysis, which sums up the total expenditures made by manufacturers and others to achieve these air pollution reductions. The most reasonable estimate of the value Americans place on avoiding those severe no-control scenario conditions, however, is provided by measuring the amount of compensation Americans would have demanded from polluting companies and others to accept, willingly, all of that extra pollution and its associated risks of premature death. Under this concept of welfare change measurement, there is no inherent limit on the amount of money citizens would demand from companies to accept their pollution and so individual personal wealth does not constrain this value.

The monetized benefit estimate presented in this study, therefore, does not necessarily represent an attempt to mirror what Americans would pay out of their own pockets to reduce air pollution from levels they never experienced; rather, it provides an estimate of the value Americans place on the protection they received against the dire air pollution conditions which might have prevailed in the absence of the 1970 and 1977 Clean Air Acts and related programs. Viewed from this perspective, the estimated monetized benefits presented herein appear entirely plausible.

Comparison of Monetized Benefits and Costs

Table 18 presents summary quantitative results for the retrospective assessment. Annual results are presented for four individual years, with all dollar figures expressed as inflation-adjusted 1990 dollars. The final column sums the stream of costs and benefits from 1970 to 1990, discounted (i.e., compounded) to 1990 at five percent. "Monetized benefits" indicate both the mean of the Monte Carlo analysis and the credible range. "Net Benefits" are mean monetized benefits less annualized costs for each year. The table also notes the benefit/cost ratios implied by the benefit ranges. The distribution of benefits changes little (except in scale) from year to year: The mean estimate is somewhat greater than twice the fifth percentile estimate, and the ninety-fifth percentile estimate is somewhat less than twice the mean estimate. The distribution shape changes little across years because the sources of uncertainty (i.e., CR functions and economic valuations) and their characterizations are unchanged from year to year. Some variability is induced by changes in relative pollutant concentrations over time, which then change the relative impact of individual CR functions.

Several measures of "cost" are available for use in this analysis (see Chapter 2). The Project Team

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[65] "Urban Air Pollution in Megacities of the World," UNEP/WHO, 1992a, Published by the World Health Organization and United Nations Environment Program, Blackwell Publishers, Oxford, England, 1992. "City Air Quality Trends," UNEP/WHO, 1992b, Published by the United Nations Environment Program, Nairobi, Kenya, 1992.

Table 18. Quantified Uncertainty Ranges for Monetized Annual Benefits and Benefit/Cost Ratios, 1970-1990 (in billions of 1990-value dollars).

	1975	1980	1985	1990	PV
Monetized Benefits					
5th percentile	87	235	293	329	5,600
Mean estimate	355	930	1,155	1,248	22,200
95th percentile	799	2,063	2,569	2,762	49,400
Annualized Costs (5%)	14	21	25	26	523
Net Benefits					
Mean benefits - Costs	341	909	1,130	1,220	21,700
Benefit/Cost ratio					
5th percentile	6/1	11/1	12/1	13/1	11/1
Mean estimate	25/1	44/1	46/1	48/1	42/1
95th percentile	57/1	98/1	103/1	106/1	94/1

Notes: PV=1990 present value reflecting compounding of costs and benefits from 1971 to 1990 at 5 percent.

employs "annualized cost" as the primary cost measure because it measures cost in a fashion most analogous to the benefits estimation method. An alternative measure, "compliance expenditure," is a reasonable cost measure. Some capital expenditures, however, generate a benefit stream beyond the period of the analysis (i.e., beyond 1990). Those post-1990 benefits are not, in general, included in the benefit estimates presented above. The annualization procedure reduces the bias introduced by the use of capital expenditures by spreading the cost of the capital investment over its expected life, then counting as a "cost" only those costs incurred in the 1970 to 1990 period.

The macroeconomic analysis employed for this analysis (see Chapter 2) indicates that compliance expenditures induce significant second-order effects, and it can be argued that those effects should be included in a comprehensive cost analysis. Benefits resulting from compliance expenditures should also induce second-order macroeconomic effects (which would, one would expect, partly or completely offset the estimated second-order adverse effects induced by compliance expenditures). Due to the sequencing of the analytical steps in this assessment, it was not practical to estimate the second-order cost and benefit impacts induced by the estimated health and welfare benefits. Because second-order impacts of benefits are not estimated, the Project Team refrained from choosing as the primary cost measure one that included second-order impacts, and instead employed "annualized costs" as the primary cost measure.

Major Sources of Uncertainty

The methods used to aggregate monetized benefits and characterize the uncertainty surrounding estimates of these benefits have been discussed above, and the resulting estimates of aggregate benefits have been compared to the corresponding estimates of cost. Additional insights into key assumptions and findings can, however, be obtained by further analysis of potentially important variables.

For some factors in the present analysis, both the degree of uncertainty and the direction of any associated bias are unknown; for some other factors, no employable quantitative estimates could be used even though available evidence suggests a positive and potentially substantial value. An example of the latter deficiency is the lack of quantitative estimates for some human health effects, some human welfare effects, and all ecological effects. Despite the exclusion of potentially important variables, it is worthwhile to evaluate the relative contribution of included variables to quantifiable uncertainty in the net benefit estimate. One of these variables, premature mortality valuation, is also given special attention in the subsequent section on alternative results.

The estimated uncertainty ranges for each endpoint category summarized in Table 16 reflect the measured uncertainty associated with both avoided incidence and economic valuation. The Project Team conducted a sensitivity analysis to determine the variables with the greatest contribution to the quantified uncertainty range. The results of this sensitivity analysis are illustrated in Figure 20.

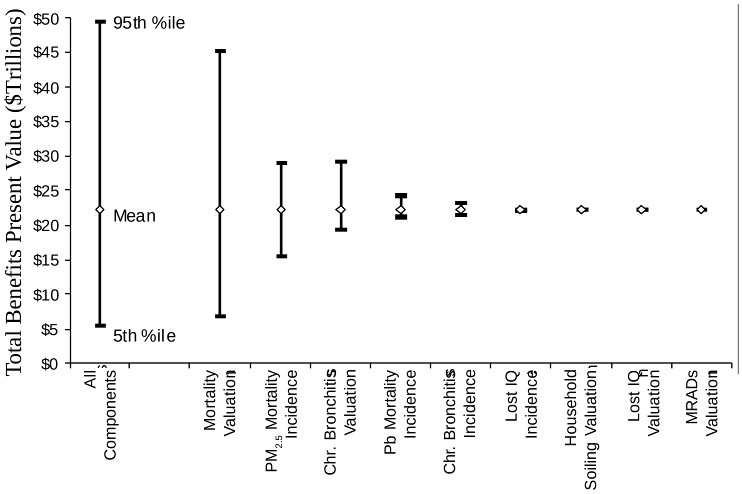
Figure 20. Uncertainty Ranges Deriving From Individual Uncertainty Factors

In this sensitivity analysis, all the inputs to the Monte Carlo uncertainty analysis are held constant (at their mean values), allowing only one variable -- for example, the economic valuation of mortality -- to vary across the range of that variable's uncertainty. The sensitivity analysis then isolates how this single source of uncertainty contributes to the total measured uncertainty in estimated aggregate benefits. The first uncertainty bar represents the credible range associated with the total monetized benefits of the Clean Air Act, as reported above. This captures the multiple uncertainties in the quantified benefits estimation. The rest of the uncertainty bars represent the quantified uncertainty ranges generated by single variables. As shown in Figure 20, the most important contributors to aggregate quantified uncertainty are mortality valuation and incidence, followed by chronic bronchitis valuation and incidence.

Alternative Results

The primary results of this analysis, including aggregate cost and benefit estimates and the uncertainty associated with them, are presented and discussed above. However, although the range of net benefit estimates presented reflects uncertainty in many important elements of the analysis, there are two key variables which require further discussion and analysis: PM-related mortality valuation and the discount rate. This additional treatment is necessary because reasonable people may disagree with the Project Team's methodological choices for these two variables, and these choices might be considered ex ante to significantly influence the results of the study. The purpose of this section, therefore, is to present alternative quantitative results which reflect, separately, (1) an alternative approach to valuation of premature mortality associated with particulate matter exposure, and (2) alternative values for the discount rate used to adjust the monetary values of effects occurring in various years to a particular reference year (i.e., 1990).

PM Mortality Valuation Based on Life-Years Lost

The primary analytical results presented earlier in this chapter assign the same economic value to incidences of premature mortality regardless of the age and health status of those affected. Although this has been the traditional practice for benefit-cost studies conducted within the Agency, this may not be the most appropriate method for valuation of premature mortality caused by PM exposure. Some short-term PM exposure studies suggest that a significantly disproportionate share of PM-related premature mortality occurs among persons 65 years of age or older. Combining standard life expectancy tables with the limited available data on age-specific incidence allows crude approximations of the number of life-years lost by those who die prematurely as a result of exposure to PM or, alternatively, the changes in age-specific life expectancy of those who are exposed to PM.

The ability to estimate, however crudely, changes in age-specific life expectancy raises the issue of whether available measures of the economic value of mortality risk reduction can, and should, be adapted to measure the value of specific numbers of life-years saved.[66] Although the Agency has on occasion performed sensitivity calculations which adjust mortality values for those over age 65, the Agency is skeptical that the current state of knowledge and available analytical tools support using a life-years lost approach or any other approach which assigns different risk reduction values to people of different ages or circumstances. This skepticism is mirrored in the OMB guidance on implementing Executive Order 12866 pertaining to economic analysis methods, which states on page 31:

> *While there are theoretical advantages to using a value of statistical life-year-extended approach, current research does not provide a definitive way of developing estimates of VSLY that are sensitive to such factors as current age, latency of effect, life years remaining, and social valuation of different risk reductions. In lieu of such information, there are several options for deriving the value of a life-year saved from an estimate of the value of life, but each of these methods has drawbacks. One approach is to use results from the wage compensation literature (which focuses on the effect of age on WTP to avoid risk of occupational fatality). However, these results may not be appropriate for other types of risks. Another approach is to annualize the VSL using an appropriate rate of discount and the average life years remaining. This approach does not provide an independent estimate of VSLY; it simply rescales the VSL estimate. Agencies should consider providing estimates of both VSL and VSLY, while recognizing the developing state of knowledge in this area.*

While the Agency continues to prefer an approach which makes no valuation distinctions based on age or other characteristics of the affected population, alternative results based on a VSLY approach are presented below. The method used to develop VSLY estimates is described briefly in Chapter 6 and in more detail in Appendix I.

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[66] This issue was extensively discussed during the Science Advisory Board Council review of drafts of the present study. The Council suggested it would be reasonable and appropriate to show PM mortality benefit estimates based on value of statistical life-years (VSLY) saved as well as the value of statistical life (VSL) approach traditionally applied by the Agency to all incidences of premature mortality.

Table 19 summarizes and compares the results of the VSL and VSLY approaches. Estimated 1970 to 1990 benefits from PM-related mortality alone and total assessment benefits are reported, along with total compliance costs for the same period, in 1990 dollars discounted to 1990 at five percent. The results indicate that the choice of valuation methodology significantly affects the estimated monetized value of historical reductions in air pollution-related premature mortality. However, the downward adjustment which would result from applying a VSLY approach in lieu of a VSL approach does not change the basic outcome of this study, viz. the estimated monetized benefits of the historical CAA substantially exceed the historical costs of compliance.

Table 19. Alternative Mortality Benefits Mean Estimates for 1970 to 1990 (in trillions of 1990 dollars, discounted at 5 percent) Compared to Total 1970 to 1990 Compliance Costs.

	Benefits	
Benefit Estimation Method	PM	Tot.
Statistical life method ($4.8M/case)	16.6	18.0
Life-years lost method ($293,000/year)	9.1	10.1
Total compliance cost	---	0.5

Alternative Discount Rates

In some instances, the choice of discount rate can have an important effect on the results of a benefit-cost analysis; particularly for those analyses with relatively long time horizons for costs and/or benefits. In this assessment, the discount rate affects only four factors: IQ-related benefits estimates (especially estimates of changes in discounted lifetime income), lifetime income losses due to other health effects (e.g., stroke), annualized costs (i.e., amortized capital expenditures), and compounding of all costs and benefits to 1990. Table 20 summarizes the effect of alternative discount rates on the “best estimate” results of this analysis. Because monetized benefits exceed costs for all years in the analysis period, net benefits increase as the discount rate increases. Because the annual benefit/cost ratio increases as one moves from 1970 toward 1990 (see Table 18 above), benefit cost ratios decline as the discount rate increases (because earlier periods are given greater weight). Overall, the results of the benefit-cost assessment appear to be generally insensitive to the choice of discount rate.

Table 20. Effect of Alternative Discount Rates on Present Value of Total Monetized Benefits/Costs for 1970 to 1990 (in trillions of 1990 dollars).

	Discount rate		
	3%	5%	7%
Mean Estimated Benefits	19.2	22.2	25.8
Annualized Costs	0.4	0.5	0.7
Net Benefits	18.8	21.7	25.1
Benefit/Cost ratio	48/1	42/1	37/1

Appendix A: Cost and Macroeconomic Modeling

Introduction

The purpose of this appendix is to describe in detail the estimation of direct compliance costs associated with the CAA and the effect of those expenditures on U.S. economic conditions from 1970 to 1990. The first section of this appendix describes the dynamic, general equilibrium macroeconomic model used to examine economy-wide effects. Two broad categories of models were considered for use in the assessment: Macroeconomic forecasting models (e.g., the Data Resources Inc. model of the U.S. economy), and general equilibrium models (e.g., Hazilla and Kopp [1990], and Jorgenson and Wilcoxen [1990a]). The project team selected the Jorgenson-Wilcoxen (J/W) general equilibrium model of the United States for this analysis (Jorgenson and Wilcoxen [1990a]). There are two main reasons for choosing a dynamic general equilibrium approach: To capture both the direct and indirect economic effects of environmental regulation, and to capture the long-run dynamics of the adjustment of the economy. The general equilibrium framework enabled the project team to assess shifts in economic activity between industries, including changes in distributions of labor, capital, and other production factors within the economy, and changes in the distribution of goods and services.

The second section describes the data sources for direct compliance expenditures and presents estimates of historical air pollution control expenditures. These estimates are derived primarily from EPA's 1990 report entitled "Environmental Investments: The Cost of a Clean Environment"[1] (hereafter referred to as *Cost of Clean*). Specific adjustments to the *Cost of Clean* stationary source and mobile source O&M data needed to adapt these data for use in the present study are also described. These adjusted expenditure estimates represent the compliance cost data used as inputs to the J/W model to determine macroeconomic effects.

The final section presents a summary of the direct expenditure data, presents direct costs in a form that can be compared to the benefits estimates found elsewhere in the study, and discusses indirect effects arising from compliance expenditures estimated by the macroeconomic model. The indirect effects reported by the model are sectoral impacts and changes in aggregate measures of economic activity such as household consumption and gross national product. These indirect effects are second-order impacts of compliance expenditures — a parallel modeling exercise to estimate second-order economic impacts arising from the benefits of compliance (e.g., increased output as a result of improved longevity or fewer workdays lost as a result of non-fatal heart attacks) has not been attempted.

Macroeconomic Modeling

EPA analyses of the costs of environmental regulations typically quantify the direct costs of pollution abatement equipment and related operating and maintenance expenses. However, this approach does not fully account for all of the broader economic consequences of reallocating resources to the production and use of pollution abatement equipment. A general equilibrium, macroeconomic model could, in theory, capture the complex interactions between sectors in the economy and assess the full economic cost of air pollution control. This would be particularly useful for assessing regulations that may produce significant interaction effects between markets. Another advantage of a general equilibrium, macroeconomic framework is that it is internally consistent. The consistency of sectoral forecasts with realistic projections of U.S. economic growth is ensured since they are estimated within the context of a single model.[2] This contrasts with typical EPA analyses that compile cost estimates from disparate sectoral and partial equilibrium models.

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[1] Environmental Investments: The Cost of a Clean Environment, Report of the Administrator of the Environmental Protection Agency to the Congress of the United States, EPA-230-11-90-083, November 1990.

[2] In the present study, both benefits and costs are driven by of the same macroeconomic projections from the Jorgenson/Wilcoxen model, to ensure that the estimates are based on a consistent set of economic assumptions.

The economic effects of the CAA may be over- or underestimated, if general equilibrium effects are ignored, to the extent that sectors not directly regulated are affected. For example, it is well known that the CAA imposed significant direct costs on the energy industry. Economic sectors not directly regulated will nonetheless be affected by changes in energy prices. However, an examination of the broader effects of the CAA on the entire economy might reveal that the CAA also led to more rapid technological development and market penetration of environmentally "clean" renewable sources of energy (e.g., photovoltaics). These effects would partially offset adverse effects on the energy industry, and lead to a different estimate of the total economic cost to society of the CAA.

The significance of general equilibrium effects in the context of any particular analysis is an empirical question. Kokoski and Smith (1987) used a computable general equilibrium model to demonstrate that partial-equilibrium welfare measures can offer reasonable approximations of the true welfare changes for large exogenous changes. In contrast, the results of Jorgenson and Wilcoxen (1990a) and Hazilla and Kopp (1990) suggest that total pollution abatement in the U.S. has been a major claimant on productive resources, and the effect on long-run economic growth may be significant. Again, such conclusions must be considered in light of the limitations of general equilibrium models.

Choice of Macroeconomic Model

The adequacy of any model or modeling approach must be judged in light of the policy questions being asked. One goal of the present study is to assess the effects of clean air regulations on macroeconomic activity. Two broad categories of macroeconomic models were considered for use in the assessment: short run, Keynesian models and long-run, general equilibrium models.

Recognizing that structural differences exist between the models, one needs to focus in on the particular questions that should be answered with any particular model. The Congressional Budget Office (1990) noted:

> "Both the [Data Resources Incorporated] DRI and the IPCAEO models show relatively limited possibilities for increasing energy efficiency and substituting other goods for energy in the short run... Both models focus primarily on short-term responses to higher energy prices, and *neither is very good at examining how the structure of the economy could change in response to changing energy prices.* The [Jorgenson-Wilcoxen] model completes this part of the picture..."[3]

One strategy for assessing the macroeconomic effects of the CAA would be to use a DRI-type model in conjunction with the Jorgenson-Wilcoxen model to assess both the long-term effects and the short-run transitions, in much the same way that the Congressional Budget Office used these models to assess the effects of carbon taxes. However, because of significant difficulties in trying to implement the DRI model in a meaningful way, the project team chose to focus on the long-run effects of the CAA. Structural changes (e.g., changes in employment in the coal sector due to the CAA) can be identified with the Jorgenson-Wilcoxen model.

Overview of the Jorgenson-Wilcoxen Model

The discussion below focuses on those characteristics of the Jorgenson-Wilcoxen model that have important implications for its use in the assessment of environmental regulations (see Table A-1). The J/W model is a detailed dynamic general equilibrium model of the U.S. economy designed for medium run analysis of regulatory and tax policy (Jorgenson and Wilcoxen [1990a]). It provides projections of key macroeconomic variables, such as GNP and aggregate consumption, as well as energy flows between economic sectors. As a result, the model is particularly useful for examining how the structure of the economy could change in response to changes in re-

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[3] The Congressional Budget Office report (1990) refers to an older (1981) version of the Jorgenson model, not the current (1988) version. The approach to long-run dynamics differs between the two models. The newer Jorgenson-Wilcoxen model contains both the capital accumulation equation and the capital asset pricing equation. The 1981 version of the model contained only the capital accumulation equation.

Table A-1. Key Distinguishing Characteristics of the Jorgenson-Wilcoxen Model.

- Dynamic, general equilibrium, macroeconomic model of the U.S. economy.
- Econometrically estimated using historic data.
- Free mobility of a single type of capital and labor between industries.
- Detailed treatment of production and consumption.
- Rigorous representation of savings and investment.
- Endogenous model of technical change.
- Does not capture unemployment, underemployment, or the costs of moving capital from one industry to another.

source prices. For the purpose of this study, it has five key features: a detailed treatment of production and consumption, parameters estimated econometrically from historical data, an endogenous model of technical change, a rigorous representation of saving and investment, and free mobility of labor and capital between industries.

The first two features, industry and consumer detail and econometric estimation, allow the model to capture the effects of the CAA at each point in time for given levels of technology and the size of the economy's capital stock. A detailed treatment of production and consumption is important because the principal effects of the Clean Air Act fell most heavily on a handful of industries. The J/W model divides total U.S. production into 35 industries which allows the primary economic effects of the CAA to be captured. Econometric estimation is equally important because it ensures that the behavior of households and firms in the model is consistent with the historical record.

The model's second two features —its representations of technical change and capital accumulation— complement the model's intratemporal features by providing specific information on how the Act affected technical change and the accumulation of capital. Many analyses of environmental regulations overlook or ignore intertemporal effects but these effects can be very important. Jorgenson and Wilcoxen (1990a) suggests that the largest cost of all U.S. environmental regulations together was that the regulations reduced the rate of capital accumulation.

The model's last feature, free mobility of a single type of capital and a single type of labor, is important because it limits the model's ability to measure the short run costs of changes in policy. J/W is a full-employment model that describes the long-run dynamics of transitions from one equilibrium to another. Capital and labor are both assumed to be freely mobile between sectors (that is, they can be moved from one industry to another at zero cost) and to be fully used at all times. Over the medium to long run, this is a reasonable assumption, but in the short run it is too optimistic. In particular, the model will understate the short run costs of a change in policy because it does not capture unemployment, underemployment, or the costs of moving capital from one industry to another. A single rate of return on capital exists that efficiently allocates the capital in each period among sectors. Similarly, a single equilibrium wage rate allocates labor throughout the economy.

Structure of the Jorgenson-Wilcoxen Model

The J/W model assesses a broad array of economic effects of environmental regulations. Direct costs are captured as increased expenditures on factors of production —capital, labor, energy and materials— that the various industries must make to comply with the regulations, as well as additional out-of-pocket expenditures that consumers must make. Indirect costs are captured as general equilibrium effects that occur throughout the economy as the prices of factors of production change (e.g., energy prices). Also, the rate of technological change can respond to changes in the prices of factors of production, causing changes in productivity (Jorgenson and Fraumeni, 1981).

The model is divided into four major sectors: the business, household, government, and rest-of-the-world sectors. The business sector is further subdivided into 35 industries (see Table A-2).[4] Each sector produces a primary product, and some produce secondary products. These outputs serve as inputs to the production processes of the other industries, are used for investment, satisfy final demands by the household and government sectors, and are exported. The model also allows for imports from the rest of the world.

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[4] The 35 industries roughly correspond to a two-digit SIC code classification scheme.

Table A-2. Definitions of Industries Within the J/W Model.

Industry Number	Description
1	Agriculture, forestry, and fisheries
2	Metal mining
3	Coal mining
4	Crude petroleum and natural gas
5	Nonmetallic mineral mining
6	Construction
7	Food and kindred products
8	Tobacco manufacturers
9	Textile mill products
10	Apparel and other textile products
11	Lumber and wood products
12	Furniture and fixtures
13	Paper and allied products
14	Printing and publishing
15	Chemicals and allied products
16	Petroleum refining
17	Rubber and plastic products
18	Leather and leather products
19	Stone, clay, and glass products
20	Primary metals
21	Fabricated metal products
22	Machinery, except electrical
23	Electrical machinery
24	Motor vehicles
25	Other transportation equipment
26	Instruments
27	Miscellaneous manufacturing
28	Transportation and warehousing
29	Communication
30	Electric utilities
31	Gas utilities
32	Trade
33	Finance, insurance, and real estate
34	Other services
35	Government enterprises

The Business Sector

The model of producer behavior allocates the value of output of each industry among the inputs of the 35 commodity groups, capital services, labor services, and noncompeting imports. Output supply and factor demands of each sector are modeled as the results of choices made by wealth maximizing, price taking firms which are subject to technological constraints. Firms have perfect foresight of all future prices and interest rates. Production technologies are represented by econometrically estimated cost functions that fully capture factor substitution possibilities and industry-level biased technological change.

Capital and energy are specified separately in the factor demand functions of each industry. The ability of the model to estimate the degree of substitutability between factor inputs facilitates the assessment of the effect of environmental regulations. A high degree of substitutability between inputs implies that the cost of environmental regulation is low, while a low degree of substitutability implies high costs of environmental regulation. Also, different types of regulations lead to different responses on the part of producers. Some regulations require the use of specific types of equipment. Others regulations restrict the use of particular factor inputs; for example, through restrictions on the combustion of certain types of fuels. Both of these effects can change the rate of productivity growth in an industry through changes in factor prices.

The Household Sector

In the model of consumer behavior, consumer choices between labor and leisure and between consumption and saving are determined. A system of individual, demographically defined household demand functions are also econometrically estimated. Household consumption is modeled as a three stage optimization process. In the first stage households allocate lifetime wealth to full consumption in current and future time periods to maximize intertemporal utility. Lifetime wealth includes financial wealth, discounted labor income, and the imputed value of leisure. Households have perfect foresight of future prices and interest rates. In the second stage, for each time period full consumption is allocated between goods and services and leisure to maximize intratemporal utility. This yields an allocation of a household's time endowment between the labor market (giving rise to labor supply and labor income) and leisure time and demands for goods and services. In the third stage, personal consumption expenditures are allocated among capital, labor, noncompeting imports and the outputs of the 35 production sectors to maximize a subutility function for goods consumption. As with the business sector, substitution possibilities exist in consumption decisions. The model's flexibility enables it to capture the substitution of nonpolluting products for polluting ones that may be induced by environmental regulations. Towards this end, purchases of energy and capital services by households are specified separately within the consumer demand functions for individual commodities.

It is important to be clear regarding the notions of labor supply and demand within the J/W model, and what is meant by "employment" throughout this report. Labor demands and supplies are represented as quality-adjusted hours denominated in constant dollars. The labor market clears in each period; the quantity of labor services offered by households is absorbed fully by the economy's producing sectors. However, inferences regarding the number of persons employed require information on labor quality and work-hours per person over time and across simulations. Neither of these are explicitly modeled.

The Government Sector

The behavior of government is constrained by exogenously specified budget deficits. Government tax revenues are determined by exogenously specified tax rates applied to appropriate transactions in the business and household sectors. Levels of economic activity in these sectors are endogenously determined. Capital income from government enterprises (determined endogenously), and nontax receipts (given exogenously), are added to tax revenues to obtain total government revenues. Government expenditures adjust to satisfy the exogenous budget deficit constraint.

The Rest-of-the-World Sector

The current account balance is exogenous, limiting the usefulness of the model to assess trade competitiveness effects. Imports are treated as imperfect substitutes for similar domestic commodities and compete on price. Export demands are functions of foreign incomes and ratios of commodity prices in U.S. currency to the exchange rate. Import prices, foreign incomes, and tariff policies are exogenously specified. Foreign prices of U.S. exports are determined endogenously by domestic prices and the exchange rate. The exchange rate adjusts to satisfy the exogenous constraint on net exports.

Environmental Regulation, Investment, and Capital Formation

Environmental regulations have several important effects on capital formation. At the most obvious level, regulations often require investment in specific pieces of pollution abatement equipment. If the economy's pool of savings were essentially fixed, the need to invest in abatement equipment would reduce, or crowd out, investment in other kinds of capital on a dollar for dollar basis. On the other hand, if the supply of savings were very elastic then abatement investments might not crowd out other investment at all. In the J/W model, both the current account and government budget deficits are fixed exogenously so any change in the supply of funds for domestic investment must come from a change in domestic savings. Because households choose consumption, and hence savings, to maximize a lifetime utility function, domestic savings will be somewhat elastic. Thus, abatement investment will crowd out other investment, although not on a dollar for dollar basis.

The J/W assumption that the current account does not change as a result of environmental regulation is probably unrealistic, but it is not at all clear that this biases the crowding out effects in any particular direction. By itself, the need to invest in abatement capital would tend to raise U.S. interest rates and draw in foreign savings. To the extent this occurred, crowding out would be reduced. At the same time, however, regulation reduces the profitability of domestic firms. This effect would tend to lower the return on domestic assets, leading to a reduced supply of foreign savings which would exacerbate crowding out. Which effect dominates is an empirical question beyond the scope of this study.

In additional to crowding out ordinary investment, environmental regulation also has a more subtle effect on the rate of capital formation. Regulations raise the prices of intermediate goods used to produce new capital. This leads to a reduction in the number of capital goods which can be purchased with a given pool of savings. This is not crowding out in the usual sense of the term, but it is an important means by which regulation reduces capital formation.[5]

The General Equilibrium

The J/W framework contains intertemporal and intratemporal models (Jorgenson and Wilcoxen [1990c]). In any particular time period, all markets clear. This market clearing process occurs in response to any changes in the levels of variables that are specified exogenously to the model. The interactions among sectors determine, for each period, aggregate domestic output, capital accumulation, employment, the composition of output, the allocation of output across different household types, and other variables.

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[5] Wilcoxen (1988) suggests that environmental regulation may actually lead to a "crowding in" phenomenon. Wilcoxen examined the effects of regulation at the firm level, and introduced costs into the model related to the installation of capital. He found that when firms shut down their plants to install environmental capital, they take account of the adjustment costs and often concurrently replace other older capital equipment. This effect, however, is not captured in the current version of the Jorgenson-Wilcoxen model.

The model also produces an intertemporal equilibrium path from the initial conditions at the start of the simulation to the stationary state. (A stationary solution for the model is obtained by merging the intertemporal and intratemporal models.) The dynamics of the J/W model have two elements: An accumulation equation for capital, and a capital asset pricing equation. Changes in exogenous variables cause several adjustments to occur within the model. First, the single stock of capital is efficiently allocated among all sectors, including the household sector. Capital is assumed to be perfectly malleable and mobile among sectors, so that the price of capital services in each sector is proportional to a single capital service price for the economy as a whole. The value of capital services is equal to capital income. The supply of capital available in each period is the result of past investment, i.e., capital at the end of each period is a function of investment during the period and capital at the beginning of the period. This capital accumulation equation is backward-looking and captures the effect of investments in all past periods on the capital available in the current period.

The capital asset pricing equation specifies the price of capital services in terms of the price of investment goods at the beginning and end of each period, the rate of return to capital for the economy as a whole, the rate of depreciation, and variables describing the tax structure for income from capital. The current price of investment goods incorporates an assumption of perfect foresight or rational expectations. Under this assumption, the price of investment goods in every period is based on expectations of future capital service prices and discount rates that are fulfilled by the solution of the model. This equation for the investment goods price in each time period is forward-looking.[6]

One way to characterize the J/W model —or any other neoclassical growth model— is that the short-run supply of capital is perfectly inelastic, since it is completely determined by past investment. However, the supply of capital is perfectly elastic in the long run. The capital stock adjusts to the time endowment, while the rate of return depends only on the intertemporal preferences of the household sector.

A predetermined amount of technical progress also takes place that serves to lower the cost of sectoral production. Finally, the quality of labor is enhanced, giving rise to higher productivity and lower costs of production.

Given all of these changes, the model solves for a new price vector and attains a new general equilibrium. Across all time periods, the model solves for the time paths of the capital stock, household consumption, and prices. The outcomes represent a general equilibrium in all time periods and in all markets covered by the J/W model.

Configuration of the No-control Scenario

One of the difficulties in describing the no-control scenario is ascertaining how much environmental regulation would have been initiated by state and local governments in the absence of a federal program. It may reasonably be argued that many state and local governments would have initiated their own control programs in the absence of a federal role. This view is further supported by the fact that many states and localities have, in fact, issued rules and ordinances which are significantly more stringent and encompassing than federal minimum requirements. However, it may also be argued that the federal CAA has motivated a substantial number of stringent state and local control programs.

Specifying the range and stringency of state and local programs that would have occurred in the absence of the federal CAA would be almost entirely speculative. For example, factors which would complicate developing assumptions about stringency and scope of unilateral state and local programs include: (i) the significance of federal funding to support state and local program development; (ii) the influence of more severe air pollution episodes which might be expected in the absence of federally-mandated controls; (iii) the potential emergence of pollution havens, as well as anti-pollution havens, motivated by local political and economic conditions; (iv) the influence of federally-sponsored research on the development of pollution effects information and control technologies; and (v) the need to make specific assumptions about individual state and local control levels for individual pollutants to allow estimation of incremental reductions attributable to federal control programs.

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[6] The price of capital assets is also equal to the cost of production, so that changes in the rate of capital accumulation result in an increase in the cost of producing investment goods. This has to be equilibrated with the discounted value of future rentals in order to produce an intertemporal equilibrium. The rising cost of producing investment is a cost of adjusting to a new intertemporal equilibrium path.

Another complication associated with the no-control scenario is the treatment of air pollution control requirements among the major trading partners of the U.S. Real-world manifestation of a no-control scenario would imply that public health and environmental goals were not deemed sufficiently compelling by U.S. policy makers. Under these conditions, major trading partners of the U.S. in Japan, Europe, and Canada may well reach similar policy conclusions. Simply put, if the U.S. saw no need for air pollution controls, there is little reason to assume other developed industrial countries would have either. In this case, some of the estimated economic benefits of reducing or eliminating air pollution controls in the U.S. would not materialize because U.S. manufacturers would not necessarily gain a production cost advantage over foreign competitors. However, like the question of state and local programs in the absence of a federal program, foreign government policies under a no-control scenario would be highly speculative.

Given the severity of these confounding factors, the only analytically feasible assumptions with respect to the no-control scenario are that (a) no new control programs would have been initiated after 1970 by the states or local governments in the absence of a federal role, and (b) environmental policies of U.S. trading partners remain constant regardless of U.S. policy.

Elimination of Compliance Costs in the No-Control Case

Industries that are affected by environmental regulations can generally respond in three ways: (i) with process changes (e.g., fluidized bed combustion); (ii) through input substitution (e.g., switching from high sulfur coal to low sulfur coal); and (iii) end-of-pipe abatement (e.g., the use of electrostatic precipitation to reduce the emissions of particulates by combustion equipment).[7] Clean air regulations have typically led to the latter two responses, especially in the short run. End-of-pipe abatement is usually the method of choice for existing facilities, since modifying existing production processes can be costly. This approach is also encouraged by EPA's setting of standards based on the notion of "best available technology" (Freeman, 1978).

All three possible responses may lead to: (i) unanticipated losses to equity owners; (ii) changes in current output; and (iii) changes in long-run profitability. If firms were initially maximizing profits, then any of the above three responses will increase its costs. Fixed costs of investment will be capitalized immediately. This will result in a loss to owners of equity when regulations are introduced. As far as firms are concerned, this is just like a lump sum tax on sunk capital. Such effects will not affect growth or efficiency. However, regulations could also change marginal costs and therefore current output. In addition, they could change profits (i.e., the earnings of capital), and thus affect investment. Both of these effects will reduce the measured output of the economy.

On the consumption side, environmental regulations change consumers' expectations of their lifetime wealth. In the no-control scenario of this assessment, lifetime wealth increases. This causes an increase in consumption. In fact, with perfect foresight, consumption rises more in earlier time periods. This also results in a change in savings.

Capital Costs - Stationary Sources

To appropriately model investment in pollution control requires a recognition that the CAA had two different effects on capital markets. First, CAA regulations led to the retrofitting of existing capital stock in order to meet environmental standards. In the no-control scenario, these expenditures do not occur. Instead, the resources that were invested in pollution abatement equipment to retrofit existing sources are available to go to other competing investments. Thus, at each point in time, these resources might go to investments in capital in the regulated industry, or may go into investments in other industries, depending upon relative rates of return on those investments. This will affect the processes of capital formation and deepening.

Second, the CAA placed restrictions on new sources of emissions. When making investment decisions, firms take into account the additional cost of pollution abatement equipment. Effectively, the

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[7] Regulation may also affect the rate of investment, and change the rate of capital accumulation.

"price" of investment goods is higher because more units of capital are required to produce the same amount of output. In the no-control scenario, there are no restrictions on new sources and hence no requirements for pollution control expenditures. Effectively, the "price" of investment goods is lower. Thus, at each point in time, investors are faced with a lower price of investment goods. This results in a different profile for investment over time.

Operating and Maintenance Costs - Stationary Sources

In addition to purchasing pollution abatement equipment, firms incurred costs to run and maintain the pollution abatement equipment. In the no-control scenario, resources used to pay for these operating and maintenance (O&M) costs are freed up for other uses. The model assumes that the resources required to run and maintain pollution control equipment are in the same proportions as the factor inputs used in the underlying production technology. For example, if 1 unit of labor and 2 units of materials are used to produce 1 unit of output, then one-third of pollution control O&M costs are allocated to labor and two-thirds are allocated to materials. These adjustments were introduced at the sector level. O&M expenditures are exclusive of depreciation charges and offset by any recovered costs.

Capital Costs - Mobile Sources

Capital costs associated with pollution control equipment were represented by changing costs for motor vehicles (sector 24) and other transportation equipment (sector 26). Prices (unit costs) were reduced in proportion to the value of the pollution control devices contained in cars, trucks, motorcycles, and aircraft.

Operating and Maintenance - Mobile Sources

Prices for refined petroleum products (sector 16) were changed to reflect the resource costs associated with producing unleaded and reduced lead gasoline (fuel price penalty), the change in fuel economy for vehicles equipped with pollution control devices (fuel economy penalty), and the change in fuel economy due to the increased fuel density of lower leaded and no lead gasoline (fuel economy credit). Third, inspection and maintenance costs and a maintenance credit associated with the use of unleaded and lower leaded (i.e., unleaded and lower leaded gasoline is less corrosive, and therefore results in fewer muffler replacements, less spark plug corrosion, and less degradation of engine oil) were represented as changes in prices for other services (sector 34).

Direct Compliance Expenditures Data

Sources of Cost Data

Cost data for this study are derived primarily from the 1990 *Cost of Clean* report. EPA publishes cost data in response to requirements of the Clean Air and Clean Water Acts. The following subsections describe *Cost of Clean* data in detail, as well as adjustments made to the data and data from other sources.

Cost of Clean Data

EPA is required to compile and publish public and private costs resulting from enactment of the Clean Air Act and the Clean Water Act. The 1990 *Cost of Clean* report presents estimates of historical pollution control expenditures for the years 1972 through 1988 and projected future costs for the years 1989 through 2000. This includes federal, state, and local governments as well as the private sector. Estimates of capital costs, operation and maintenance (O&M) costs, and total annualized costs for five categories of environmental media, including air, water, land, chemical, and multi-media, are presented. It should be noted that these estimates represent direct regulatory implementation and compliance costs rather than social costs. The *Cost of Clean* relied on data from two governmental sources, the EPA and the U.S. Department of Commerce (Commerce).

EPA Data

EPA expenditures were estimated from EPA budget justification documents.[8] Estimates of capital and operating costs resulting from new and forthcoming regulations were derived from EPA's Regulatory Impact Analyses (RIAs). RIAs have been prepared prior to the issuance of all major regulations since 1981. Finally, special analyses conducted by EPA program offices or contractors were used when other data sources did not provide adequate or reliable data.

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[8] The main source of data for EPA expenditures is the *Justification of Appropriation Estimates for Committee on Appropriations.*

Commerce Data

Data collected by Commerce were used extensively in the *Cost of Clean* for estimates of historical pollution control expenditures made by government agencies other than EPA and by the private sector. Two Commerce agencies, the Bureau of Economic Analysis (BEA) and the Bureau of the Census (Census), have collected capital and operating costs for compliance with environmental regulations since the early 1970's. Commerce is, in fact, the primary source of original survey data for environmental regulation compliance costs. Commerce publishes a number of documents that report responses to surveys and comprise most of the current domain of known pollution abatement and control costs in the United States, including:

- A series of articles entitled "Pollution Abatement and Control Expenditures" published annually in the *Survey of Current Business* by BEA (BEA articles);
- A series of documents entitled "Pollution Abatement Costs and Expenditures" published annually in the *Current Industrial Reports* by Census (PACE reports); and
- A series of documents entitled *Government Finances* published annually by Census (Government Finances).

BEA articles contain data derived from a number of sources, including two key agency surveys —the "Pollution Abatement Costs and Expenditures Survey" (PACE Survey) and the "Pollution Abatement Plant and Equipment Survey" (PAPE Survey)—which are conducted annually by Census for BEA. Data have been reported for 1972 through 1987.[9]

PACE reports have been published annually since 1973 with the exception of 1987. Figures for 1987 were estimated on the basis of historical shares within total manufacturing. These reports contain expenditure estimates derived from surveys of about 20,000 manufacturing establishments. Pollution abatement expenditures for air, water and solid waste are reported by state and Standard Industrial Code (SIC) at the four-digit level. According to Census, surveys conducted since 1976 have not included establishments with fewer than 20 employees because early surveys showed that they contributed only about 2 percent to the pollution estimates while constituting more than 10 percent of the sample size.

Each year Census conducts a survey of state, local, and county governments; and survey results are published in *Government Finances.* Census asks government units to report revenue and expenditures, including expenditures for pollution control and abatement.

Non-EPA Federal expenditures were estimated from surveys completed by federal agencies detailing their pollution control expenditures, which are submitted to BEA. Private sector air pollution control expenditures, as well as state and local government air pollution expenditures, were taken from BEA articles.

Stationary Source Cost Data

Capital Expenditures Data

Capital expenditures for stationary air pollution control are made by factories and electric utilities for plant and equipment that abate pollutants through end-of-line (EOL) techniques or that reduce or eliminate the generation of pollutants through changes in production processes (CIPP). For the purposes of this report EOL and CIPP expenditures are aggregated.[10] Table A-3 summarizes capital expenditures for stationary air pollution control, categorized as "nonfarm business" or "government enterprise" expenditures.

Nonfarm business capital expenditures consist of plant and equipment expenditures made by 1) manufacturing companies, 2) privately and cooperatively owned electric utilities, and 3) other nonmanufacturing companies. "Government enterprise" is, according to BEA, an agency of the government whose operating costs, to a substantial extent, are covered by the sale of goods and services. Here, government enterprise means specifically government enterprise electric

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[9] The most recent BEA article used as a source for air pollution control costs in the Cost of Clean was "Pollution Abatement and Control Expenditures, 1984-87" in *Survey of Current Business*, June 1989.

[10] Survey respondents to the Census annual Pollution Abatement Surveys report the difference between expenditures for CIPP and what they would have spent for comparable plant and equipment without pollution abatement features. Disaggregated capital expenditures by private manufacturing establishments can be found in annual issues of Census reports.

Table A-3. Estimated Capital and O&M Expenditures for Stationary Source Air Pollution Control (millions of current dollars).

	Nonfarm Business		Government Enterprise	
Year	Cap.[a]	O&M[b]	Cap.[c]	O&M[d]
1972	2,172		63	
1973	2,968	1,407	82	29
1974	3,328	1,839	104	56
1975	3,914	2,195	102	45
1976	3,798	2,607	156	58
1977	3,811	3,163	197	60
1978	3,977	3,652	205	72
1979	4,613	4,499	285	106
1980	5,051	5,420	398	148
1981	5,135	5,988	451	135
1982	5,086	5,674	508	141
1983	4,155	6,149	422	143
1984	4,282	6,690	416	147
1985	4,141	6,997	328	189
1986	4,090	7,116	312	140
1987	4,179	7,469	277	130
1988	4,267	7,313	243	161
1989	4,760	7,743	235	173
1990	4,169	8,688	226	154

Sources:
a. Non-farm capital expenditures for 1972-87 are from *Cost of Clean*, Table B-1, line 2.
b. Non-farm O&M expenditures for 1973-85 are from *Cost of Clean*, Table B-1, line 8.
c. Government enterprise capital expenditures for 1972-87 are from *Cost of Clean,* Table B-9, line 1.
d. Government enterprise O&M expenditures for 1973-85 are from *Cost of Clean,* Table B-9, line 5.
All other reported expenditures are EPA estimates.

utilities. Government enterprise capital expenditures are pollution abatement expenditures made by publicly owned electric utilities.[11]

Operation and Maintenance Expenditures Data

Stationary source O&M expenditures are made by manufacturing establishments, private and public electric utilities, and other nonmanufacturing businesses to operate air pollution abatement equipment. O&M expenditures for electric utilities are made up of two parts: 1) expenditures for operating air pollution equipment and 2) the additional expenditures associated with switching to alternative fuels that have lower sulfur content (fuel differential). Expenditures to operate air pollution abatement equipment are for the collection and disposal of flyash, bottom ash, sulfur and sulfur products, and other products from flue gases.[12] O&M expenditures are net of depreciation and payments to governmental units, and are summarized in Table A-3. O&M data were disaggregated to the two digit SIC level for use in the macroeconomic model.

For both capital and O&M expenditures, historical survey data were not available for each year through 1990 prior to publication of *Cost of Clean*. For the purpose of the section 812 analysis, EPA projected 1988-1990 capital expenditures and 1986-1990 O&M expenditures. Those projections were used in the macroeconomic simulation, and have been retained as cost estimates to ensure consistency between the macroeconomic results and the direct cost estimates. Since completion of the macroeconomic modeling, however, BEA has published expenditure estimates through 1990. A comparison of more recent BEA estimates with the EPA projections used in the section 812 analysis can be found in the "Uncertainties in the Cost Analysis" section, below.

Recovered Costs

"Recovered costs" are costs recovered (i.e., revenues realized) by private manufacturing establishments through abatement activities. According to instructions provided to survey participants by Census, recovered costs consist of 1) the value of materials or energy reclaimed through abatement activities that were reused in production and 2) revenue that was obtained from the sale of materials or energy reclaimed through abatement activities. Estimates of recovered costs were obtained from the PACE reports and are summarized in Table A-4. In this analysis, recovered costs were removed from total stationary source air pollution control O&M costs — that is, net O&M cost in any year would be O&M expenditures (see Table A-3) less recovered costs. Recovered cost data were disaggregated to the two digit SIC level for use in the macroeconomic model.

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[11] BEA calculates these expenditures using numbers obtained from Energy Information Agency (EIA) Form 767 on steam-electric plant air quality control.

[12] Farber, Kit D. and Gary L. Rutledge, "Pollution Abatement and Control Expenditures: Methods and Sources for Current-Dollar Estimates," Unpublished paper, Bureau of Economic Analysis, U.S. Department of Commerce, October 1989.

Table A-4. Estimated Recovered Costs for Stationary Source Air Pollution Control (millions of current dollars).

Year	PACE*	Estimated
1972		248
1973		199
1974		296
1975		389
1976		496
1977		557
1978		617
1979	750	750
1980	862	862
1981	1,000	997
1982	858	857
1983	822	822
1984	866	870
1985	767	768
1986	860	867
1987		987
1988	1,103	1,107
1989		1,122
1990		1,256

* Air cost recovered as reported in PACE
Source: "Pollution Abatement Costs and Expenditures" published annually in the Current Industrial Reports by Census.

Mobile Source Cost Data

Costs of controlling pollution emissions from motor vehicles were estimated by calculating the purchase price and O&M cost premiums associated with vehicles equipped with pollution abatement controls over the costs for vehicles not equipped with such controls. These costs were derived using EPA analyses, including EPA RIAs, the *Cost of Clean*, and other EPA reports.[13] This Appendix summarizes the section 812 mobile source compliance cost estimates and provides references to published data sources where possible. Further information on specific methods, analytical steps, and assumptions can be found in McConnell *et al.* (1995),[14] which provides a detailed description of the section 812 mobile source cost estimation exercise and compares the method and results to other similar analyses (including *Cost of Clean* (1990)).

Capital Expenditures Data

Capital expenditures for mobile source emission control are associated primarily with pollution abatement equipment on passenger cars, which comprise the bulk of all mobile sources of pollution. These capital costs reflect increasingly stringent regulatory requirements and improvements in pollution control technologies over time. Each of the following devices have been used at one time or another dating back to the Clean Air Act Amendments of 1965: air pumps, exhaust-gas recirculation valves, high altitude controls, evaporative emissions controls, and catalysts. The cost estimates for each component were computed on a per-vehicle basis by engineering cost analyses commissioned by EPA. The resulting per-vehicle capital costs were multiplied by vehicle production estimates to determine annual capital costs. Table A-5 summarizes mobile source capital costs.

Operation and Maintenance Expenditures Data

Costs for operation and maintenance of emission abatement devices include the costs of maintaining pollution control equipment plus the cost of vehicle inspection/maintenance programs. Operating costs per vehicle were multiplied by total vehicles in use to determine annual cost. Mobile source O&M costs are made up of three factors: 1) fuel price penalty, 2) fuel economy penalty, and 3) inspection and maintenance program costs as described below. These costs are mitigated by cost savings in the form of maintenance economy and fuel density economy. Table A-6 summarizes mobile source O&M expenditures and cost savings by categories, with net O&M costs summarized above in Table A-5. The following sections describe the components of the mobile source O&M cost estimates.

Fuel Price Penalty

Historically, the price of unleaded fuel has been several cents per gallon higher than the price of leaded fuel. CAA costs were calculated as the difference be-

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[13] A complete listing of sources used in calculating mobile source capital and operating expenditures can be found in *Environmental Investments: The Cost of a Clean Environment*, Report of the Administrator of the Environmental Protection Agency to the Congress of the United State, EPA-230-11-90-083, November 1990.

[14] *Evaluating the Cost of Compliance with Mobile Source Emission Control Requirements: Retrospective Analysis*, Resources for the Future Discussion Paper, 1995. Note that McConnell *et al.* refer to the section 812 estimates as: *Cost of Clean* (1993, unpublished).

Table A-5. Estimated Capital and Operation and Maintenance Expenditures for Mobile Source Air Pollution Control (millions of current dollars).

Year	Capital[a]	O&M[b]
1973	276	1,765
1974	242	2,351
1975	1,570	2,282
1976	1,961	2,060
1977	2,248	1,786
1978	2,513	908
1979	2,941	1,229
1980	2,949	1,790
1981	3,534	1,389
1982	3,551	555
1983	4,331	-155
1984	5,679	-326
1985	6,387	337
1986	6,886	-1,394
1987	6,851	-1,302
1988	7,206	-1,575
1989	7,053	-1,636
1990	7,299	-1,816

Sources:
a. Capital exp.: *Cost of Clean*, Tables C-2 to C-9, line 3 on each; Tables C-2A to C-9A, line 10 on each; converted from $1986 to current dollars.
b. O&M exp.: EPA analyses based on sources and methods in: *Costs and Benefits of Reducing Lead in Gasoline: Final Regulatory Impact Analysis*, U.S. Environmental Protection Agency, Office of Policy Analysis, EPA-230-05-85-006, February 1985; and *Cost of Clean*.

tween the cost of making unleaded gasoline and leaded gasoline with lower lead levels and the cost of making only leaded gasoline with a lead content set at pre-regulatory levels. These cost estimates were developed using a linear programming model of the refinery industry. Prices of crude oil and other unfinished oils, along with the prices of refinery outputs, were adjusted annually according to price indices for imported crude oil over the period of analysis. The relative shares of leaded and unleaded gasoline and the average lead content in leaded gasoline also were adjusted annually according to the historical record.

These estimates may tend to understate costs due to a number of biases inherent in the analysis process. For example, the refinery model was allowed to optimize process capacities in each year. This procedure is likely to understate costs because regulatory requirements and market developments cannot be perfectly anticipated over time. This procedure resulted in estimates that are about ten percent less than estimates in other EPA reports.[15] However, new process technologies that were developed in the mid-1980s were not reflected in either the base case or regulatory case runs. It is reasonable to expect that regulatory requirements would have encouraged development of technologies at a faster rate than would have occurred otherwise.

Fuel Economy Penalty

The fuel economy penalty benefit is the cost associated with the increased/decreased amount of fuel used by automobiles with air pollution control devices (all else being equal). An assumption that can be made is that the addition of devices, such as catalytic con-

Table A-6. O&M Costs and Credits (millions of current dollars).

Year	Fuel Price Penalty	Fuel Econ. Penalty	Net I& M*	Total Costs
1973	91	1700	-26	1765
1974	244	2205	-98	2351
1975	358	2213	-289	2282
1976	468	2106	-514	2060
1977	568	1956	-738	1786
1978	766	1669	-1527	908
1979	1187	1868	-1826	1229
1980	1912	1998	-2120	1790
1981	2181	1594	-2386	1389
1982	2071	1026	-2542	555
1983	1956	628	-2739	-155
1984	2012	313	-2651	-326
1985	3057	118	-2838	337
1986	2505	-40	-3859	-1394
1987	2982	-158	-4126	-1302
1988	3127	-210	-4492	-1575
1989	3476	-318	-4794	-1636
1990	3754	-481	-5089	-1816

* Inspection and maintenance costs less fuel density savings and maintenance savings.

Sources: All results are presented in Jorgenson *et al.* (1993), pg. A.17. FPP results are based on a petroleum refinery cost model run for the retrospective analysis. FEP and Net I&M are based on data and methods from *Costs and Benefits of Reducing Lead in Gasoline: Final Regulatory Impact Analysis*, U.S. Environmental Protection Agency, Office of Policy Analysis, EPA-230-05-85-006, February 1985; and *Cost of Clean* (1990). Specific analytic procedures are summarized in McConnell *et al.* (1995).

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[15] Costs and Benefits of Reducing Lead in Gasoline: Final Regulatory Impact Analysis, U.S. Environmental Protection Agency, Office of Policy Analysis, EPA-230-05-85-006, February 1985.

verters, decrease automobile fuel efficiency.[16] If this assumption is true, air pollution control devices increase the total fuel cost to consumers. An alternative assumption is that the use of catalytic converters has increased fuel economy. This increase has been attributed in large measure to the feedback mechanism built into three-way catalytic converters.[17] Under this assumption, the decrease in total fuel cost to consumers is considered a benefit of the program.

For the purposes of this study, sensitivity analyses were performed using data presented in the *Cost of Clean* report. These analyses were conducted to evaluate the significance of assumptions about the relationship between mile per gallon (MPG) values for controlled automobiles and MPG values for uncontrolled cars. Based on results of these and other analyses, fuel economy was assumed to be equal for controlled and uncontrolled vehicles from 1976 onward. This may bias the cost estimates although in an unknown direction.

Inspection and Maintenance Programs

Inspection and maintenance programs are administered by a number of states. Although these programs are required by the Clean Air Act, the details of administration were left to the discretion of state or local officials. The primary purpose of inspection and maintenance programs is to identify cars that require maintenance —including cars that 1) have had poor maintenance, 2) have been deliberately tampered with or had pollution control devices removed, or 3) have used leaded gasoline when unleaded is required— and force the owners of those cars to make necessary repairs or adjustments.[18] Expenditures for inspection and maintenance were taken from the *Cost of Clean.*

Beneficial effects of the mobile source control program associated with maintenance and fuel density were also identified. These cost savings were included in this study as credits to be attributed to the mobile source control program. Credits were estimated based on an EPA study,[19] where more detailed explanations may be found.

Maintenance Credits

Catalytic converters require the use of unleaded fuel, which is less corrosive than leaded gasoline. On the basis of fleet trials, the use of unleaded or lower leaded gasoline results in fewer muffler replacements, less spark plug corrosion, and less degradation of engine oil, thus reducing maintenance costs. Maintenance credits account for the majority of the direct (non-health) economic benefits of reducing the lead concentration in gasoline.

Fuel Density Credits

The process of refining unleaded gasoline increases its density. The result is a gasoline that has higher energy content. Furthermore, unleaded gasoline generates more deposits in engine combustion chambers, resulting in slightly increased compression and engine efficiency. Higher energy content of unleaded gasoline and increased engine efficiency from the used of unleaded gasoline yield greater fuel economy and therefore savings in refining, distribution, and retailing costs.

Other Direct Cost Data

The *Cost of Clean* report includes several other categories of cost that are not easily classified as either stationary source or mobile source expenditures. Federal and state governments incur air pollution abatement costs; additionally, federal and state governments incur costs to develop and enforce CAA regulations. Research and development expenditures by the federal government, state and local governments, and (especially) the private sector can be attributed to the CAA. These data are summarized by year in Table A-7.

Unlike the other private sector expenditure data used for this analysis, the survey data used as a source for private sector R&D expenditures cannot be disaggregated into industry-specific expenditure totals. Consequently, private sector R&D expenditures are

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[16] Memo from Joel Schwartz (EPA/OPPE) to Joe Somers and Jim DeMocker dated December 12, 1991, and entitled “Fuel Economy Benefits.” Schwartz states that since this analysis is relative to a no Clean Air Act baseline, not a 1973 baseline, fuel economy benefits are not relevant. In the absence of regulation, tuning of engines for maximum economy would presumably be optimal in the base case as well.

[17] Memo from Joseph H. Somers, EPA Office of Mobile Sources, to Anne Grambsch (EPA/OPPE) and Joel Schwartz (EPA/OPPE) entitled “Fuel Economy Penalties for section 812 Report,” December 23, 1991.

[18] Walsh, Michael P., “Motor Vehicles and Fuels: The Problem,” *EPA Journal*, Vol. 17, No. 1, January/February 1991, p. 12.

[19] Schwartz, J., *et al. Costs and Benefits of Reducing Lead in Gasoline: Final Regulatory Impact Analysis*, U. S. Environmental Protection Agency, Economic Analysis Division, Office of Policy Analysis, February 1985.

Table A-7. Other Air Pollution Control Expenditures (millions of current dollars).

Year	Abatement		Regulations and Monitoring		Research and Development			Total
	Fed.[a]	State & Local[b]	Fed.[c]	State & Local[d]	Private[e]	Fed.[f]	State & Local[g]	
1973	47	0	50	115	492	126	6	836
1974	56	0	52	131	520	100	7	866
1975	88	1	66	139	487	108	8	897
1976	105	1	69	135	562	131	6	1,009
1977	106	1	80	161	675	144	7	1,174
1978	90	0	93	183	805	146	8	1,325
1979	103	0	100	200	933	105	7	1,448
1980	95	0	122	207	851	130	5	1,410
1981	85	0	108	226	798	131	0	1,348
1982	87	0	93	230	761	126	2	1,229
1983	136	4	88	239	691	133	6	1,297
1984	115	14	101	250	665	165	4	1,314
1985	98	12	103	250	775	247	3	1,488
1986	67	14	106	307	833	217	4	1,548
1987	80	15	110	300	887	200	2	1,594
1988	65	10	120	320	934	220	1	1,670
1989	70	12	130	360	984	230	2	1,788
1990	71	13	133	343	749	231	2	1,542

Sources:

a. Federal government abatement expenditures: 1973-82, "Pollution Abatement and Control Expenditures", Survey of Current Business (BEA) July 1986 Table 9 line 13; 1983-87, BEA June 1989 Table 7 line 13; 1988-90, BEA May 1995 Table 7 line 13.

b. State and local abatement expenditures: 1973-87, *Cost of Clean*, Table B-9 line 2; 1988-90, BEA May 1995 Table 7 line 14.

c. Federal government "regs/monitoring" expenditures: 1973-82, BEA July 1986, Table 9 line 17; 1983-87, BEA June 1989 Table 6 line 17; 1988-90, BEA May 1995 Table 7 line 17.

d. State and local government "regs/monitoring" expenditures: 1973-87, *Cost of Clean*, Table B-9 line 3; 1988-90, BEA May 1995 Table 7 line 18.

e. Private sector R&D expenditures: 1973-86, BEA May 1994 Table 4 (no line #) [total R&D expenditures in $1987 are converted to current dollars using the GDP price deflator series found elsewhere in this Appendix -- netting out public sector R&D leaves private sector expenditures]; 1987-90, BEA May 1995 Table 7 line 20.

f. Federal government R&D expenditures: 1973-82, BEA July 1986 Table 9 line 21; 1983-87, BEA June 1989 Table 6 line 21; 1988-90, BEA May 1995, Table 7 line 21.

g. State and local government R&D expenditures: 1973-87, *Cost of Clean*, Table B-9 line 4; 1988-90, BEA May 1995 Table 7 line 22.

omitted from the macroeconomic modeling exercise (the macro model is industry-specific). The R&D expenditures are, however, included in aggregate cost totals used in the benefit-cost analysis.

The *Cost of Clean* and the series of articles "Pollution Abatement and Control Expenditures" in the Survey of Current Business (various issues) are the data sources for "Other Air Pollution Control Expenditures." State and local expenditures through 1987 are found in *Cost of Clean*; 1988-90 expenditures are from more recent issues of the Survey of Current Business (BEA). Federal government expenditures are from BEA (various issues). Private R&D expenditures were reported in *Cost of Clean*. Since publication of *Cost of Clean*, however, BEA has revised its private sector R&D expenditure series (BEA, 1994 and 1995). Since private R&D expenditures were not included in the macroeconomic modeling exercise, the revised series can be (and has been) used without causing inconsistency with other portions of the section 812 analysis.

Assessment Results

Compliance Expenditures and Costs

Compliance with the CAA imposed direct costs on businesses, consumers, and governmental units, and triggered other expenditures such as governmental regulation and monitoring costs and expenditures for research and development by both government and industry. As shown in Table A-8, annual CAA compliance expenditures – including R&D, etc.– over the period from 1973 to 1990 were remarkably stable[20], ranging from about $20 billion to $25 billion in inflation-adjusted 1990 dollars (expenditures are adjusted to 1990 dollars through application of the GDP Implicit Price Deflator). This is equal to approximately one third of one percent of total domestic output during that period, with the percentage falling from one half of one percent of total output in 1973 to one quarter of one percent in 1990.

Although useful for many purposes, a summary of direct annual expenditures is not the best cost measure to use when comparing costs to benefits. Capital expenditures are investments, generating a stream of benefits (and opportunity cost) over the life of the investment. The appropriate accounting technique to use for capital expenditures in a cost/benefit analysis is to *annualize* the expenditure — i.e., to spread the capital cost over the useful life of the investment, applying a discount rate to account for the time value of money.

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[20] While total expenditures remained relatively constant over the period, the sector-specific data presented in Tables A-3 and A-5 above indicate that capital expenditures for stationary sources fell significantly throughout the period but that this decline was offset by significant increases in mobile source capital expenditures.

Table A-8. Summary of Expenditures and Conversion to 1990 Dollars (millions of dollars).

	CURRENT YEAR DOLLARS								1990 DOLLARS						
	Stationary		Rec. Costs	Mobile Source		Other	TOTAL EXP	GDP price defl.	Stationary		Rec. Costs	Mobile Source		Other	TOTAL EXP
	K	O&M		K	O&M				K	O&M		K	O&M		
1972	2,235	na	na	na	na	na	na	38.8	6,521						
1973	3,050	1,436	199	276	1,765	836	7,164	41.3	8,360	3,936	545	756	4,838	2,290	19,635
1974	3,432	1,895	296	242	2,351	866	8,490	44.9	8,653	4,778	746	610	5,927	2,184	21,405
1975	4,016	2,240	389	1,570	2,282	897	10,616	49.2	9,240	5,154	895	3,612	5,250	2,063	24,425
1976	3,954	2,665	496	1,961	2,060	1,009	11,153	52.3	8,558	5,768	1,074	4,244	4,459	2,183	24,139
1977	4,008	3,223	557	2,248	1,786	1,174	11,882	55.9	8,116	6,527	1,128	4,552	3,617	2,378	24,062
1978	4,182	3,724	617	2,513	908	1,325	12,035	60.3	7,851	6,991	1,158	4,718	1,705	2,487	22,593
1979	4,898	4,605	750	2,941	1,229	1,448	14,371	65.5	8,465	7,959	1,296	5,083	2,124	2,503	24,837
1980	5,449	5,568	862	2,949	1,790	1,410	16,304	71.7	8,603	8,791	1,361	4,656	2,826	2,226	25,741
1981	5,586	6,123	997	3,534	1,389	1,348	16,983	78.9	8,014	8,785	1,430	5,070	1,993	1,935	24,367
1982	5,594	5,815	857	3,551	555	1,299	15,957	83.8	7,557	7,855	1,158	4,797	750	1,755	21,555
1983	4,577	6,292	822	4,331	(155)	1,297	15,520	87.2	5,942	8,168	1,067	5,622	(201)	1,684	20,148
1984	4,698	6,837	870	5,679	(326)	1,314	17,332	91	5,844	8,505	1,082	7,064	(406)	1,634	21,560
1985	4,469	7,186	768	6,387	337	1,488	19,099	94.4	5,359	8,617	921	7,659	404	1,785	22,903
1986	4,402	7,256	867	6,886	(1,394)	1,548	17,831	96.9	5,142	8,477	1,013	8,044	(1,628)	1,809	20,831
1987	4,456	7,599	987	6,851	(1,302)	1,594	18,211	100	5,044	8,602	1,117	7,755	(1,474)	1,804	20,615
1988	4,510	7,474	1,107	7,206	(1,575)	1,670	18,178	103.9	4,914	8,143	1,206	7,851	(1,716)	1,819	19,805
1989	4,995	7,916	1,122	7,053	(1,636)	1,788	18,994	108.5	5,211	8,259	1,171	7,359	(1,707)	1,865	19,817
1990	4,395	8,842	1,256	7,312	(1,816)	1,542	19,019	113.2	4,395	8,842	1,256	7,312	(1,816)	1,542	19,019

K = Capital expenditures; O&M = Operation and Maintenance expenditures.
Rec. Costs = recovered costs. Total expenditures are the sum of stationary source, mobile source, and "other" expenditures, less recovered costs.
Stationary source expenditures are the sum of "Nonfarm Business" and "Government Enterprise" expenditures (from Table A-3).
To calculate expenditures in 1990 dollars, current year expenditures are multiplied by the ratio of the 1990 price deflator to the current year deflator. For example, 1989 expenditures are multiplied by (113.2/108.5).
Source for price deflator series: Economic Report of the President, February 1995, Table B-3.

Annualization Method

For this cost/benefit analysis, all capital expenditures have been annualized at 3 percent, 5 percent, and 7 percent (real) rates of interest. Therefore, "annualized" costs reported for any given year are equal to O&M expenditures (plus R&D, etc., expenditures, minus recovered costs) plus amortized capital costs (i.e., depreciation plus interest costs associated with the pre-existing capital *stock*) for that year. Stationary source air pollution control capital costs are amortized over twenty years; mobile source air pollution control costs are amortized over ten years. Capital expenditures are amortized using the formula for an annuity [that is, $r/(1-(1+r)^{-t})$, where r is the rate of interest and t is the amortization period].[21] Multiplying the expenditure by the appropriate annuity factor gives a constant annual cost to be incurred for t years, the present value of which is equal to the expenditure.

Due to data limitations, the cost analysis for this CAA retrospective starts in 1973, missing costs incurred in 1970-72. *Cost of Clean*, however, includes stationary source capital expenditures for 1972. In this analysis, amortized costs arising from 1972 capital investments *are included* in the 1973-1990 annualized costs, even though 1972 costs are not otherwise included in the analysis. Conversely, only a portion of the (e.g.) 1989 capital expenditures are reflected in the 1990 annualized costs — the remainder of the costs are spread through the following two decades, which fall outside of the scope of this study (similarly, benefits arising from emission reductions in, e.g., 1995 caused by 1990 capital investments are not captured by the benefits analysis). Table A-9 presents CAA compliance costs from 1973 to 1990, in 1990 dollars, with capital expenditures amortized at a five percent real interest rate. "Total" costs are the sum of stationary source, mobile source, and "other" costs, minus recovered costs.

Tables A-10 and A-11 provide details of the amortization calculation (using a five percent interest rate) for stationary sources and mobile sources, respectively. Similar calculations were performed to derive the annualized cost results using discount rates of three percent and seven percent.

The Stationary Source table reports a capital expenditure of $6,521 million for 1972 (in 1990 dollars). The cost is spread over the following twenty years (which is the assumed useful life of the investment) using a discount rate of five percent; thus, the amortization factor to be used is f(20)=0.0802. Multiplying $6,521 million by 0.0802 gives an annuity of $523 million. That annuity is noted on the first data row of the table, signifying that the 1972 expenditure of $6,521 million implies an annual cost of $523 million for the entire twenty-year period of 1973 to 1992 (the years following 1990 are not included on the tables, since costs incurred in those years are not included in this retrospective assessment). The first summary row near the bottom of the table (labeled "SUM") reports aggregate annualized capital costs: for 1973 (the first data column), capital costs are $523 million.

Capital expenditures in 1973 amounted to $8,360 million. Using the amortization technique explained above, one can compute an annualized cost of $671 million, incurred for the twenty-year period of 1974 to 1993. Aggregate annualized capital costs for 1974 include cost flows arising from 1972 and 1973 invest-

Table A-9. Annualized Costs, 1973-1990 (millions of 1990 dollars; capital expenditures annualized at 5 percent).

	Stationary		rec.	Mobile Source			
	K	O&M	costs	K	O&M	other	Total
1973	523	3,936	545	0	4,838	2,290	11,042
1974	1,194	4,778	746	98	5,927	2,184	13,435
1975	1,888	5,154	895	177	5,250	2,063	13,638
1976	2,630	5,768	1,074	645	4,459	2,183	14,611
1977	3,317	6,527	1,128	1,194	3,617	2,378	15,904
1978	3,968	6,991	1,158	1,784	1,705	2,487	15,776
1979	4,598	7,959	1,296	2,395	2,124	2,503	18,282
1980	5,277	8,791	1,361	3,053	2,826	2,226	20,812
1981	5,967	8,785	1,430	3,656	1,993	1,935	20,905
1982	6,610	7,855	1,158	4,313	750	1,755	20,125
1983	7,217	8,168	1,067	4,934	(201)	1,684	20,734
1984	7,694	8,505	1,082	5,564	(406)	1,634	21,909
1985	8,163	8,617	921	6,400	404	1,785	24,447
1986	8,593	8,477	1,013	6,924	(1,628)	1,809	23,161
1987	9,005	8,602	1,117	7,416	(1,474)	1,804	24,237
1988	9,410	8,143	1,206	7,831	(1,716)	1,819	24,281
1989	9,804	8,259	1,171	8,237	(1,707)	1,865	25,288
1990	10,222	8,842	1,256	8,531	(1,816)	1,542	26,066

Source: Stationary source capital costs and mobile source capital costs are from Tables A-10 and A-11, respectively. All other costs and offsets are from Table A-8.

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[21] Using an interest rate of five percent, the factor for a twenty year amortization period is 0.0802; that for a ten year amortization period is 0.1295.

Table A-10. Amortization of Capital Expenditures for Stationary Sources (millions of 1990 dollars).

	EXPEND	1973	1974	1975	1976	1977	1978	1979	1980	1981	1982	1983	1984	1985	1986	1987	1988	1989	1990
1972	6,521	523	523	523	523	523	523	523	523	523	523	523	523	523	523	523	523	523	523
1973	8,360		671	671	671	671	671	671	671	671	671	671	671	671	671	671	671	671	671
1974	8,653			694	694	694	694	694	694	694	694	694	694	694	694	694	694	694	694
1975	9,240				741	741	741	741	741	741	741	741	741	741	741	741	741	741	741
1976	8,558					687	687	687	687	687	687	687	687	687	687	687	687	687	687
1977	8,116						651	651	651	651	651	651	651	651	651	651	651	651	651
1978	7,851							630	630	630	630	630	630	630	630	630	630	630	630
1979	8,465								679	679	679	679	679	679	679	679	679	679	679
1980	8,603									690	690	690	690	690	690	690	690	690	690
1981	8,014										643	643	643	643	643	643	643	643	643
1982	7,557											606	606	606	606	606	606	606	606
1983	5,942												477	477	477	477	477	477	477
1984	5,844													469	469	469	469	469	469
1985	5,359														430	430	430	430	430
1986	5,142															413	413	413	413
1987	5,044																405	405	405
1988	4,914																	394	394
1989	5,211																		418
1990	4,395																		
SUM		523	1,194	1,888	2,630	3,317	3,968	4,598	5,277	5,967	6,610	7,217	7,694	8,163	8,593	9,005	9,410	9,804	10,222
Expenditures		8,360	8,653	9,240	8,558	8,116	7,851	8,465	8,603	8,014	7,557	5,942	5,844	5,359	5,142	5,044	4,914	5,211	4,395
K stock		6,521	14,880	23,533	32,773	41,331	49,448	57,299	65,763	74,366	82,381	89,937	95,879	101,723	107,082	112,225	117,269	122,182	127,394
K stock net depr.		6,521	14,684	22,876	31,372	38,869	45,612	51,776	58,232	64,469	69,740	74,173	76,606	78,587	79,713	80,249	80,300	79,819	79,217
Int		326	734	1,144	1,569	1,943	2,281	2,589	2,912	3,223	3,487	3,709	3,830	3,929	3,986	4,012	4,015	3,991	3,961
Depr		197	460	745	1,061	1,373	1,687	2,009	2,365	2,744	3,123	3,508	3,863	4,233	4,607	4,993	5,395	5,813	6,262

Capital expenditures for each year are found in the “EXPEND” column. Expenditures are amortized over 20 years (i.e., years (t+1) to (t+20)) using a 5% real interest rate to derive a constant cost per year for the entire amortization period. The present value (in year t) of the cost flow is equal to the expenditure in year t. Annualized CAA compliance capital cost for each year (displayed in row “SUM”) is the sum of the annuities calculated for capital expenditures from previous years. The capital stock (“K stock”) in place at the start of each year is equal to the sum of expenditures from previous years. Subtracting depreciation from the capital stock leaves “K stock net depr.” Annual interest expense is 5% of net capital stock. Annual interest expense plus depreciation equals annualized compliance cost (row “SUM”).

Table A-11. Amortization of Capital Expenditures for Mobile Sources (millions of 1990 dollars).

	EXPEND	1974	1975	1976	1977	1978	1979	1980	1981	1982	1983	1984	1985	1986	1987	1988	1989	1990
1972	0																	
1973	756	98	98	98	98	98	98	98	98	98	98							
1974	610		79	79	79	79	79	79	79	79	79	79						
1975	3,612			468	468	468	468	468	468	468	468	468	468					
1976	4,244				550	550	550	550	550	550	550	550	550	550				
1977	4,552					590	590	590	590	590	590	590	590	590	590			
1978	4,718						611	611	611	611	611	611	611	611	611	611		
1979	5,083							658	658	658	658	658	658	658	658	658	658	
1980	4,656								603	603	603	603	603	603	603	603	603	603
1981	5,070									657	657	657	657	657	657	657	657	657
1982	4,797										621	621	621	621	621	621	621	621
1983	5,622											728	728	728	728	728	728	728
1984	7,064												915	915	915	915	915	915
1985	7,659													992	992	992	992	992
1986	8,044														1,042	1,042	1,042	1,042
1987	7,755															1,004	1,004	1,004
1988	7,851																1,017	1,017
1989	7,359																	953
1990	7,312																	
SUM		98	177	645	1,194	1,784	2,395	3,053	3,656	4,313	4,934	5,564	6,400	6,924	7,416	7,831	8,237	8,531
Expenditures		610	3,612	4,244	4,552	4,718	5,083	4,656	5,070	4,797	5,622	7,064	7,659	8,044	7,755	7,851	7,359	7,312
K stock		756	1,367	4,979	9,223	13,776	18,493	23,576	28,232	33,302	38,099	42,965	49,419	53,466	57,266	60,469	63,602	65,878
K stock net depr.		756	1,306	4,807	8,647	12,437	15,993	19,480	22,057	24,574	26,287	28,289	31,204	34,023	36,845	39,026	40,997	42,169
Int		38	65	240	432	622	800	974	1,103	1,229	1,314	1,414	1,560	1,701	1,842	1,951	2,050	2,108
Depr		60	112	404	762	1,162	1,595	2,079	2,553	3,084	3,620	4,150	4,840	5,223	5,574	5,880	6,187	6,423

Capital expenditures for each year are found in the "EXPEND" column. Expenditures are amortized over 10 years (i.e., years (t+1) to (t+10)) using a 5% real interest rate to derive a constant cost per year for the entire amortization period. The present value (in year t) of the cost flow is equal to the expenditure in year t. Annualized CAA compliance capital cost for each year (displayed in row "SUM") is the sum of the annuities calculated for capital expenditures from previous years. The capital stock ("K stock") in place at the start of each year is equal to the sum of expenditures from the previous ten years. The sum of all previous expenditures less depreciation leaves "K stock net depr." Annual interest expense is 5% of net capital stock. Annual interest expense plus depreciation equals annualized compliance cost (row "SUM").

ments: that is, \$523 million plus \$671 million, or \$1,194 million (see the "SUM" row). Similar calculations are conducted for every year through 1990, to derive aggregate annualized capital costs that increase monotonically from 1973 to 1990, even though capital expenditures decline after 1975.[22]

An alternative calculation technique is available that is procedurally simpler but analytically identical to that outlined above. Instead of calculating an annuity for each capital expenditure (by multiplying the expenditure by the annuity factor *f*), then summing the annuities associated with all expenditures in previous years, one can sum all previous expenditures and multiply the sum (i..e., the capital stock at the start of the year) by *f*. The third summary row (labeled "K stock") near the bottom of the amortization summary tables give the pollution control capital stock at the start of each year. For example, the stationary sources capital stock in place at the start of 1975 was \$23,533 million (this is the sum of 1972, 1973, and 1974 capital expenditures). Multiplying the capital stock by the annuity factor 0.0802 gives \$1,888 million, which is the aggregate annualized stationary source capital cost for 1975.

One can perform further calculations to decompose the annualized capital costs into "interest" and "financial depreciation" components.[23] For example, at the start of 1973, the stationary source capital stock was \$6,521 million. A five percent interest rate implies an "interest expense" for 1973 of \$326 million. Given a 1973 annualized cost of \$523 million, this implies a "depreciation expense" for that year of (\$523 million minus \$326 million =) \$197 million. For 1974, the existing capital stock net of "financial depreciation" was \$14,684 million (that is, the \$6,521 million in place at the start of 1973, plus the investment of \$8,360 million during 1973, minus the depreciation of \$197 million during 1973); five percent of \$14,684 million is the interest expense of \$734 million. Since the annualized capital cost for 1974 is \$1,194 million, depreciation expense is \$460 million (i.e., the difference between annualized cost and the interest component of annualized cost). This procedure is repeated to determine interest and depreciation for each year through 1990 (see the last three rows of Table A-11).

The three tables above all present costs (and intermediate calculations) assuming a five percent interest rate. As noted above, the Project Team also employed rates of three percent and seven percent to calculate costs. Those calculations and intermediate results are not replicated here. The method employed, however, is identical to that employed to derive the five percent results (with the only difference being the interest rate employed in the annuity factor calculation). Table A-12 presents a summary of expenditures and annualized costs at the three interest rates.

Table A-12. Compliance Expenditures and Annualized Costs, 1973-1990 (\$1990 millions).

		Annualized Costs		
Year	**Expend.**	**at 3%**	**at 5%**	**at 7%**
1973	19,635	10,957	11,042	11,134
1974	21,405	13,231	13,435	13,655
1975	24,425	13,314	13,638	13,988
1976	24,139	14,123	14,611	15,139
1977	24,062	15,253	15,904	16,608
1978	22,593	14,963	15,776	16,653
1979	24,837	17,309	18,282	19,331
1980	25,741	19,666	20,812	22,046
1981	24,367	19,590	20,905	22,321
1982	21,555	18,643	20,125	21,720
1983	20,148	19,095	20,734	22,498
1984	21,560	20,133	21,909	23,819
1985	22,903	22,516	24,447	26,523
1986	20,831	21,109	23,161	25,364
1987	20,615	22,072	24,237	26,562
1988	19,805	22,012	24,281	26,719
1989	19,817	22,916	25,288	27,836
1990	19,019	23,598	26,066	28,717

Discounting Costs and Expenditures

The stream of costs from 1973 to 1990 can be expressed as a single cost number by *discounting* all costs to a common year. In this analysis, all costs and benefits are discounted to 1990 (in addition, all costs and benefits are converted to 1990 dollars, removing the effects of price inflation).[24] There is a broad range of opinion in the economics profession regarding the appropriate discount rate to use in analyses such as this. Some economists believe that the appropriate rate is one that approximates the social rate of time preference — three percent, for example (all rates used here are "real", i.e., net of price inflation impacts). Others believe that a rate that approximates the opportunity cost of capital (e.g., seven percent or greater) should be used. A third school of thought holds that some combination of the social rate of time preference and the opportunity cost of capital is appropriate, with the combination effected either by use of an intermediate rate or by use of a multiple-step procedure which uses the social rate of time preference as the "discount rate," but still accounts for the cost of capital. The section 812 Project Team chose to use a range of discount rates (three, five, and seven percent) for the analysis.

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[22] Similar calculations were performed for mobile source control capital costs, where the assumed amortization period is ten years.

[23] One might, for example, wish to examine the relative importance of the "time value" component of the computed capital costs.

[24] Unlike most cost-benefit analyses, where future expected costs and benefits are discounted back to the present, this exercise brings past costs closer to the present. That is, the discounting procedure used here is actually compounding past costs and benefits.

Expenditures and annualized costs discounted to 1990 are found on Table A-13. Expenditures are discounted at all three rates; annualized costs are discounted at the rate corresponding to that used in the annualization procedure (i.e., the "annualized at 3%" cost stream is discounted to 1990 at three percent). The final row presents the result of an explicit combination of two rates: Capital costs are annualized at seven percent, then the entire cost stream is discounted to 1990 at three percent.

Table A-13. Costs Discounted to 1990 ($1990 millions).

	3%	5%	7%
Expenditures	520,475	627,621	760,751
Annualized Costs	416,804	522,906	657,003
Annualized at 7%	476,329		

Indirect Economic Effects of the CAA

In addition to imposing direct compliance costs on the economy, the CAA induced indirect economic effects, primarily by changing the size and composition of consumption and investment flows. Although this analysis does not add these indirect effects to the direct costs and include them in the comparison to benefits, they are important to note. This section summarizes the most important indirect economic effects of the CAA, as estimated by the J/W macroeconomic simulation.

GNP and Personal Consumption

Under the no-control scenario, the level of GNP increases by one percent in 1990 relative to the control case (see Table A-14). During the period 1973-1990, the percent change in real GNP rises monotonically from 0.26 percent to 1.0 percent. The increase in the level of GNP is attributable to a rapid accumulation of capital, which is driven by changes in the price of investment goods. The capital accumulation effect is augmented by a decline in energy prices relative to the base case. Lower energy prices that correspond to a world with no CAA regulations decreases costs and increases real household income, thus increasing consumption.

Table A-14. Differences in Gross National Product Between the Control and No-control Scenarios.

Year	Nominal % Change	Real % Change
1973	-0.09	0.26
1974	-0.18	0.27
1975	-0.10	0.44
1976	-0.00	0.49
1977	-0.10	0.54
1978	-0.16	0.56
1979	-0.16	0.63
1980	-0.14	0.69
1981	-0.14	0.73
1982	-0.19	0.74
1983	-0.19	0.78
1984	-0.17	0.84
1985	-0.12	0.95
1986	-0.14	0.98
1987	-0.15	1.01
1988	-0.20	1.00
1989	-0.21	0.99
1990	-0.18	1.00

Removing the pollution control component of new capital is equivalent to lowering the marginal price of investment goods. Combining this with the windfall gain of not having to bring existing capital into compliance leads to an initial surge in the economy's rate of return, raising the level of real investment. The investment effects are summarized in Figure A-1. More rapid (ordinary) capital accumulation leads to a decline in the rental price of capital services which, in turn, stimulates the demand for capital services by producers *and* consumers. The capital rental price reductions also serve to lower the prices of goods and services and, so, the overall price level. Obviously, the more capital intensive sectors exhibit larger price reductions.[25] The price effects from investment changes are compounded by the cost reductions associated with releasing resources from the operation and maintenance of pollution control equipment and by the elimination of higher prices due to regulations on mobile sources.

To households, no-control scenario conditions are manifest as an increase in permanent future real earnings which supports an increase in real consumption in all periods and, generally, an increase in the demand for leisure (see Table A-15). Households marginally reduce their offer of labor services as the income effects of higher real earnings dominate the substitution effects of lower goods prices. The increase in consumption is dampened by an increase in the rate of return that produces greater investment (and personal savings).

Table A-15. Difference in Personal Consumption Between the Control and No-Control Scenarios.

Year	Nominal % Change	Real % Change
1973	-0.02	0.33
1974	-0.01	0.43
1975	-0.10	0.24
1976	-0.10	0.39
1977	-0.10	0.54
1978	-0.09	0.63
1979	-0.11	0.68
1980	-0.12	0.71
1981	-0.13	0.74
1982	-0.12	0.81
1983	-0.13	0.85
1984	-0.15	0.86
1985	-0.19	0.88
1986	-0.19	0.94
1987	-0.19	0.98
1988	-0.17	1.03
1989	-0.17	1.04
1990	-0.18	1.01

Finally, technical change is a very important aspect of the supply-side adjustments under the no-control scenario. Lower factor prices increase the endogenous rates of technical change in those industries that are factor-using. Lower rental prices for capital benefit the capital-using sectors, lower materials prices benefit the materials-using sectors, and lower energy prices benefit the energy-using sectors. On balance, a significant portion of the increase in economic growth is attributable to accelerated productivity growth. Under the no-control scenario, economic growth averages 0.05 percentage points higher over the interval 1973-1990. The increased availability of capital accounts for 60 percent of this increase while faster productivity growth accounts for the remaining 40 percent. Thus, the principal effect arising from the costs associated with CAA initiatives is to slow the economy's rates of capital accumulation and productivity growth. This finding is consistent with recent analyses suggesting a potential association between higher reported air, water, and solid waste pollution abatement costs and lower plant-level productivity in some manufacturing industries (Gray and Shadbegian, 1993 and 1995).

As with the cost and expenditure data presented above, it is possible to present the stream of GNP and consumption changes as single values by discounting the streams to a single year. Table A-16 summarizes the results of the discounting procedure, and also includes discounted expenditure and annualized cost data for reference. Accumulated (and discounted to 1990) losses to GNP over the 1973-1990 period were half again as large as expenditures during the same period, and approximately twice as large as annualized costs. Losses in household consumption were approximately as great as annualized costs.

Table A-16. GNP and Consumption Impacts Discounted to 1990 ($1990 billions).

	3%	5%	7%
Expenditures	520	628	761
Annualized Costs	417	523	657
GNP	880	1005	1151
Household Consumption	500	569	653
HH and Gov't Consumption	676	769	881

Source: Expenditures and annualized costs from above; macroeconomic impacts from Jorgenson et al. (1993), Table 4.1

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[25] Not surprisingly, at the industry level, the principal beneficiaries in the long run of eliminating the costs associated with air pollution abatement are the most heavily regulated industries. The largest changes in industry prices and outputs occur in the motor vehicles industry. Other industries that benefit significantly from the elimination of environmental controls are refined petroleum products, electric utilities, and other transportation equipment. Turning to manufacturing industries, metal mining and the primary metals have the largest gains in output from elimination of air pollution controls.

Figure A-1. Percent Difference in Real Investment Between Control and No-control Scenarios.

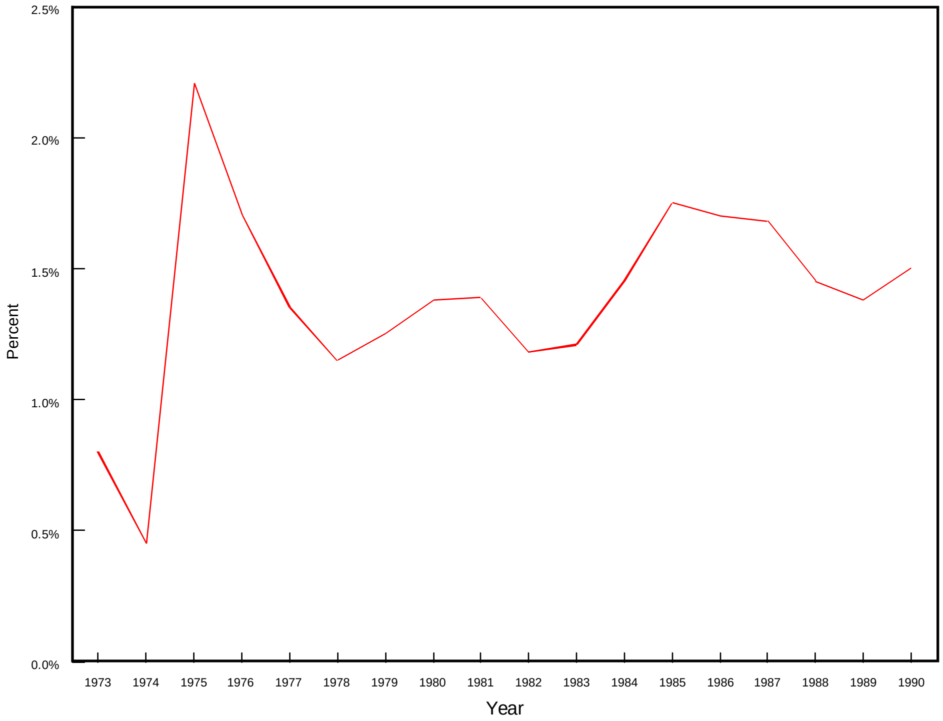
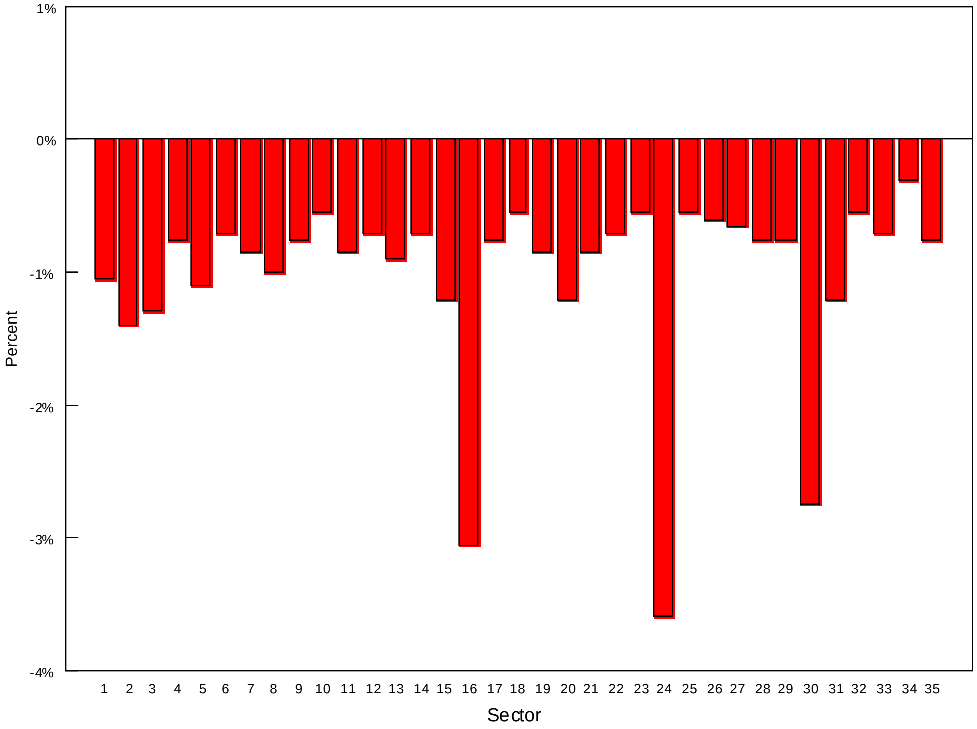


Figure A-2. Percent Difference in Price of Output by Sector Between Control and No-control Scenario for 1990.



Although they have value as descriptors of the magnitude of changes in economic activity, neither GNP nor consumption changes are perfect measures of changes in social welfare. A better measure is Equivalent Variations (EVs), which measure the change in income that is equivalent to the change in (lifetime) welfare due to removal of the CAA. As part of its macroeconomic exercise, EPA measured the EVs associated with removal of the CAA. Elimination of CAA compliance costs (disregarding benefits) represents a welfare gain of $493 billion to $621 billion, depending on assumptions used in the analysis.[26] This result does not differ greatly from the range of results represented by expenditures, anualized costs, and consumption changes.

Prices

One principal consequence of the Clean Air Act is that it changes prices. The largest price reductions accrue to the most heavily regulated industries which are the large energy producers and consumers (see Table A-17). But these are also the most capital intensive sectors and it is the investment effects that are the dominant influences in altering the course of the economy. Focusing on energy prices, under the no-control scenario the price of coal in 1990 declines by 1.3 percent, refined petroleum declines by 3.03 percent, electricity from electric utilities declines by 2.75 percent, and the price of natural gas from gas utilities declines by 1.2 percent. The declining price of fossil fuels induces substitution toward fossil fuel energy sources and toward energy in general. Total Btu consumption also increases.

Table A-17. Percentage Difference in Energy Prices Between the Control and No-control Scenarios.

Year	Coal	Refined Petroleum	Electric Utilities	Gas Utilities
1973	-0.44	-5.99	-2.11	-0.32
1974	-0.47	-4.84	-2.53	-0.44
1975	-0.42	-4.28	-2.19	-0.31
1976	-0.57	-3.83	-2.12	-0.44
1977	-0.74	-3.43	-2.22	-0.59
1978	-0.86	-3.28	-2.39	-0.68
1979	-0.91	-2.92	-2.81	-0.71
1980	-0.94	-2.76	-2.97	-0.69
1981	-0.97	-2.50	-2.76	-0.71
1982	-0.98	-2.42	-2.63	-0.77
1983	-1.09	-2.35	-2.58	-0.85
1984	-1.12	-2.26	-2.49	-0.91
1985	-1.21	-2.89	-2.62	-0.97
1986	-1.27	-3.35	-2.69	-1.12
1987	-1.31	-3.50	-2.78	-1.18
1988	-1.30	-3.61	-2.75	-1.19
1989	-1.31	-3.45	-2.74	-1.19
1990	-1.30	-3.03	-2.75	-1.20

Sectoral Effects: Changes in Prices and Output by Industry

At the commodity level, the effect of the CAA varies considerably. Figure A-2 shows the changes in the supply price of the 35 commodities measured as changes between the no-control case and the control-case for 1990.

In 1990, the largest change occurs in the price of motor vehicles (commodity 24), which declines by 3.8 percent in the no-control case. Other prices showing significant effects are those for refined petroleum products (commodity 16) which declines by 3.0 percent, and electricity (commodity 30) which declines 2.7 percent. Eight of the remaining industries have decreases in prices of 1.0 to 1.4 percent under the no-control scenario. The rest are largely unaffected by environmental regulations, exhibiting price decreases between 0.3 and 0.8 percent.

To assess the intertemporal consequences of the CAA, consider the model's dynamic results and the adjustment of prices between 1975 and 1990. Initially, in 1975, the biggest effect is on the price of output from petroleum refining (sector 16), which declines by 4.3 percent. But by 1990, the price of petroleum refining is about 3.0 percent below control scenario levels. In contrast, the price of motor vehicles (sector 24) is about 2.4 percent below baseline levels in 1975, but falls to about 3.8 percent below baseline levels in 1990.

The price changes affect commodity demands, which in turn determine how industry outputs are affected. Figure A-3 shows percentage changes in quantities produced by the 35 industries for 1990. As noted earlier, the principal beneficiaries under the no-control scenario are the most heavily regulated industries: motor vehicles, petroleum refining, and electric utilities.

In 1990, the motor vehicle sector (sector 24) shows the largest change in output, partly due to the fact that the demand for motor vehicles is price elastic. Recall

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[26] Jorgenson et al., 1993.

Figure A-3. Percent Difference in Quantity of Output by Sector Between Control and No-control Scenario for 1990.

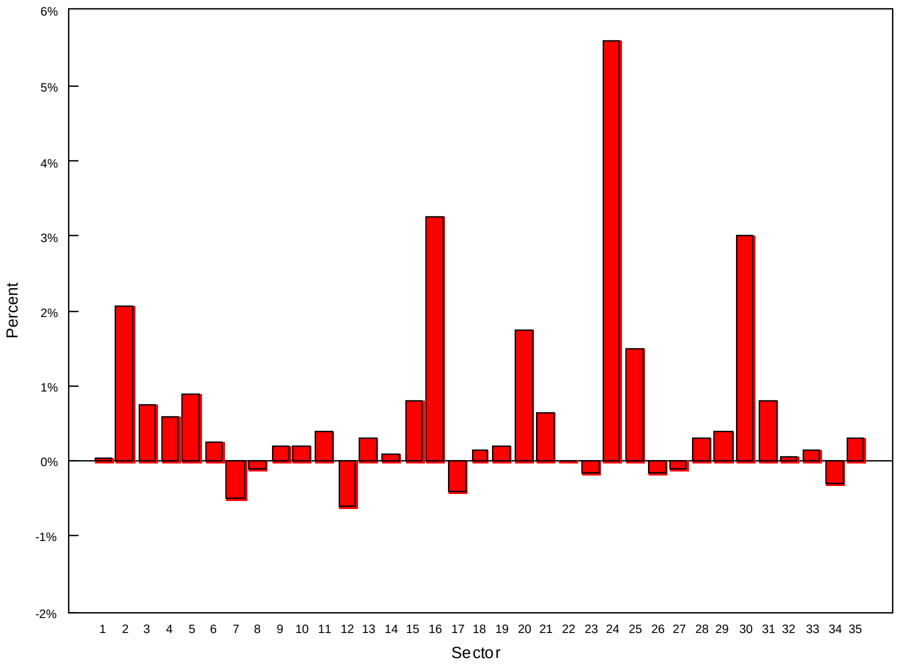
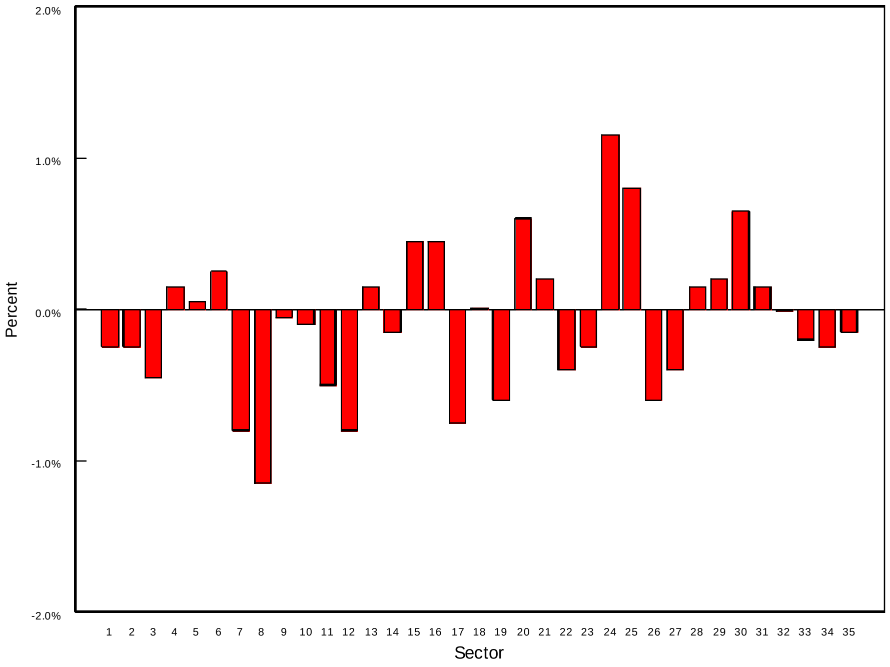


Figure A-4. Percent Difference in Employment by Sector Between Control and No-control Scenario for 1990.



that the largest increase in prices also occurred in the motor vehicles sector. The 3.8 percent reduction in prices produces an increase in output of 5.3 percent relative to the base case.

Significant output effects are also seen in the petroleum refining sector (sector 16) with a 3.2 percent increase, in electricity (sector 30) with a 3.0 percent increase, and in other transportation equipment (sector 25) with a 1.6 percent increase. The large gains in output for these industries are mostly due to the decline in their prices. In manufacturing, the sectors exhibiting the most significant output effects are metal mining (sector 2) with a 2.0 percent increase, and primary metals (sector 20) with a 1.8 percent increase. Twenty of the remaining industries exhibit increase in output of less than 0.9 percent after pollution controls are removed.

While most sectors increase output under the no-control scenario, a few sectors decline in size in the absence of air pollution controls. The most notable of these are food and kindred products (sector 7) which decline by 0.5 percent, furniture and fixtures (sector 12) which decline by 0.6 percent, and rubber and plastic products (sector 17) which decline by 0.3 percent. These sectors are among the least capital intensive, so the fall in the rental price of capital services has little effect on the prices of outputs. Buyers of the commodities produced by these industries face higher relative prices and substitute other commodities in both intermediate and final demand. The rest of the sectors are largely unaffected by environmental regulations.

Changes in Employment Across Industries

The effect of the CAA on employment presents a much more complicated picture. Although Jorgenson-Wilcoxen is a full-employment model and cannot be used to simulate unemployment effects, it is useful for gaining insights about changes in the patterns of employment across industries. Percentage changes in employment by sector for 1990 are presented in Figure A-4.

For 1990, the most significant changes in the level of employment relative to the control scenario occur in motor vehicles (sector 24) which increases 1.2 percent, other transportation equipment (sector 25) which increases 0.8 percent, electric utilities (sector 30) which increases 0.7 percent, and primary metals (sector 20) which increases 0.6 percent. The level of employment is higher relative to the control case in 10 other industries.

For a few sectors, the no-control scenario results in changes in real wages which cause *reductions* in employment. The most notable reductions in employment under the no-control scenario occur in tobacco manufacturing (sector 8) which declines 1.2 percent, furniture and fixtures (sector 12) which declines 0.8 percent, rubber and plastic products (sector 17) which declines 0.8 percent, food and kindred products (sector 7) which declines 0.7 percent, stone, clay and glass products (sector 19) which declines 0.6 percent, and instruments (sector 26) which declines 0.6 percent. These sectors are generally those in which the level of output was lower in 1990 relative to the control scenario, since they are among the least capital intensive and the fall in the rental price of capital services has little effect on the prices of outputs. Buyers of the commodities produced by these industries face higher relative prices and substitute other commodities in both intermediate and final demand. It is interesting to note that several of the least capital intensive sectors experience insignificant employment effects in the short run (1975) under the no-control scenario, but increasingly adverse effects over the 20-year period of analysis. These include food and kindred products, furniture and fixtures, rubber and plastic products, stone, clay and glass products, and instruments.

Examination of the transition of employment in the economy from the initial equilibrium to 1990 reveals that the employment effects of the CAA on motor vehicles, transportation equipment, electric utilities, and primary metals persist over the entire period of analysis. Employment varies from: an increase of 1.7 percent in 1975 to 1.2 percent in 1990 in motor vehicles; an increase of 0.7 in 1975 to 0.8 percent in 1990 in transportation equipment; an increase of 1.2 percent in 1975 to 0.7 percent in 1990 in electric utilities; and an increase of 0.8 percent in 1975 to 0.6 percent in 1990.

Uncertainties in the Cost Analysis

Potential Sources of Error in the Cost Data

Because of the importance of the *Cost of Clean* data for this assessment, the project team investigated potential sources of error due to the use of industry's self-reported costs of compliance with air pollution abatement requirements. Concerns about the accuracy of responses include (1) misreporting by firms in response to federal agency surveys, and (2) omission of important categories of compliance cost from the data collected or reported by these federal agencies.[27] Table A-18 contains a summary of the results of the analysis. This analysis is consistent with the findings of two recent studies comparing combined air, water, and solid waste pollution abatement costs, as reported in federal abatement cost surveys, to their observed effects on productivity levels. These studies suggest that, since observed productivity decreases exceed those expected to result from the reported abatement costs, there may be additional pollution abatement costs not captured or reported in the survey data, and that total abatement costs for the three manufacturing industries studied may be under-reported by as much as a factor of two in the most extreme case (Gray and Shadbegian, 1993 and 1995; Gray, 1996).

The major finding from this analysis indicates that total O&M costs are likely to be under-reported due to exclusion of private research and development

Table A-18. Potential Sources of Error and Their Effect on Total Costs of Compliance.

Source of Error	Effect on Capital Costs	Effect on O&M Costs
Lack of Data at Firm Level	Under-reported Percent Unknown	Under-reported Percent Unknown
Misallocation of Costs:		
Inclusion of OSHA and Other Regulatory Costs	Over-reported Percent Unknown	Over-reported Percent Unknown
Exclusion of Solid Waste Disposal Costs Related to Air Pollution Abatement	—	Under-reported Percent Unknown
Exclusion of Costs:		
Exclusion of Private R&D Expenses	—	Under-reported by 14 to 17% (varies by year)
Exclusion of Energy Use by Pollution Abatement Devices[(a)]	—	Under-reported by 1 to 3% (varies by year)
Exclusion of Depreciation Expenses[(a)]	—	Under-reported by 1 to 2% (varies by year)
Exclusion of Recovered Costs	—	Over-reported by 1% Plus
Omission of Small Firms	Under-reported by 1 to 2%	Under-reported by 1 to 2%
NET EFFECT	Under-reported	Under-reported

[(a)] Energy outlays *are* part of the data on O&M costs and depreciation expenses *are not*. Accordingly, in the J/W model, energy outlays are considered along with other operating expenditures in terms of their impacts on unit costs. Depreciation is represented fully in the capital accumulation process, as the undepreciated capital stock at the beginning of any period gives rise to the flow of capital services available to producers and consumers.

Source: Industrial Economics, Incorporated, memorandum to Jim DeMocker, EPA/OAR, "Sources of Error in Reported Costs of Compliance with Air Pollution Abatement Requirements," October 16, 1991.

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[27] Memorandum from Industrial Economics, Incorporated to Jim DeMocker (EPA/OAR) dated 10/16/91 and entitled "Sources of Error in Reported Costs of Compliance with Air Pollution Abatement Requirements."

(R&D) expenditures. Note, however, that although these costs were excluded from those used for the macroeconomic modeling, they were included in the overall direct cost estimate of the CAA; see "Other Direct Costs," above. These costs are excluded from the macromodeling because they cannot be disaggregated by industry and, more importantly, because there is no information on what was purchased or obtained as a result of these expenditures.

Based on the need indicated by the IEc review, modifications to the BEA data were made to remedy some of the biases noted above. In particular, recovered costs for stationary source air pollution, e.g. sulfur removed using scrubbers that is then sold in the chemical market, have been accounted for in the data set used in the model runs.

Table A-19. Stationary Source O&M Expenditures as a Percentage of Capital Stock (millions of 1990 dollars).

				O&M divided by	
	K stock	Net K	O&M	K stock	Net K
1973	6,521	6,521	3,936	0.60	0.60
1974	14,880	14,684	4,778	0.32	0.33
1975	23,533	22,876	5,154	0.22	0.23
1976	32,773	31,372	5,768	0.18	0.18
1977	41,331	38,869	6,527	0.16	0.17
1978	49,448	45,612	6,991	0.14	0.15
1979	57,299	51,776	7,959	0.14	0.15
1980	65,763	58,232	8,791	0.13	0.15
1981	74,366	64,469	8,785	0.12	0.14
1982	82,381	69,740	7,855	0.10	0.11
1983	89,937	74,173	8,168	0.09	0.11
1984	95,879	76,606	8,505	0.09	0.11
1985	101,723	78,587	8,617	0.08	0.11
1986	107,082	79,713	8,477	0.08	0.11
1987	112,225	80,249	8,602	0.08	0.11
1988	117,269	80,300	8,143	0.07	0.10
1989	122,182	79,819	8,259	0.07	0.10
1990	127,394	79,217	8,842	0.07	0.11

"K stock" is the accumulated undepreciated stationary source control capital stock available at the beginning of each year, from Table A-10.
"Net K" is the stationary source control capital stock less depreciation implied by amortization at 5%; from Table A-10.
"O&M" is the stationary source control O&M expenditures; from Table A-9.
The final two columns are ratios: O&M divided by capital stock; and O&M divided by net capital.

An additional set of concerns relates directly to reporting of costs by firms. Some have noted an unexpected temporal pattern of stationary source control expenditures in the BEA data that might lead one to question the accuracy of the Census survey responses. One would expect that stationary source O&M expenditures over time would be roughly proportional to the accumulated stationary source control capital stock. Yet, as illustrated in Table A-19, O&M expenditures as a fraction of accumulated capital stock decline over time (even if one discounts the first few years because of the dramatic percentage increases in capital stock during those years). It is true that the ratio of O&M expenditures to the *depreciated* capital stock (in the far right column, labeled "net K") is reasonably stable after 1981. The depreciation shown here, however, is a *financial* depreciation only, depicting the declining value of a piece of equipment over time, rather than a measure of physical asset shrinkage. Assuming a twenty-year useful lifetime, *all* of the stationary source control capital stock put in place since 1972 could conceivably still be in place in 1990. If anything, one would expect the O&M/K ratio to *increase* as the capital depreciates (i.e., ages), until the equipment is scrapped, because aging equipment requires increasing maintenance. Consequently, one might infer from this information that firms have systematically under-reported O&M expenditures, or have over-reported capital expenditures.

The apparent anomaly might be explained by an examination of the types of O&M expenditures reported. If more than a token percentage of O&M expenditures are unrelated to "operation and maintenance" of pollution control devices, then the observed O&M/K ratio would not appear unusual.

The Census PACE survey[28] required respondents to report air pollution abatement O&M expenses in the following categories: salaries and wages; fuel and electricity; contract work; and materials, leasing, and "miscellaneous."[29] In later versions of the survey, additional information relating to the types of expenses to report was provided as a guide to respondents. The types of expenses listed that are relevant to air pollution abatement include:

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[28] *Pollution Abatement Costs and Expenditures*, various years.

[29] Census also requested a reporting of "depreciation" expenses as a component of O&M. BEA, however, removed depreciation expense from the reported O&M costs because retaining depreciation would have amounted to double-counting, since BEA also reported capital expenditures.

(1) operating and maintaining pollution abatement equipment;
(2) fuel and power costs for operating pollution abatement equipment;
(3) parts for pollution abatement equipment replacement and repair;
(4) testing and monitoring of emissions;
(5) incremental costs for consumption of environmentally preferable materials and fuels;
(6) conducting environmental studies for development or expansion;
(7) leasing of pollution abatement equipment;
(8) compliance and environmental auditing;
(9) salaries and wages for time spent completing environmental reporting requirements; and
(10) developing pollution abatement operating procedures.[30]

The magnitude of the expenditures associated with the first three items should be correlated with the size of the existing stock of air pollution abatement capital. Expenditures associated with items four through ten, however, should be independent of the size of the existing capital stock (expenditures associated with item seven, leasing of pollution abatement equipment, could be negatively correlated with the size of the capital stock). *If* items four through ten account for a non-negligible proportion of total O&M expenditures, and *if* respondents included these cost categories even though they were not explicitly listed in the survey instructions before 1991, *then* one would expect to see the O&M/K ratio declining during the study period. Thus, even though it is possible that O&M expenditures are underreported (or that capital expenditures are overreported), one cannot be certain.

Mobile Source Costs

For the section 812 analysis, EPA used the best available information on the estimated cost of mobile source air pollution control. Several other sources of cost estimates exist, however, including a cost series produced by the Department of Commerce Bureau of Economic Analysis (BEA). The BEA cost series is summarized in Table A-20. The BEA estimates differ significantly from EPA estimates, particularly with respect to estimates of capital costs and the "fuel price penalty" associated with the use of unleaded gasoline.

EPA's capital cost estimates are based on estimates of the cost of equipment required by mobile source regulations. BEA's estimates are based on survey data from the Bureau of Labor Statistics (BLS) that measures the increase in the per-automobile cost (relative to the previous model year) due to pollution control and fuel economy changes for that model year. The difference in approach is significant: BEA's annual capital cost estimates exceed EPA's by a factor of (roughly) two. EPA may underestimate costs to the extent that engineering cost estimates of components exclude design and development costs for those components. The BLS estimates add the incremental annual costs to all past costs to derive total current-year costs. Such an approach overestimates costs to the extent that it fails to account for cost savings due to changes in component mixes over time.

Table A-20. Comparison of EPA and BEA Stationary Source Expenditure Estimates (millions of current dollars).

	Private sector		Gov't. Enterprise		Total
Year	capital	O&M	capital	O&M	Expend.
	EPA Estimates				
1986	4,090	7,116	312	140	11,658
1987	4,179	7,469	277	130	12,055
1988	4,267	7,313	243	161	11,984
1989	4,760	7,743	235	173	12,911
1990	4,169	8,688	226	154	13,237
	BEA Estimates				
1986	4,090	7,072	312	182	11,656
1987	3,482	5,843	246	141	9,712
1988	3,120	6,230	121	161	9,632
1989	3,266	6,292	229	152	9,939
1990	4,102	6,799	200	154	11,255

"Recovered Costs" are not included in this table.
Sources for "BEA Estimates": for 1986, "Pollution Abatement and Control Expenditures," Survey of Current Business (BEA) June 1989, Table 7; for 1987-90, BEA May 1995, Table 8.

Some mobile source pollution control devices required the use of unleaded fuel. Unleaded gasoline is more costly to produce than is leaded gasoline, and generally has a greater retail price, thus imposing a cost on consumers. EPA estimated the "fuel price penalty" by using a petroleum refinery cost model to determine the expected difference in production cost between leaded and unleaded gasoline. BEA's "fuel price penalty" was the difference between the retail price of unleaded gasoline and that of leaded gasoline.

A detailed description of the data sources, analytic methods, and assumptions that underlie the EPA and BEA mobile source cost estimates can be found in McConnell et al. (1995).

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[30] *Pollution Abatement Costs and Expenditures, 1992*, pg. A-9.

Stationary Source Cost Estimate Revisions

As noted above, the costs used for stationary sources in the macro-modeling (and retained in this cost analysis) were projected for several years in the late 1980s. Since that time, BEA has released historical expenditure estimates for those years based on survey data. A comparison of the expenditure series can be found in Table A-21. Apparently, EPA's projections overestimated stationary source compliance expenditures by approximately $2 billion per year for the period 1987-1990. Since expenditures from all sources are estimated to be $18 billion -$19 billion (current dollars) per year during 1987-1990, this implies that EPA has overestimated compliance expenditures by more than ten percent during this period. Although a substantial overstatement for those years, the $2 billion per year overestimate would have little impact (probably less than two percent) on the discounted present value, in 1990 dollars, of the 1973-1990 expenditure stream.

Table A-21. BEA Estimates of Mobile Source Costs.

Year	Capital Exp.	Net I&M*	Fuel Price Penalty	Fuel Economy Penalty
1973	1,013	1,104		697
1974	1,118	1,380	5	1,180
1975	2,131	1,520	97	1,344
1976	2,802	1,420	309	1,363
1977	3,371	1,289	701	1,408
1978	3,935	1,136	1,209	1,397
1979	4,634	931	1,636	1,792
1980	5,563	726	2,217	2,320
1981	7,529	552	2,996	2,252
1982	7,663	409	3,518	1,876
1983	9,526	274	4,235	1,582
1984	11,900	118	4,427	1,370
1985	13,210	165	4,995	1,133
1986	14,368	(331)	4,522	895
1987	13,725	(453)	3,672	658
1988	16,157	(631)	3,736	420
1989	15,340	(271)	1,972	183
1990	14,521	(719)	1,370	(55)

* Inspection and maintenance costs less fuel density savings and maintenance savings.

Endogenous Productivity Growth in the Macro Model

For each industry in the simulation, the JW model separates price-induced changes in factor use from changes resulting strictly from technical change. Thus, simulated productivity growth for each industry has two components: (a) an exogenous component that varies over time, and (b) an endogenous component that varies with policy changes. Some reviewers have noted that, although not incorrect, use of endogenous productivity growth is uncommon in the economic growth literature. EPA conducted a sensitivity run of the J/W model, setting endogenous growth parameters to zero (i.e., removing endogenous productivity growth from the model).[31]

Endogenous productivity growth is an important factor in the J/W model. For example, for the period 1973-1990, removal of the endogenous productivity growth assumptions reduces household income by 2.9 to 3.0 percent (depending on whether one uses a world with CAA or one without CAA as the baseline). In comparison, removal of CAA compliance costs results in a 0.6 to 0.7 percent change in household income (depending on whether one uses, as a baseline, a world with or one without endgenous productivity growth). That is, use of the endogenous productivity growth assumption has four to five times the impact of that of CAA compliance costs.

Although very important to the simulated growth of the economy within any policy setting, the endogenous productivity growth assumption is less important across policy settings. Under the base (i.e., "with endogenous productivity growth") scenario, the aggregate welfare effect (measured as EVs, see above) of CAA compliance costs and indirect effects is estimated to be 493 billion to 621 billion in 1990 dollars. If one removes the endogenous productivity growth assumption, the aggregate welfare effect declines to the range 391 billion to 494 billion in 1990 dollars (Jorgenson et al., 1993, pg. 6-15), a reduction of about twenty percent.

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[31] For greater detail, see Jorgenson et al., 1993.

Amortization Period for Stationary Source Plant and Equipment

In developing annualized costs, stationary source capital expenditues were amortized over a twenty-year period. That is, it was assumed that plant and equipment would depreciate over twenty years. It is possible that stationary source plant and equipment has, on average, a useful lifetime significantly greater than twenty years. The Project Team tested the sensitivity of the cost analysis results to changes in stationary source capital amortization periods.

Table A-22 presents total annualized compliance costs assuming a 40-year amortization period for stationary source capital expenditures (all other cost components are unchanged from the base analysis). All costs are in 1990-value dollars, ad three alternative discount rates are used in the annualization period. Table A-23 presents the results discounted to 1990, and compared to the base case results (i.e., using a twenty-year amortization period). Doubling the amortization period to 40 years decreases the 1990 present value of the 1973-1990 cost stream by approximately 40 billion dollars. This represents a change of six percent to nine percent, depending on the discount rate employed.

Table A-22. Annualized Costs Assuming 40-Year Stationary Source Capital Amortization Period, 1973-1990 (millions of 1990 dollars).

	Annualized Costs		
Year	**at 3%**	**at 5%**	**at 7%**
1973	10,801	10,899	11,008
1974	12,875	13,108	13,366
1975	12,751	13,121	13,532
1976	13,338	13,891	14,504
1977	14,263	14,996	15,807
1978	13,778	14,690	15,695
1979	15,936	17,024	18,220
1980	18,091	19,368	20,771
1981	17,809	19,272	20,880
1982	16,670	18,316	20,123
1983	16,941	18,759	20,754
1984	17,836	19,803	21,960
1985	20,079	22,213	24,551
1986	18,544	20,809	23,288
1987	19,384	21,772	24,387
1988	19,203	21,706	24,446
1989	19,989	22,604	25,467
1990	20,546	23,268	26,247

Table A-23. Effect of Amortization Periods on Annualized Costs Discounted to 1990 (billions of 1990 dollars).

	Discount rate		
	3%	5%	7%
20-yr amortization period	417	523	657
40-yr amortization period	379	483	617

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Appendix B: Emissions Modeling

Introduction

This appendix provides additional details of the methodologies used to estimate control and no-control scenario emissions and the results obtained by these methods. Methodological information and results are provided for each of the six principal emission sectors: industrial combustion, industrial processes, electric utilities, on-highway vehicles, off-highway vehicles, and commercial/residential sources.

The initial section of this appendix assesses the emissions projections presented in this analysis by (1) comparing the 1970 to 1990 control scenario projections with recent EPA *Trends* report estimates for the same years and (2) comparing the 1970 to 1990 trend in no-control scenario projections with 1950 to 1970 emissions as reported in *Trends*. The first comparison indicates that control scenario emissions projections approximate, but do not precisely match, the EPA *Trends* data. The reason for this mismatch is discussed below. The second comparison is useful for demonstrating that pre-1970 emissions trends would not provide a satisfactory basis for extrapolating emissions trends into the 1970 to 1990 period. The inability to simply extrapolate pre-1970 trends provides further justification for applying the present modeling methodologies to generate no-control scenario emissions projections.

The remainder of the appendix provides further details of the emissions modeling conducted in support of the present analysis, and is largely adapted from the draft report “The Impact of the Clean Air Act on 1970 to 1990 Emissions; section 812 retrospective analysis,” March 1, 1995 by Pechan Associates. The draft Pechan report surveys the methodologies and results associated with the sector-specific emission modeling efforts by Argonne National Laboratory (ANL), ICF Resources Incorporated (ICF), Abt Associates (Abt), and the Environmental Law Institute (ELI).

Comparison of Emissions Projections with Other EPA Data

Control Scenario Projections Versus EPA Trends Projections

The control scenario emission results are similar, but not identical, to official EPA historical emission estimates provided by the EPA National Air Pollutant Emission Trends Reports.[1] Comparisons between the current estimates and the *Trends* data for SO_2, NO_x, VOC, CO, and TSP are presented in Figures B-1, B-2, B-3, B-4, and B-5 respectively. More detailed tables providing emission estimates by sector and by target year for TSP, SO_2, NO_x, VOC, CO, and Lead are presented in Tables B-16, B-17, B-18, B-19, B-20, and B-21, respectively, at the end of this appendix.

Though the EPA *Trends* and the present study emission profiles are similar to each other, they should not be expected to match precisely. This is because the emission estimates developed for the present study are based on modeled macroeconomic and emission sector conditions. Even though the macroeconomic and sector models themselves are constructed and calibrated using historical data, modeled replications of historical trends would not be expected to precisely capture actual historical events and conditions which affect emissions. Relying on modeled historical scenarios is considered reasonable for the present analysis since its purpose is to estimate the differences between conditions with and without the CAA. Comparing actual historical emissions with modeled no-control emissions would lead to an inconsistent basis for comparisons between scenarios. Using models for both scenarios allows potential model biases to essentially cancel out.

In general, however, these comparisons show close correspondence between control scenario and *Trends* estimates with the largest differences occur-

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[1] EPA/OAQPS, “National Air Pollutant Emission Trends 1900 - 1994,” EPA-454/R-95-011, October 1995.

Figure B-1. Comparison of Control, No-control, and *Trends* SO_2 Emission Estimates.

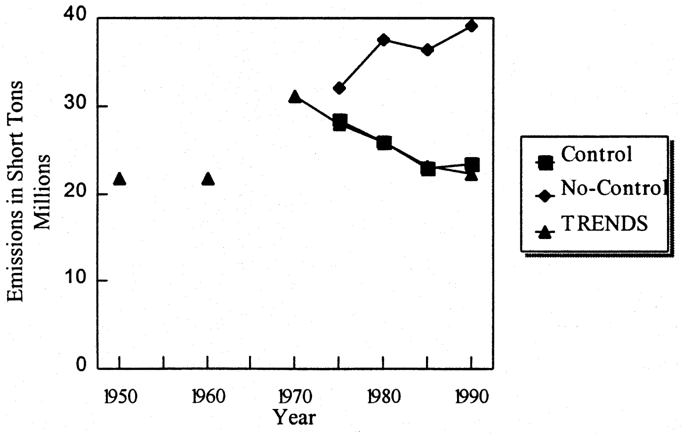


Figure B-2. Comparison of Control, No-control, and *Trends* NO_X Emission Estimates.

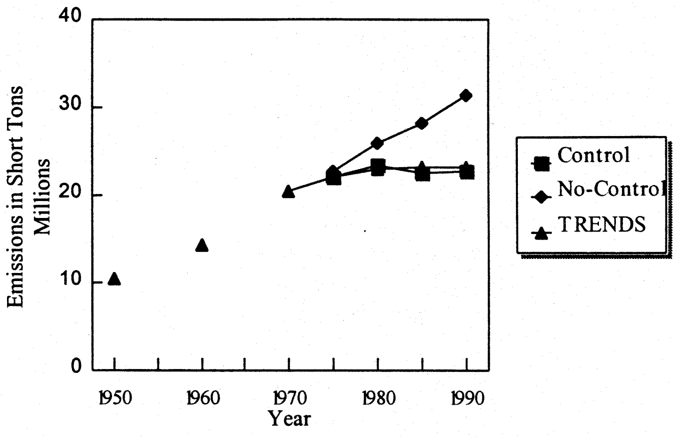


Figure B-3. Comparison of Control, No-control, and *Trends* VOC Emission Estimates.

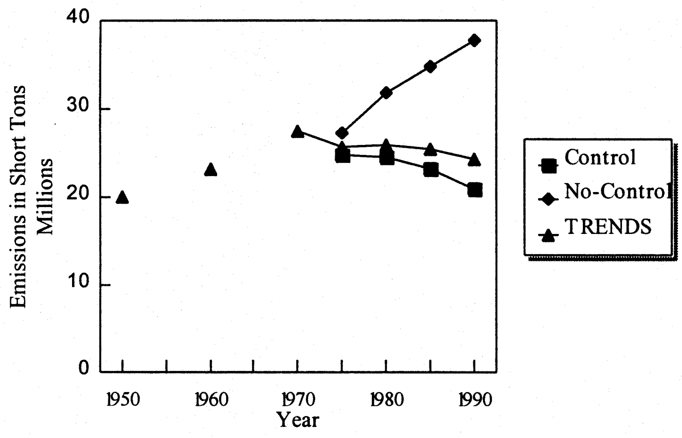


Figure B-4. Comparison of Control, No-control, and *Trends* CO Emission Estimates.

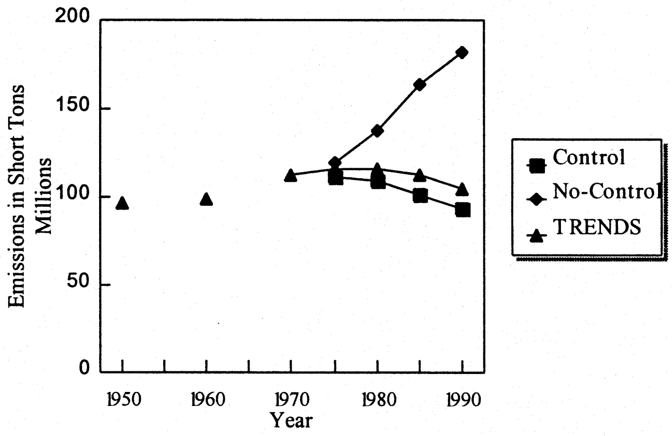
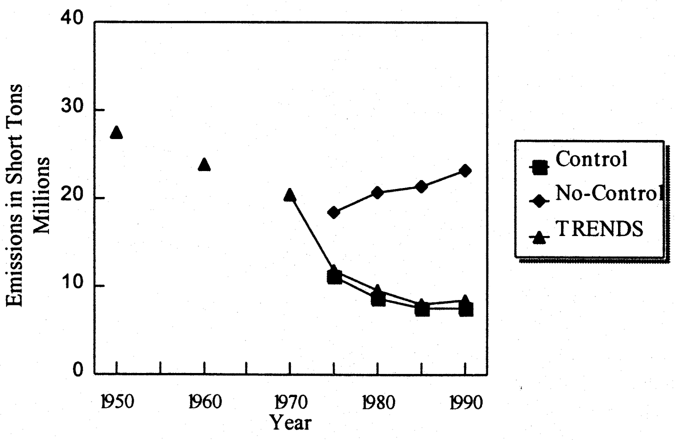


Figure B-5. Comparison of Control, No-control, and *Trends* TSP Emission Estimates.



ring for VOC and CO emissions. The *Trends* report VOC estimates are generally higher than the control scenario estimates due to the inclusion of Waste Disposal and Recycling as a VOC source in the *Trends* report. This inconsistency is of no consequence since Waste Disposal and Recycling sources were essentially uncontrolled by the historical CAA and therefore do not appear as a difference between the control and no-control scenarios. The higher CO emission estimates in the *Trends* Report are primarily associated with higher off-highway vehicle emissions estimates. Again, since off-highway emissions do not change between the control and no-control scenario in the present analysis, this inconsistency is of no consequence.

No-Control Scenario Projections Versus Historical EPA Trends Data

Comparisons between the control scenario emissions estimates generated for the present study and 1970 to 1990 emissions estimates obtained from the *Trends* Report are useful for assessing the reasonableness of the control scenario estimates. As indicated above, there is close correspondence between the control scenario and the *Trends* Report. It may also be useful to compare the pre-1970 historical emissions data from the *Trends* Report[2] with the no-control scenario estimates presented herein to assess whether these pre-1970 trends can be reasonably extrapolated to the 1970 to 1990 period. In addition, examination of any significant changes in emissions trends between the pre-1970 *Trends* data and post-1970 no-control projections might indicate flaws in the emissions modeling conducted for the present study.

For SO_2, the 1950 to 1970 *Trends* data in Figure B-1 demonstrate the effects of the huge increase in fossil fuel combustion between 1960 and 1970. This net increase occurred, despite the obsolescence of coal-fired locomotives and reductions in coal refuse burning, largely because utility emissions nearly doubled between 1950 and 1960, and nearly doubled again between 1960 and 1970.[3] Although no-control scenario projections for the post-1970 period show significant additional increases in SO_2 emissions, the rate of growth is markedly slower than during the 1950 to 1970 period.

The *Trends* data for 1950 to 1970 NO_x shown in Figure B-2 indicate the steady increase in emissions resulting from increased combustion of natural gas and gasoline.[4] The post-1970 emissions estimates derived for the present study reflect a continuation of this trend.

Emissions of VOCs increased steadily over the 1950 to 1970 period, as shown in Figure B-3, primarily due to increases in industrial production and vehicular travel.[5] The no-control scenario emission estimates continue this trend throughout the 1970 to 1990 period, with some acceleration of the rate of change due to the rapid increase in VMT projected under this scenario.

The *Trends* data shown in Figure B-4 for CO indicate an overall increase between 1950 and 1970. This increase occurred despite significant reductions in emissions from stationary source fuel combustion and industrial processes because mobile source emissions nearly doubled during this period.[6] Under the no-control scenario of the present study, additional reductions from stationary sources are not available to offset the transportation-related increases; therefore, the rate of increase in CO emissions after 1970 under the no-control scenario reflects the rapid increase in mobile source emissions caused by increases in vehicle miles traveled.

Finally, Figure B-5 demonstrates a directional shift in emissions of primary particulates between the 1950 to 1970 *Trends* data and the post-1970 no-control scenario. The declining trend from 1950 to 1970 indicated by the *Trends* data, however, is largely due to reductions in use of coal-fired locomotives, reductions in residential coal-burning, coarse (i.e., visible) particle emissions controls installed on fossil fuel combustors and industrial processes, and reductions in forest fires and other open burning.[7] Since the reductions achievable from these sources were largely achieved by 1970, they are no longer available to offset the increases observed from other source categories (e.g., highway vehicles). The no-control scenario therefore shows a steady increase in overall emissions of primary particulates after 1975.

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[2] While 1970 to 1990 Trends data were obtained from more recent *Trends* reports, the 1950 to 1970 *data* were obtained from the November 1991 report since this was the last year the *Trends* report series included data for this period.

[3] U.S. EPA, “National Air Pollutant Emission Estimates, 1940 - 1990”, EPA-450/4-91-026, November 1991, Table 4, p. 16.

[4] U.S. EPA, “National Air Pollutant Emission Estimates, 1940 - 1990”, EPA-450/4-91-026, November 1991, p. 42.

[5] U.S. EPA, “National Air Pollutant Emission Estimates, 1940 - 1990”, EPA-450/4-91-026, November 1991, p. 42.

[6] U.S. EPA, “National Air Pollutant Emission Estimates, 1940 - 1990”, EPA-450/4-91-026, November 1991, Table 7, p. 19.

[7] U.S. EPA, “National Air Pollutant Emission Estimates, 1940 - 1990”, EPA-450/4-91-026, November 1991, Table 3, p. 15.

The following sections of this appendix summarize the methodologies used to model control and no-control scenario emissions for each of the six major emission sectors. Additional details can be found in the supporting documents listed in the References section of this appendix.

Industrial Boilers and Processes

For the purposes of the retrospective analysis, the industrial sector was divided into two components: (1) boilers; and (2) industrial processes and process heaters. The factors affecting emissions from these two source types are different, and, as a result, separate methods were used to calculate control and no-control scenario emissions in each of the target years. To analyze the change in emissions from industrial boilers, ANL used the ICE model (Hogan, 1988). This model was developed under the auspices of NAPAP to forecast State-level fuel choice and emissions from conventional, steam raising, industrial boilers. For the retrospective analysis of industrial processes and fuel use emissions from process heaters, ELI used the EPA *Trends* methods and the ANL MSCET data base (EPA, 1991; Kohout et al., 1990). The *Trends* report contains estimates of national emissions for a variety of industrial sources for the time period of interest. The MSCET data base provided the spatial distribution used to calculate State-level emissions.

The distinction between industrial boilers and non-boiler industrial processes was necessitated by the structure of the CAA regulations and by the factors affecting emission levels from these two source types. Boilers are regulated differently from processes and process heaters. Emissions from industrial processes are primarily a function of levels of industrial activity. The emissions from fuel combustion, however, are a function of energy use and fuel choice as well as industrial activity. Fossil fuel emissions in the absence of the CAA are not proportional to industrial output, since the level of energy use is a decision variable for the firm in its production process. Therefore, in the ICE model simulations used to estimate no-control scenario boiler emissions, the level (and type) of energy use were determined first, and then the effects of emission regulation were taken into account.

Overview of Approach

Industrial Boilers

ICE model inputs include fuel prices, total boiler fossil fuel demand by industry type, and environmental control costs. The outputs of the ICE model were SO_2, NO_x, and TSP emissions by State, industry, and boiler size class. The model runs in 5-year increments and has a current base year of 1985.

The model required boiler demand input data at the State level. Seven industry types were included in the ICE model: Standard Industrial Classification (SIC) codes 20, 22, 26, 28, 29, 33, and “other manufacturing.” ANL’s approach assumed that industrial boiler fuel use occurs only in the manufacturing sector. The model also required fuel price data in each of the target years at the Federal Region level. Prices by grade of coal and petroleum product, such as sulfur content and heating value, were used by the model to determine the cost of compliance, and to determine emissions when the regulations are not binding.

Control costs were computed by engineering subroutines in the model. These costs were used by the ICE model’s fuel choice component to determine the effect of CAA-related costs on the market share of a particular fuel. This fuel choice decision only applies to new industrial boilers, since the cost of existing emission controls are not in the ICE data base and fuel choice is not re-evaluated for existing boilers.

Industrial Processes and In-Process Fuel Combustion

The calculation of historical emissions from industrial processes uses EPA *Trends* methods to estimate national emissions for the analysis years, then allocates these emissions to States using the State shares from the MSCET data base.

MSCET uses a variety of methods to estimate historical emissions for the various industrial sectors. For industrial process emissions, MSCET is based on historical data on industrial activity to allocate emissions based on the State level distribution of the polluting activities. The State level distribution and benchmark is based on the 1985 NAPAP Inventory (EPA, 1989). This approach implies that the MSCET data corresponds directly to the 1985 NAPAP Inventory, and that, for any State, the sum of the emissions from Source Classification Codes (SCCs) that comprise the MSCET industry sector are equal to the MSCET data for that State and sector. Data from *Trends* are used by MSCET to provide information on changes in the aggregate level of control for years other than the 1985 benchmark. Since no direct correspondence existed between the *Trends* data and MSCET, a relationship was developed to link MSCET sectors to *Trends* industry categories and to industry categories in the J/W model, which was used to change activity levels for the no-control scenario.

Table B-1 shows the relationship between the sector definition used by MSCET, *Trends*, and the J/W model. The mapping from MSCET to J/W and *Trends* is used to provide the changes in aggregate activity and emission control for the calculation of no-control scenario emissions.

Establishment of Control Scenario Emissions

Energy use and corresponding emissions were broken down between boilers and non-boiler industrial processes. The latter category includes furnaces, kilns, internal combustion engines (e.g., compressors), and other non-steam types of process heat. The focus of this analysis is on boiler emissions, which were subject to increasingly stringent regulations over the 1970 to 1990 period. (Emissions from some types of industrial processes were also regulated, but regulation of non-boiler sources was targeted on the emissions from the industrial process itself, not on its fuel combustion) For this study, ANL assumed that only boiler fuel use is affected by emission regulations. The non-steam boiler portion of industrial fuel use is not directly affected by the CAA. This portion of the emissions may be affected indirectly by changes in industry activity level and fuel consumption. The emissions from non-boiler industrial processes were calculated separately by ELI.

Control Scenario Boiler Emissions

Control scenario boiler SO_2, NO_x, and TSP emissions were calculated by the ICE model. The MSCET data base provided an estimate of historical emissions for total fossil fuel combustion by industry. Since MSCET does not identify the two required components of boiler and non-boiler emissions, ANL defined the residual of the ICE model control scenario and MSCET as the non-boiler or in-process fuel use emissions. For the relevant study period, MSCET provided a control scenario estimate of total boiler and non-boiler emissions, which was used to calculate the control scenario State-level boiler emissions based on a special run of the ICE model.[8]

In order to use ICE to model the historical emissions path, it was necessary to construct a new ICE model base year file and new user input file so that the model could begin its calculations from 1975 conditions. Construction of the base year file was completed in two stages, using two different data sources, as discussed below. The user input file has several elements, including energy prices and historical boiler fuel use; its construction is discussed in the next section. The model base year file provided the energy use in boilers and corresponding emission control regulations (State Implementation Plans –SIPs– for example) by several categories. These categories include:

- State;
- Industry group (one of seven);
- Fuel type (natural gas, distillate or residual fuel oil, and coal);
- Boiler size class (MMBTU/hr, one of eight categories);
- Utilization rate (one of five categories); and
- Air quality control region (AQCR).

For the purposes of ANL's analysis, only the first three categories were assumed to vary. In other words, for each State, industry, and fuel type combination, the distribution of boiler size, utilization rate, and AQCR was assumed to be constant. Over time, however, changes in the aggregate composition of State, industry, and fuel type would cause corresponding changes in the aggregate composition of the other three characteristics. As mentioned previously, the current base year file was 1985. The retrospective analysis required a 1975 base year. Because of data limitations, the approach to construct a new base year was achieved in the following two steps: the construction of a 1980 interim base year file from the 1985 file, and then the construction of the 1975 file from the interim 1980 file.

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[8] MSCET does not provide State-level estimates of TSP, while ICE does. To estimate total regional TSP from fuel combustion, the *Trends* model was employed. These national emissions estimates were allocated to the States based on the State-level shares of TSP from the NAPAP inventory.

Table B-1. Correspondence Between Process Emissions Categories Used by MSCET, *Trends*, and J/W Industrial Sectors and Identifier Codes.

MSCET Category	**MSCET Code**	***Trends* Industry Category**	**J/W Code**	**J/W Industry Category**
Food Proc. and Agric. Operations	FOODAG	Cattle Feed Lots (0211)	1	Agriculture/forestry/fisheries
		Cotton Ginning (0724)		
		Feed and Grain Milling (204)		
		Grain Elevators (4421,5153)		
Mining Operations	MINING	Metallic Ore Mining (10)	2	Metal Mining
		Coal Mining (1211)		
Oil and Gas Extraction	OILGAS	Crude Oil Production, Storage, and Transfer (1211,4463)	3	Oil & Gas Extraction
		Natural Gas Production (1311)		
Mining Operations	MINING	Crushed Stone (142)	5	Nonfuel mining
		Sand and Gravel (144)		
		Clays (145)		
		Potash/Phosphate Rock (1474,1475)		
Degreasing	DEGRS	Degreasing	NA	Manufacturing
Misc. Industrial Processes	MISIND	Adhesives		
Indus. Organic Solvent Use, Misc.	SOLV	Other Organic Solvent Use		
		Solvent Extraction Processes		
Surface Coating	SRFCT	Surface Coating	NA	Durable Goods
Misc. Industrial Processes	MISIND	Lumber and Plywood (24)	11	Lumber & Wood Products
Cement Production	CEMNT	Cement (3241)	19	Stone, Clay, & Glass Products
Glass Manufacturing	GLASS	Glass (321,322)	19	Stone, Clay, & Glass Products
Lime Manufacturing	LIME	Concrete, Lime, Gypsum (327)	19	Stone, Clay, & Glass Products
		Lime (3274)		
Mineral Products Processing	MINRL	Clay Sintering (3295)	19	Stone, Clay, & Glass Products
		Brick and Tile (3251)		
Iron and Steel Production	IRNST	Iron and Steel (3312)	20	Primary Metal Industries
		Ferroalloys (3313)		
		Iron and Steel Foundries (332)		
Other Primary Metals Smelting	OTHMET		20	Primary Metal Industries
Primary Aluminum Smelting	PALUM	Primary Aluminum (3334)	20	Primary Metal Industries
Primary Copper Smelting	PCOPR	Primary Copper (3331)	20	Primary Metal Industries
Primary Lead and Zinc Smelting	PLDZC	Primary Lead and Zinc (3332,3333)	20	Primary Metal Industries
Other Sec. Metal Smelting and Refining	SECMET	Primary Nonferrous Smelters (333)	20	Primary Metal Industries
Other Sec. Metal Smelting and Refining	SECMET	Secondary Nonferrous Smelters (334,336)	20	Primary Metal Industries
Secondary Lead Refining	SLEAD	Secondary Lead (3341)	20	Primary Metal Industries
Food Proc. and Agric. Operations	FOODAG	Food and Beverages (20)	7	Food & Kindred Products
Misc. Industrial Processes	MISIND	Textiles (22)	9	Textile Mill Products
Paper and Pulp Mills Operations	PAPER	Pulp Mills (261,262)	13	Paper & Allied Products
Misc. Industrial Processes	MISIND	Graphic Arts (27)	14	Printing & Publishing
Printing Operations	PRINT		14	Printing & Publishing
Organic Chemicals Manufacture	ORGCM	Organic Chemicals (286)	15	Chemicals & Allied Products
		Carbon Black (2895)		
Other Chemicals Manufacture	OTHCM	Ammonia (2873)	15	Chemicals & Allied Products
		Nitric Acid (2873)		
		Chemicals (28)		
		Sulfuric Acid (2819)		
Petroleum Refining	PTREF	Petroleum Refining (2911)	16	Petroleum & Coal Products
		Asphalt Paving and Roofing (295)	16	Petroleum & Coal Products
Plastics Production	PLAST	Plastics (2821,3079)	17	Rubber and plastic products
Rubber and Misc. Plastics Manufacture	RUBR	Rubber Tires (3011)	17	Rubber and plastic products

Estimates of boiler fossil fuel consumption in 1980 for each State and major fuel type were provided by Hogan (Hogan, 1988). These estimates are based on the assumption that the industry mix, size, utilization, and AQCR distribution within a State are constant. Through assuming this relationship, the 1985 ICE base year was scaled to match the data for 1980, thus forming the 1980 interim base year data.

To construct the 1975 base year file, the assumption of a constant industry mix for a State and fuel type was no longer necessary, since detailed data on each industry for 1980 and 1975 were available from PURchased Heat And Power (PURHAPS) model data files (Werbos, 1983). These PURHAPS data files were derived from the Annual Survey of Manufactures: Fuels and Electric Energy Purchased for Heat and Power (DOC, 1991). The available data in these files were for *total* fuel use not *boiler* fuel use. To make use of these data, it was necessary to assume that the fraction of fuel used in boilers, for any given State and industry, remained constant from 1975 to 1980. To the extent that the fraction of boilers' heat versus process heat applications is a function of the specific industrial production process, this assumption is reasonable.

Based on the assumption of constant boiler fuel fraction of total fuel use, the ratio of 1975 to 1980 energy use for each State, industry, and fuel type was applied to the corresponding record of the 1980 interim base year file to produce 1975 base year files.

Control Scenario Industrial Process Emissions

To estimate boiler emissions of sulfur oxides (SO_x), NO_x, and VOC from industrial processes, data from *Trends* were used. The percentage change in national emissions by *Trends* category was applied to the appropriate sector from MSCET to obtain State-level emissions. In some cases there are several categories in *Trends* that match directly with MSCET categories (see Table B-1). In these cases, the *Trends* sectors were aggregated and the percentage change was computed. It was assumed that the level of control in each industry sector implied by *Trends* was uniform across States. The changes in emissions in each State are not equal to those at the national level, since the industry composition in each State varies.

Development of Economic Driver Data for the Control Scenario - Industrial Boilers and Processes

The results of the J/W model were the primary source of activity in the ICE model driver data. These results were also used by ELI to produce the national results for industrial processes from *Trends*. Both ICE and *Trends* use the forecasted change in industrial activity that results under the no-control scenario. These data were in the form of industry specific changes in energy consumption and industrial output, for boilers and industrial processes.

Economic Driver Data for Industrial Boiler Approach

Using the 1975 base year file as a starting point, the ICE model estimated fuel choice and emissions based on a user input file containing total boiler energy demand and regional energy prices. The 1975, interim 1980, and original 1985 base year files contained the required information on energy demand for each industry group and State, so the data in these three files were aggregated across fuel type, and other boiler characteristics (for example, size). These aggregated data provided the energy demand for three of the target years. Since 1990 State-level data on energy use by industry group were not available at the time of the study, the NAPAP base case forecast for the ICE model for 1990 was used to provide the demand data for this year.

The user input file for ICE also requires a price input for each target year. These prices were input by Federal Region for distillate oil, 4 grades of residual oil (by sulfur content), natural gas, and 11 grades of coal (by sulfur content and coal rank, i.e., bituminous and sub-bituminous). Prices for 1985 and 1990 were obtained from the NAPAP base case user input file. The prices for 1975 and 1980 are from U.S. Department of Energy (DOE) data on State-level industrial energy prices (DOE, 1990). Regional prices of natural gas, distillate oil, steam coal, and residual oil were constructed by aggregating expenditures across States within each region and dividing by total British thermal unit (BTU) consumption for the years 1975, 1980, and 1985. Since prices by sulfur content grade are not reported by this DOE source, ANL assumed that the sulfur premium implied by the 1985 ICE model input file was proportional to the average price. Based on this assumption, the ratio of the regional coal and residual oil price in 1975 and 1980 to the 1985 price was applied to the 1985 price in the ICE model base case file for each grade of fuel. To provide additional consistency between the NAPAP analysis and ANL's study, the distillate oil and natural gas prices were benchmarked to the 1985 ICE model prices as well.

One possible inconsistency arises using this procedure. The residual oil and natural gas markets are closely linked, particularly for industrial customers. These markets, specifically the gas market, underwent tremendous changes over the study period. To model the effect of these structural changes on the sulfur premiums in residual oil would require a detailed oil and gas supply model that was beyond the scope of this project. Moreover, the CAA regulations themselves create the potential for sulfur premiums. This potential effect of the CAA was not captured, though, because of the assumption of proportional fuel sulfur premiums on residual fuel oil. The relationship between market driven sulfur premiums in the coal market and the CAA was given additional consideration in this analysis through the use of an explicit coal supply model.

The J/W data for industrial energy consumptions was supplied in the form of percentage change in cost shares. In order to compute the percentage change in the quantity of energy used, ANL used the following identity:

$$1n\left(\frac{P_E \times E}{P_Q \times Q}\right) = 1n\,(P_E) + 1n\,(E) - 1n\,(P_Q \times Q), \text{ or}(1)$$

$$1n\left(\frac{P_E \times E}{P_Q \times Q}\right) - 1n\,(P_E) + 1n\,(P_Q \times Q) = 1n\,(E), \text{ or}(2)$$

The percentage change in E is the percentage change in cost share, minus the change in price, plus the change in value of shipments. These calculations were performed for each energy type and industry sector in the J/W model. The ICE model requires total fuel use, so the fuel specific percentages were weighted by historical fuel consumption to produce an aggregate change in fuel consumption to apply to the ICE model input data files.[9]

ICE also uses energy prices to simulate boiler fuel choices. The control scenario forecasts of energy prices in ICE were adjusted based on the percentage changes in energy prices, by coal, oil and natural gas. This implicitly assumes that the oil and coal fuel sulfur premiums, by region, are proportional to the average national price. To test this assumption for the coal market, additional modeling of the coal prices was performed using the coal market component of the ARGUS model.

It is possible that in some regions low sulfur coal prices to the industrial sector may be lower than the national average. This was not found to be the case. For example, in 1990, delivered regional industrial coal prices change by less than two-thirds of one percent. In most cases, the percentage change was near zero. This result appears to occur because of the highly regional nature of the coal market. While the artificial demand for low sulfur coal may fall, power plants near low sulfur coal reserves now find it advantageous to buy this local coal, which raises the price back to an equilibrium level near to that of the control scenario. This is even more likely to be true of industrial delivered prices, since industrial prices are more affected by transportation costs than are the utility prices. No additional ICE modeling was performed.

Economic Driver Data for the Industrial Process Approach

The J/W model was also used to account for activity level changes in the calculation of industrial process emissions under the no-control scenario. The correspondence between *Trends*, MSCET, and the J/W model was used to apply changes in industrial activity in each target year to each industrial process.

No-control Scenario Emissions

Industrial Boiler Emissions of SO_2, NO_x, and TSP

The CAA imposed different regulations, SIPs, and New Source Performance Standards (NSPS) that apply to industrial boilers of varying size. The primary effect of CAA regulations on industrial boilers was simulated by defining the Air Quality Control Region (AQCR), the resulting SIPs, and subsequent NSPS for boilers. The industrial boiler SIP regulations were included in the ICE base year file discussed in the previous section. Since the ICE model estimates new boiler emissions for each target year, the boiler NSPS are input through the ICE user files. Industrial NSPS were implemented in two phases. The 1971 regulations are imposed for the study years 1975 and 1980.

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[9] ICE uses six of the manufacturing industries from the J/W model directly. The remaining industries' percentage changes were weighted to produce the "other" category.

The 1984 NSPS revisions are imposed in the study years 1985 and 1990. For the no-control scenario, ANL set the SIPs and NSPS to a flag that indicated "no regulation."

Industrial Boiler Emissions of CO and VOC

Two of the criteria pollutants emitted by industrial fuel combustors, CO and VOC, were not included as outputs of the ICE model. Therefore, CO and VOC emissions were analyzed separately using *Trends* methods. Control scenario CO and VOC emissions were taken directly from *Trends*.

To estimate CO and VOC emissions from industrial combustion for the no-control scenario, fuel use for industrial manufacturing was adjusted, reflecting fuel consumption changes estimated by the J/W model. These changes in the level of fuel consumption by industrial combustion were also used in ANL's ICE boiler model. Changes in industrial combustion fuel use by manufacturing between the control and no-control scenarios are reported in Table B-2. These estimates represent an average of several sectors, which were developed by ANL as part of the modeling process for ICE.

No-control scenario emissions were computed using 1970 emission factors. Since there were no add-on controls for industrial combustion VOC and CO emissions, it was not necessary to adjust the no-control scenario for changes in control efficiency.

Table B-2. Fuel Use Changes Between Control and No-control Scenarios.

Year	Fuel Type	Fuel Use Changes
1975	Coal	-.0042
	Oil	+.0311
	Gas	-.0064
1980	Coal	-.0061
	Oil	+.0107
	Gas	-.0095
1985	Coal	-.0061
	Oil	+.0089
	Gas	-.0097
1990	Coal	-.0079
	Oil	+.0091
	Gas	-.0099

Emission estimates were regionalized using State-level emissions data from industrial boilers recorded in MSCET. For the control scenario estimates, VOCs were regionalized using the MSCET State-level shares for industrial fuel combustion. In the no-control scenario, the State-level shares were held constant. The control scenario emissions of CO were regionalized using the control scenario NO_x emissions from the ICE model. This approach assumes that CO emissions are consistent with NO_x emissions. The no-control scenario CO emission estimates from industrial combustion sources were regionalized using no-control NO_x emission estimates from industrial combustion sources.

Industrial Process Emissions

A wide range of controls were imposed on industrial processes. These emission limits are embodied in the assumptions of control efficiencies in the *Trends* model. Data on national no-control scenario emissions from industrial processes were provided by EPA. These data were combined with MSCET to produce regional-level results.

Lead Emissions

Estimates of lead emissions from industrial boilers and industrial processes were completed by Abt Associates. The methods used for calculating lead emissions from industrial processes and industrial boilers were similar. The starting point was the TRI, which provides air toxics emissions data for manufacturing facilities with more than 10 employees. To estimate lead emissions from industrial boilers and processes, 1990 facility-level lead emissions data were extracted from the TRI. These data were then adjusted to create estimates of lead emissions from industrial sources under the control and no-control scenarios for each of the target years. For the control scenario, lead emissions for 1975, 1980, and 1985 were obtained by extracting an emission factor and a control efficiency for each lead-emitting industrial process in the *Trends* data base. These emission factors and control efficiencies were multiplied by the economic activity data for each year for each process as reported in *Trends* to yield estimated control scenario emissions by industrial process. Each industrial process was assigned a code to correspond with energy consumption data by industrial process compiled in the National Energy Accounts (NEA) by the Bureau of Economic Analysis, and emissions were summed over all processes to obtain a total for each target year.

For consistency with the other emission estimates in this analysis, industrial process no-control scenario lead emissions were adjusted for changes in industrial output, and for changes in emissions per unit of output due to control technology applications. Changes in industrial output were accounted for using results from the J/W model. Lead-emitting industrial processes in the *Trends* data base were assigned to a J/W sector. For each sector, the percentage change in economic output was used to adjust the economic activity data for that process from the *Trends* data base. These adjusted economic output figures were used with the 1970 emission factors and control efficiencies to derive the estimated no-control scenario lead emissions for each industrial process in each target year. The process-level emissions were then aggregated to the NEA-code level as in the control scenario.

The lead emission estimates from industrial processes, by NEA code, were used to derive percentage changes in emissions under the control and no-control scenarios by NEA code for application to the TRI emissions data. Since TRI data are reported by SIC code, NEA codes were “mapped” to the appropriate SIC codes, and then the percentage change for each NEA code was used to represent the percentage change for all SIC codes covered by that NEA code.

To calculate lead emissions from industrial boilers, Abt Associates developed estimates of lead emissions from industrial combustion under the CAA for each of the target years. The *Trends* data base contains national aggregate industrial fuel consumption data by fuel type. For each fuel type, the fuel consumption estimate was disaggregated by the share of that fuel used by each NEA industrial category. The *Trends* data base also contains emission factors for industrial fuel use, by fuel type, as well as control efficiencies. The lead emissions from industrial combustion for each NEA category were derived by multiplying the fuel-specific combustion estimate for each NEA category by the emission factor and control efficiency for that fuel type. The result was emissions of lead by NEA code and by fuel type. Emissions from all fuel types were then summed by NEA code. The NEA data were used to disaggregate the industrial fuel consumption figures, based on the assumption that the ICE are the same among all industries covered by a given NEA code.

To estimate no-control scenario lead emissions, the macroeconomic effect of the CAA and the change in emissions per unit of output that resulted from specific pollution control mandates of the CAA were both taken into account. As in the control scenario, the national aggregate industrial fuel consumption estimate by fuel type was disaggregated by the share of that fuel used by each NEA industrial category. The fuel use was then adjusted in two ways: some NEA codes were specifically modeled by the ICE model, and for the remaining NEA codes, J/W percentage changes in fuel use were applied. These fuel use estimates were then combined with the 1970 emission factors and control efficiencies for industrial combustion by fuel type from the *Trends* data base to obtain no-control scenario combustion-related lead emissions from industrial boilers by NEA code. These estimates of total lead emissions by NEA codes were matched to SIC codes, and then to the data in the TRI data base. This approach assumed that an average emission value was assigned to all reporting TRI facilities in a given SIC code.

Off-Highway Vehicles

The off-highway vehicle sector includes all transportation sources that are not counted as highway vehicles. Therefore, this sector includes marine vessels, railroads, aircraft, and off-road internal combustion engines and vehicles. As a whole, off-highway vehicle emissions are a relatively small fraction of total national anthropogenic emissions.

Overview of Approach

The process used by ELI to determine the national level of emissions from the off- highway transportation sector is similar to the procedure outlined above for industrial processes. To estimate the emissions of criteria air pollutants from these sources under the no-control scenario, the historical activity levels were held constant, rather than attempting to calculate a new no-control scenario level of off-highway vehicle activity. This assumption was necessary since the off-highway activity indicators (amount of fuel consumed, and landing and take-off cycles for aircraft) do not have direct correspondence with a given J/W category. The national no-control scenario emissions of criteria air pollutants from these sources were simply derived by recalculating emissions using 1970 emission factors.

Development of Control Scenario

To estimate control scenario emissions, the analysis relied on *Trends* methods, using historical activity indicators, emission factors, and control efficiencies. Essentially, the estimates of off-highway emissions under the control scenario represent the historical estimates from the *Trends* data base.

No-control Scenario Emissions Estimates

The calculation of off-highway emissions for the no-control scenario required the *Trends* data to be adjusted to reflect changes in controls and economic activity in each of the target years. Linking source activity changes with economic activity for this section is not straightforward. The economic activity data for off-highway engines and vehicles are expressed either in terms of amount of fuel consumed, or in terms of landing and take-off cycles for aircraft. Neither of these off-highway activity indicators has a direct correspondence with a given J/W sector, making the sort of direct linkage between *Trends* categories and J/W sectoral outputs that was used for industrial processes inappropriate.

In the absence of a link between the economic factors that are determinants of emissions from this sector and the available economic activity forecasts, the no-control scenario emissions of criteria air pollutants from off-highway mobile sources were estimated based on the same historical activity levels used for the control scenario. Although there were changes in sectoral output and personal income that might have had an effect on off-highway vehicle usage, these changes were deemed to be small and not likely to have a major effect on the emissions from this sector.

Emission factors for each of the off-highway sources were also held constant at 1970 levels to calculate no-control scenario emissions for each target year. The national emissions of criteria air pollutants from these sources were then recalculated using 1970 emission factors.

National and State-Level Off-Highway Emission Estimates

Table B-3 summarizes national-level emission estimates for off-highway sources. The emission estimates derived from using the methodology discussed above yielded results that seem counter-intuitive. The emissions from off-highway sources, in particular the emissions from aircraft, are lower in the no-control scenario than those projected for the control scenario for most pollutants. This is a result of calculating emissions using 1970 emission factors, since the 1970 emission factors for aircraft are lower than the aircraft emission factors in later years.

ELI identified several potential sources of uncertainty in the emission estimates for this sector. First, the assumption that the total level of off-highway vehicle fuel consumption is constant between the two scenarios may be flawed. Second, the use of 1970 emission factors in the no-control scenario may fail to capture significant changes in technology. These technological changes are implicitly captured in the control scenario and it is possible that these technological changes may also have occurred under a no-control scenario.

One possible response to the biases created by the use of 1970 emission factors for all years in the no-control scenario is to test how results might differ if the emission factors used for the control scenario, which would include technological change, were also used for the no-control scenario. However, using this treatment of emission factors, the emissions projections from the adopted methodology from non-highway sources in the no-control scenario would be identical to the emissions projections under the control scenario. The reason for this is that the economic activity levels were not adjusted for the calculation of emissions under the no-control scenario.

In order to disaggregate the national data to a State level, the methodology used the MSCET data base, which is described earlier. Emissions of VOC, SO_x, and NO_x were regionalized using the State-level shares from the MSCET methodology. The emissions of TSP were regionalized by using the State-level shares for SO_x reported by MSCET, and the emissions of CO were regionalized using the State-level shares for NO_x, also reported by MSCET. The potential bias that this introduces is likely to be small, due to the relative homogeneity of off-highway vehicle emission sources.

Table B-3. Difference in Control and No-control Scenario Off-Highway Mobile Source Emissions.

		1975	1980	1985	1990
TSP	Control Scenario:	268.6	281.1	268.7	280.9
	No-Control Scenario:	260.8	268.8	261.2	266.9
	Percentage Increase:	-3%	-4%	-3%	-4%
NO_x	Control Scenario:	1,987.6	2,176.7	2,077.5	2,085.9
	No-Control Scenario:	1,974.6	2,150.5	2,042.7	2,058.9
	Percentage Increase:	-1%	-1%	-2%	-1%
SO_2	Control Scenario:	364.6	531.1	406.4	392.5
	No-Control Scenario:	363.2	528.6	403.0	386.9
	Percentage Increase:	0%	0%	-1%	-1%
CO	Control Scenario:	8,512.8	8,101.4	7,881.9	8,079.0
	No-Control Scenario:	8,511.0	8,071.2	7,880.2	8,077.7
	Percentage Increase:	0%	0%	0%	0%
VOCs	Control Scenario:	1,374.9	1,370.8	1,334.8	1,405.0
	No-Control Scenario:	1,385.9	1,416.1	1,388.6	1,485.8
	Percentage Increase:	1%	3%	4%	6%

Note: Emission estimates are expressed in thousands of short tons. Percentage increase is the differential between scenarios divided by the Control Scenario projection.

As with regionalization of industrial process emissions, the State-level shares are held constant between the two scenarios. To the extent that the distribution of economic activity between States was not constant over the period of the analysis, holding State-level emission shares constant may bias the results, although the direction and magnitude of the potential bias is unknown.

On-Highway

This section addresses the highway vehicle portion of the transportation sector. Highway vehicle emissions depend on fuel type, vehicle type, technology, and extent of travel. Emissions from these vehicles have been regulated through Federal emission standards and enforced through in-use compliance programs, such as State-run emission inspection programs. Vehicle activity levels are related to changes in economic conditions, fuel prices, cost of regulations, and population characteristics. Emissions are a function of vehicle activity levels and emission rates per unit activity.

TEEMS was employed by ANL to analyze the transportation sector. The modeling system links several models, disaggregate and aggregate, to produce State-level estimates of criteria pollutants. The system is subdivided into two modules: an activity/energy module and an emissions module. Each module contains multiple models. TEEMS has been documented in several reports and papers (Mintz and Vyas, 1991; Vyas and Saricks, 1986; Saricks, 1985). It has been used for several policy analyses and assessment studies for DOE and NAPAP. This section presents an overview of the approach used to conduct the analysis of the transportation sector. Also included in this section is a summary of the methodology used by Abt Associates to estimate changes in lead emissions from highway vehicles in each target year.

Overview of Approach

TEEMS has two modules: an activity/energy module and an emissions module. The activity/energy module calculates emissions based on: (1) personal travel; (2) goods movement; and (3) other transportation activity inputs.

Personal Travel

Personal travel activity and resulting fuel consumption were calculated for each target year using procedures that disaggregate households by demographic and economic attributes. Economic driver data, developed from U.S. Government data and macroeconomic model(s) of the domestic economy, formed the basis for household disaggregation. Modeling procedures were employed by ANL to project movement of households between various attribute classes, and vehicle holdings were projected in terms of the number and type of vehicles held by each household type. National totals were then developed by aggregating the vehicle holding estimates for each household type, accounting for the number of households of that type. Travel estimates, in terms of VMT, were calculated using the same approach, and based on the VMT of each household type. The basis for household transportation activity projection has been empirically established through analysis of the 1983-84 Nationwide Personal Transportation Survey (NPTS) (FHWA, 1986; Mintz and Vyas, 1991). VMT are projected using this empirical relationship, and estimates of the elasticity of VMT to vehicle operating cost are then made. Energy consumption was estimated in each target year using VMT, shares of VMT by vehicle type, and exogenously developed vehicle characteristics.

The following three models and an accounting procedure were employed to develop target year personal travel activity projections:

1. The first model projected the target year distribution of households by their attributes. This model employed an iterative proportional fitting (IPF) technique and projected the number of households in each cell of the household matrix - each of which is defined by various categories within six household attributes.

2. The second model projected changes in vehicle ownership resulting from changes in income and cost of vehicle operation. The model applied estimated ownership changes to each target year household matrix such that the control values within each of the household attributes, excepting vehicle ownership, remained unchanged.

3. The third model estimated the composition of household vehicle fleet by type (cars and trucks), size, technology, and fuel.

4. An accounting procedure applied VMT per vehicle to vehicle ownership in each combination of household attributes. VMT and energy consumption were accumulated by vehicle type, size, and fuel.

Each of these models is described separately in the following subsections.

Iterative Proportional Fitting (IPF)

This IPF model modified a control scenario matrix of household counts. A household matrix was developed from the 1983 NPTS data and upgraded to the year 1985 using published aggregate data. The procedure used in constructing the 1985 household matrix has been documented elsewhere (Appendix B of Mintz and Vyas, 1991). The matrix is defined by six attributes: (1) residential location (central city, suburb, rural); (2) household income; (3) age of householder; (4) household size; (5) number of drivers; and (6) number of vehicles. The household matrix has 3,072 cells, some of which are illogical (such as 1 person, 2 drivers). Illogical cells were replaced with zeros.

Household shares within each attribute in each target year were developed exogenously using data from the Bureau of the Census and selected macroeconomic model runs. The projected total of households and shares of households in each category of an attribute were supplied to the IPF model. The model modified the control scenario household matrix to match the specified shares and total number of households.

The IPF model treated household distribution within each attribute as a set of vectors. These vectors were scaled to match the specified shares and household total. Following the initial scaling, a gradual scaling technique was used to move in the direction of the target shares. The scaling process was repeated until closure was achieved for all attribute classes. Since vehicle ownership levels were estimated by the vehicle ownership model (described in the next section), shares within the sixth household attribute (number of vehicles held) were not specified, leaving it uncontrolled. This flexibility of an uncontrolled attribute helped to facilitate the model operation. The number of households in each class of vehicle ownership within the output matrix represents distribution of households using the control scenario (1985) relationship of vehicle ownership to other household attributes.

Vehicle Ownership Projection (VOP)

The VOP model projected the changes in vehicle ownership resulting from changes in the number of licensed drivers, disposable personal income, and annual fuel cost of vehicle operation. The model is based on historical household ownership rates. A target per-driver ownership rate was computed using disposable income and fuel cost. This target rate represented desired ownership if income and fuel cost were the only determinants. A parameter representing ownership responsibilities such as acquisition effort, disposal effort, parking requirements, and other indirect aspects was applied to adjust this target. The new ownership rate was used to estimate the number of household vehicles.

The household matrix created by the IPF model was revised to match the projected household vehicle ownership. Household shares within the first five attributes remain constant while those within the sixth attribute (i.e., number of vehicles) were variable. A deviation measure was defined and its value for each class within the first five attributes was minimized. A set of simultaneous equations was solved using Lagrangian multipliers.

Projection of Vehicle Fleet Composition

The composition of household vehicles was projected for each household matrix cell using a vehicle choice model called the Disaggregate Vehicle Stock Allocation Model (DVSAM). Vehicles are defined by type (auto, light truck), size (small, mid-size, full-size auto; small pickup, small utility/minivan, standard pickup, large utility/standard van; or any other size classification), fuel (gasoline, diesel, methanol, ethanol, or compressed natural gas), and technology (stratified charge, direct injection, electric, fuel cell, or Brayton).

The model computed vehicle composition based on an individual vehicle's utility to households and household needs. A menu of vehicles classified by the previously mentioned vehicle attributes was supplied to the model. The menu specified characteristics of each vehicle available to households. Vehicles were characterized by price, operating cost, seating capacity, curb weight, and horsepower. These variables formed the basis for computing "utility" (analogous to consumer satisfaction). The household matrix provided demographic and economic attributes which, when combined with vehicle usage in miles, define household needs. Vehicle usage (VMT) was computed as a function of income, number of drivers, and number of vehicles. A logit model was applied to compute vehicle ownership shares. Several model enhancements facilitated modeling of limited range vehicles, and representation of supply constraints and/ or regulated market penetration.

Activity/Energy Computation

An accounting procedure was applied to compute personal travel activity in terms of VMT by vehicle type. Control scenario VMT per vehicle estimates for each cell in the household matrix were developed from the 1983 NPTS. These rates were adjusted within the procedure on the basis of changes in average vehicle operating cost per mile for each cell. The vehicle composition projection model computes ownership shares and share-weighted change in vehicle operating cost. Elasticity values were applied to this change.

ANL assumed that VMT per vehicle remained nearly unchanged for a household matrix cell over time (with the exception of the effect of changes in vehicle operating cost). In other words, variation of VMT across household types is far greater than within household types. VMT per household vehicle remained stable during the period from 1977 to 1984 (Klinger and Kuzmyak, 1986). Some increases were observed in recent years, which were attributed to lower fuel prices and increased household income (DOC, 1991; FHWA, 1992). (A portion of the increase could be attributed to the method of computing average VMT per vehicle.) The assumption that VMT per vehicle for each cell remained nearly constant and was elastic relative to vehicle operating cost is reasonable. As households move from one cell of the matrix to another, they "acquire" the VMT per vehicle rate of that cell. Thus, this approach accounted for changes in VMT per vehicle due to increased household affluence, increased rate of driver licensing, changes in fuel price, and changes in vehicle technology.

Goods Movement

Energy and activity demand resulting from movement of 24 aggregate categories of commodities is estimated by this subcomponent of the TEEMS activity module. Changes in commodity demand/production were provided by growth indexes by two-digit SIC generated by a macro model. A model that projects shifts in mode shares among truck, rail, marine, air, and pipeline modes was used, followed by a procedure to compute ton miles of travel for each mode, VMT by fuel type for trucks, and energy consumption by operation type for non-highway modes. The model used 1985 control scenario data, which were compiled from railroad waybill sample and publications, waterborne commerce publications, transportation statistics, and other sources. The procedure used in developing the 1985 control scenario freight data has been documented in an ANL report (Appendix A of Mintz and Vyas, 1991).

This goods movement model was not used for this retrospective analysis because of funding and time constraints. A procedure to estimate truck VMT by fuel type was employed in its place. Published historical VMT values (FHWA, 1988; 1992) were used along with VMT shares by fuel and truck type from Truck Inventory and Use Surveys (TIUS) (DOC, 1981; 1984; 1990).

Other Transportation Activities

The activity/energy module also has other models for developing activity and energy use projections for air, fleet automobiles, and bus modes. Fleet automobile activity estimates from an earlier study (Mintz and Vyas, 1991) were used while other modes were not analyzed.

Lead Emissions

Estimates of lead emissions in the transportation sector were developed by Abt Associates based on changes in reductions of lead in gasoline. This estimation required the estimates of lead in gasoline consumed over the period from 1970 to 1990 and the amount of lead content in gasoline that would have been consumed in the absence of the CAA. These values were calculated using the quantity of both leaded and unleaded gasoline sold each year and the lead concentration in leaded gasoline in each target year. Data on annual gasoline sales were taken from a report by ANL that presented gasoline sales for each State in each target year. For the control scenario, data on the fraction of gasoline sales represented by leaded gasoline were used. For the no-control scenario, all of the gasoline sold was assumed to be leaded. Data on the lead content of gasoline was obtained from ANL for 1975 through 1990. For 1970 through 1975, the analysis assumed that the 1974 lead content was used.

Estimation of No-control Scenario Emissions

TEEMS emissions projections were carried out by ANL in the following three steps:

1. Development of emission factors;
2. Allocation of highway activity to States; and
3. Development of highway pollutant estimates.

The following subsections describe the procedures used for computing highway vehicle emissions.

Development of Emission Factors

EPA's MOBILE5a Mobile Source Emission Factor model was used to provide all of the highway vehicle emission factors used to estimate 1975 to 1990 emission rates (EPA, 1994b). Documentation of the MOBILE5a model is found in the User's Guide for the MOBILE5 model.[10]

Although the actual emission factors used by ANL are not documented in either the original ANL TEEMS model report or in the Pechan summary report, the Project Team provided direction that defined the emission factors to be used. For the control scenario, ANL was directed to use the official EPA emission factors prevailing at the time for each target year. For example, the official EPA emission factor being used in 1980 for on-highway vehicle NO_x was to be used to estimate 1980 control scenario on-highway vehicle NO_x emissions. For the no-control scenario, the official EPA emission factors used to estimate emissions in 1970 were to be used throughout the 1970 to 1990 period.

It is important to note that using the 1970 on-highway vehicle emission factors to estimate no-control scenario emissions for the entire 1970 to 1990 period may bias scenario emission differentials upward. This is because it is possible that technological changes to on-highway vehicles unrelated to CAA compliance strategies may have yielded incidental reductions in emissions. However, EPA Office of Mobile Sources (EPA/OMS) experts indicate that the two major technological changes in vehicles occurring during the period of the analysis –electronic ignition and electronic fuel injection– would have yielded negligible emission reductions in the absence of catalytic converters.[11]

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[10] EPA/OAR/OMS, "User's Guide to MOBILE5," EPA-AA-AQAB-94-01, May 1994; see also 58 FR 29409, May 20, 1993.

Another potential bias is introduced by assuming the CAA had no substantial effect on vehicle turnover. However, two factors render this potential bias negligible. First and foremost, under the no-control scenario retired vehicles would be replaced by new but equally uncontrolled vehicles. Second, no-control scenario vehicle use is greater in terms of VMT per year. This means no-control scenario vehicles would reach the end of their service lives earlier, offsetting to some extent the alleged incentive to retire vehicles later due to costs imposed by CAA control requirements.

Allocation of Highway Activity to States

TEEMS' activity module generated national activity and energy estimates. These activity totals were allocated to States through a regionalization algorithm that used time series data on historical highway activity shares by State. A trend extrapolation methodology was used that stabilizes shifts after 5 years in the future. For the retrospective analysis, historical highway activity shares for each target year were developed using data published by the Federal Highway Administration (FHWA) (FHWA, 1988; 1992).

Development of Highway Pollutant Estimates

Highway emission estimates were calculated in both scenarios for each target year using VMT estimates generated by TEEMS and emission factors from MOBILE5a. Control scenario activity levels were adjusted for the no-control scenario using economic forecasts and historical data.

Control Scenario Emissions Calculation

Control scenario data for the transportation sector were compiled from several sources. Household counts and shares of households by six attributes were obtained from various editions of the *Statistical Abstracts* of the United States. Household income information was obtained from the control scenario run of the J/W model. Fuel prices were obtained from the *Annual Energy Review* (DOE, 1992) while vehicle fuel economy and aggregate VMT per vehicle were obtained from *Highway Statistics* (FHWA, 1988; 1992). B-4 lists data sources for the control scenario run.

Table B-5 shows household shares prepared for the IPF model. The total number of households increased from 63.4 million in 1970 to 93.3 million in 1990. A gradual shift from rural to urban was observed with movement to suburbs within urban areas. The effect of economic downturns in 1975 and 1980 was an increase in share for the lowest income category; more households moved to the highest income group from 1970 to 1990, while the lower middle income group share expanded and the upper middle income share declined. The rate of household formation was high during the 1970's, which resulted in increases in smaller and younger households. The trend in younger households reversed after 1980 as household formation slowed. Average household size dropped from 3.2 in 1970 to 2.67 in 1990. The number of licensed drivers increased throughout the analysis period as more and more young people were licensed to drive.

Data for the VOP model included disposable income per capita, fuel price, overall personal vehicle fuel economy, and annual usage in terms of VMT. Table B-6 shows these data for each year in the analysis period.

Data preparation for the model that projected household vehicle composition was limited to characterization of existing technology vehicles. Seven vehicle size and type combinations were characterized for 1975 and 1980 while one vehicle, minivan/ small utility, was added for 1985 and 1990. Control scenario vehicle characteristics are tabulated in Table B-7. TEEMS' activity and energy computation procedure was executed to produce personal vehicle travel and energy consumption estimates.

Commercial truck travel was not modeled but, historical data published by the FHWA (FHWA, 1987; 1991) were used. FHWA publishes truck travel by three categories: 1) 2-axle, 4-tire trucks; 2) single unit trucks; and 3) combination trucks. All 2-axle, 4-tire trucks were treated as light-duty trucks. VMT by personal light trucks were subtracted from the published totals to arrive at commercial light truck VMT. Diesel truck VMT shares of total VMT were obtained from TIUS (DOC, 1981; 1984; 1990). TIUS data were also used to split VMT by single unit and combination trucks. All combination trucks were assumed to be the heaviest, class 7 and class 8, while single unit trucks could be of any size class 3 through 8. Gasoline and diesel VMT totals were developed for these heavy-duty trucks and were kept constant for the control and no-control scenarios.

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[11] Telephone conversation between Jim DeMocker, EPA/OAR and EPA/OMS/Ann Arbor Laboratory staff (date unknown). Nevertheless, the Project Team did consider reviewing emission factors for European automobiles to attempt to estimate no-control scenario emission factors for 1975 through 1990 reflecting the use of electronic fuel injection and electronic ignition but no catalytic converter. However, the Project Team concluded that differences in fuel/air mix ratios used in Europe would probably obscure any differences in emission rates attributable to the use of electronic fuel injection and electronic ignition.

Table B-4. Sources of Data for Transportation Sector Control Scenario Activity Projection.

Data Item	**Model**	**Source**
Household total, population, household shares by four attributes (location, income, age of head, and household size).	IPF	Statistical Abstract of the United States, editions 96th, 98th, 103rd, 104th, 108th, and 113th.
Household shares by number of drivers.	IPF	Statistical Abstracts and FHWA Highway Statistics provided total drivers. The *with CAA* distribution of households trended.
Personal and Disposable income.	VOP	J/W model output and Statistical Abstracts.
Vehicle fleet on-road fuel economy.	VOP DVSAM	FHWA Highway Statistics.
Fuel Prices	VOP DVSAM	Energy Information Administration's (EIA) Annual Energy Review.
Vehicle Price	DVSAM	Ward's Automotive Yearbooks 1975-1983, Automotive News Market Data Book 1985.

IPF - Iterative Proportional Fitting
VOP - Vehicle Ownership Projection
DVSAM - Disaggregate Vehicle Stock Allocation Model
FHWA - Federal Highway Administration
EIA - Energy Information Administration

Table B-5. Distribution of Households by Demographic Attributes for Control Scenario.

Household (Million)	63.4	71.1	80.8	86.8	93.3
Population (Million)	204.0	215.5	227.2	237.9	249.5
Attribute	**Household Percentage, by Year**				
	1970	1975	1980	1985	1990
Location					
Central City	33.2	32.0	31.9	31.6	31.4
Suburbs	33.6	36.0	37.0	38.1	38.3
Rural	33.2	32.0	31.1	30.3	30.3
Income (1990 $)*					
<$13,000	25.9	26.5	26.6	25.9	25.5
$13,000 - $33,000	34.0	37.2	37.4	37.7	38.0
$33,000 - $52,500	27.6	22.7	22.4	22.2	22.2
>$52,500	12.5	13.6	13.6	14.2	14.3
Age of Householder (YR)					
<35	25.4	29.1	31.1	29.3	27.4
35 - 44	18.6	16.7	17.3	20.1	22.1
45 - 64	36.3	34.0	31.2	29.6	29.0
> = 65	19.7	20.2	20.4	21.0	21.5
Household Size					
1	17.2	19.5	22.7	23.7	24.6
2	29.0	30.7	31.3	31.6	32.2
3 - 4	33.0	33.0	33.2	33.5	32.8
> = 5	20.8	16.8	12.8	11.2	10.4
Licensed Drivers					
0	9.1	8.5	8.1	7.2	6.6
1	27.8	27.3	27.0	26.2	26.0
2	48.1	49.2	50.5	52.5	53.5
> = 3	15.0	15.0	14.4	14.1	13.9

Note: *Approximated to 1990 dollars.

Table B-6. Economic and Vehicle Usage Data for Vehicle Ownership Projection – Control Scenario.

Year	Disposable Income per Capita (84 $)	Fuel Price (84 $)/Gallon	Miles/Gallon	VMT/Vehicle
1970	7,597	0.92	13.5	10,143
1971	7,769	0.88	13.5	10,246
1972	7,990	0.84	13.4	10,350
1973	8,436	0.84	13.3	10,184
1974	8,270	1.06	13.4	9,563
1975	8,340	1.03	13.5	9,729
1976	8,553	1.02	13.5	9,833
1977	8,742	1.01	13.8	9,936
1978	9,070	0.97	14.0	10,143
1979	9,154	1.21	14.4	9,522
1980	9,052	1.53	15.5	9,212
1981	9,093	1.55	15.9	9,212
1982	9,050	1.38	16.7	9,419
1983	9,239	1.27	17.1	9,419
1984	9,691	1.20	17.8	9,550
1985	9,881	1.09	18.2	9,568
1986	10,139	0.88	18.3	9,672
1987	10,174	0.88	19.2	10,090
1988	10,564	0.86	19.9	10,100
1989	10,713	0.90	20.3	9,819
1990	10,903	1.00	20.8	9,780

Table B-7. Control Scenario Personal Characteristics.*

	1975			1980		
Vehicle Type and Size (Seats)	**Curb Weight (lb)**	**Engine Power (hp)**	**Fuel Economy (mpg)**	**Curb Weight (lb)**	**Engine Power (hp)**	**Fuel Economy (mpg)**
Automobile						
Small (2-4)	2,770	91	17.2	2,535	83	19.6
Compact (4)	3,625	115	14.6	3,335	105	16.9
Mid-size (5)	4,140	128	13.3	3,730	116	15.1
Large (6)	4,900	155	12.2	4,840	153	13.3
Light truck						
Std. truck	4,530	141	11.2	4,455	143	12.6
Compact	3,745	108	14.2	3,580	99	15.9
Std. Van/Std. Utility (11-15)	5,010	145	9.9	4,975	144	11.4
Minivan/Small Utility (7-8)						

	1985			1990		
Vehicle Type and Size (Seats)	**Curb Weight (lb)**	**Engine Power (hp)**	**Fuel Economy (mpg)**	**Curb Weight (lb)**	**Engine Power (hp)**	**Fuel Economy (mpg)**
Automobile						
Small (2-4)	2,225	75	22.7	2,135	75	24.9
Compact (4)	2,775	90	19.3	2,595	90	22.0
Mid-size (5)	3,180	108	16.8	3,050	108	19.5
Large (6)	3,975	135	14.6	3,705	130	17.1
Light truck						
Std. truck	4,160	132	13.1	4,000	128	14.1
Compact	3,495	90	17.2	3,360	90	18.9
Std. Van/Std. Utility (11-15)	4,920	142	12.4	4,765	138	12.9
Minivan/Small Utility (7-8)	4,125	101	16.7	3,910	108	18.2

Note: *Average for all vehicles of each type and size.

Table B-8. Distribution of Households by Income Class for No-control Scenario.

	Household Shares (%), by Year			
Attribute	**1975**	**1980**	**1985**	**1990**
Income (1990 $)*				
<$13,000	26.3	26.2	25.3	24.7
$13,000-33,000	37.3	37.6	38.4	38.4
$33,000-52,000	22.8	22.6	22.0	22.6
>$52,000	13.6	13.6	14.3	14.3

Note: *Approximated to 1990 dollars.

No-control Scenario Emissions

The control scenario data were modified to reflect no-control scenario emissions using economic changes predicted by the J/W model, EPA, and ANL. The J/W model predicted a slight loss of employment and drop in GNP in terms of nominal dollars. However, the lower rate of inflation coincided with a real GNP rise. ANL's information from the model did not include any indexes for converting nominal income to real income. ANL assumed real income changes to be similar to those of real GNP and modified household shares by income classes accordingly. The model also predicted a slight drop in refined petroleum price beginning in 1973. The predicted drop was the largest (5.35 percent) in 1973, reached the lowest level (2.16 percent) in 1984, then increased to a second peak (3.44 percent) in 1988, and dropped again from 1989 to 1990. Since these changes were inconsistent with historical patterns of leaded and unleaded gasoline price change, ANL developed an estimate of changes in fuel price resulting from the cost of removal of lead from gasoline and other infrastructure costs involved with distributing a new grade of fuel. Subsequently, EPA provided a set of fuel costs for use in the analysis. Both ANL and EPA fuel prices followed a similar pattern, although their magnitudes differed. The no-control scenario was analyzed with EPA fuel prices. ANL also established a relationship with cost of regulation/emission control technology, and the effect of costs on vehicle price and fuel economy directly from the EPA publication *Cost of A Clean Environment* (EPA, 1990). These changes were used in the analysis.

The IPF model was executed for target years 1975, 1980, 1985, and 1990 using a set of revised household shares by income class. Table B-8 shows the revised shares. Comparing Table B-8 no-control scenario shares with those in Table B-5 for the control scenario, there seems to be a slight shift away from travel by the lowest income group and toward the middle income groups.

The vehicle ownership projection model was executed for the above four target years using the data listed in Table B-9. Changes in fleet characteristics are summarized in Table B-10.

Table B-9. Economic and Vehicle Usage Data for Vehicle Ownership Projection – No-control Scenario.

Year	Disposable Income per Capita (84 $)	Fuel Price (84 $)/Gallon	Miles/ Gallon	VMT/Vehicle
1970	7,597	0.91	13.5	10,143
1971	7,769	0.88	13.5	10,247
1972	7,990	0.83	13.4	10,353
1973	8,463	0.84	13.3	10,189
1974	8,297	1.06	13.4	9,569
1975	8,406	1.02	13.5	9,736
1976	8,600	1.01	13.5	9,854
1977	8,795	1.01	13.8	9,963
1978	9,126	0.96	14.0	10,174
1979	9,216	1.19	14.4	9,557
1980	9,114	1.51	15.5	9,234
1981	9,158	1.53	16.0	9,234
1982	9,116	1.36	16.8	9,447
1983	9,312	1.25	17.2	9,450
1984	9,775	1.18	17.9	9,582
1985	9,976	1.06	18.3	9,607
1986	10,244	0.84	18.4	9,738
1987	10,282	0.86	19.4	10,201
1988	10,676	0.83	20.1	10,214
1989	10,827	0.88	20.5	9,902
1990	11,019	0.97	21.0	9,849

Note: The effect of reductions in vehicle price and vehicle operating cost, and increases in fuel economy and horsepower were reflected in the menu of the vehicle choice model (DVSAM). Vehicle weight and seating capacity were kept unchanged from the *with CAA* run. Table IV-7 shows the changes in various vehicle attributes.

Table B-10. Percent Changes in Key Vehicle Characteristics Between the Control and No-control Scenarios.

	1975			1980		
Vehicle	**Price**	**mpg**	**HP**	**Price**	**mpg**	**HP**
Small Auto	-2.35	0.01	0.59	-2.76	0.22	1.81
Compact Auto	-2.35	0.01	0.59	-2.76	0.22	1.81
Midsize Auto	-2.35	0.01	0.59	-2.76	0.22	1.81
Large Auto	-2.35	0.01	0.59	-2.76	0.22	1.81
Small Truck	-1.30	0.01	0.59	-2.71	0.22	1.81
Std Truck	-1.30	0.01	0.59	-2.71	0.22	1.81
Std Van/Util	-1.30	0.01	0.59	-2.71	0.22	1.81
M Vn/Sm Utility						

	1985			1990		
Vehicle	**Price**	**mpg**	**HP**	**Price**	**mpg**	**HP**
Small Auto	-3.25	0.62	2.20	-2.94	0.95	2.77
Compact Auto	-3.25	0.62	2.20	-2.94	0.95	2.77
Midsize Auto	-3.25	0.62	2.20	-2.94	0.95	2.77
Large Auto	-3.25	0.62	2.20	-2.94	0.95	2.77
Small Truck	-2.53	0.62	2.20	-2.58	0.95	2.77
Std Truck	-2.53	0.62	2.20	-2.58	0.95	2.77
Std Van/Util	-2.53	0.62	2.20	-2.58	0.95	2.77
M Vn/Sm Utility	-2.53	0.62	2.20	-2.58	0.95	2.77

Note: *Average change for each vehicle size and type combination.

Utilities

The electric utility industry retrospective analysis was prepared using two different utility simulation models. ICF utilized its CEUM to estimate control and no-control scenario emissions for SO_2, TSP, and NO_x in each of the target years. ANL's ARGUS model was used to estimate electric utility CO and VOC emissions for the same period. This mix of modeling approaches was used because, while CEUM was determined to be a better tool for examining fuel shifts that were affected by the CAA than ARGUS, the CEUM model was not initially set-up to evaluate CO or VOC emissions. Although CEUM can be (and eventually was) configured to provide emission estimates for pollutants other than SO_2, NO_x, and PM, ARGUS was already configured to provide VOC and CO emissions. However, it should also be noted that VOC and CO emissions from utilities are quite low, as efficient fuel combustion reduces both pollutants. Thus, for this sector, the presence or absence of the CAA would not produce any different VOC or CO control techniques. VOC and CO emission rates for this sector differ primarily based on the fuel and boiler type. Therefore, a simpler modeling approach was judged to be acceptable and appropriate for these two pollutants. This chapter presents the methodology used to estimate utility emissions under the control and no-control scenario using the CEUM and ARGUS models. The method used by Abt Associates to estimate lead emissions from utilities is also presented.

Overview of Approach

The CEUM model uses industry capacity data and specific unit-by-unit characteristics, operating costs data, electricity demand estimates under the control and no-control scenario, and historical fuel prices to estimate SO_2, TSP, and NO_x emissions for 1980, 1985, and 1990. Changes in electric utility emissions, costs, and regional coal production were developed using ICF's CEUM with a calibration to historical electricity generation, fuel use, and emissions. The ARGUS model, which was used by ANL to estimate utility VOC and CO emissions, is driven by operating costs, industry capacity and generation data, demand for coal, and unit-level operating characteristics. The J/W model is used to incorporate predicted changes in electricity demand under the no-control scenario. Finally, Abt Associates relied upon energy use data, the *Trends* data base, and the Interim 1990 Inventory to calculate utility lead emissions based on coal consumption. The approaches used by each of these three contractors are discussed individually in the following sections.

Establishment of Control Scenario Emissions

A common feature of the approaches taken by ICF and ANL was to identify conditions that are inputs to the CEUM and ARGUS models, respectively, in the control scenario. Later in the analysis, these variables were revised to reflect no-control scenario conditions. The next section discusses the specific assumptions used in the CEUM analysis.

Key Assumptions in the Development of the ICF Analysis

At EPA's direction, ICF made several assumptions in conducting this analysis for purposes of consistency with other ongoing EPA efforts assessing the effects of the CAA. These include the macroeconomic assumptions regarding the effects of the CAA on economic growth, or more specifically, electricity demand, developed from other EPA commissioned efforts. Each is described briefly below.

Pollution Control Equipment Costs

Only limited actual data were available for this analysis on the historical capital and operating costs of pollution control equipment. Accordingly, for this analysis, the actual capital and operating costs of scrubbers were estimated using EPA scrubber cost assumptions adjusted to reflect actual data from a survey of scrubbed power plants with scrubbers installed during the 1970s and early 1980s. For those power plants with actual survey data, actual capital costs were used. For other pre-1985 scrubbers, ICF relied on the average costs from the survey data. For particulate control equipment (primarily electrostatic precipitators, or ESPs), costs were estimated based on limited actual data, and a 1980 Electric Power Research Institute (EPRI) study of ESP and baghouse costs. Based on this information, ESPs were estimated to cost an average of $50 per kilowatt (in 1991 dollars). The development of more detailed data on actual power plant pollution control costs was beyond the scope of ICF's analysis. ICF concluded that such an effort would not significantly change the national or regional cost estimates developed by its approach.

Electricity Demand and Fuel Prices

Consistent with other EPA ongoing analyses, ICF assumed that the CAA resulted in a reduction in electricity demand of 3.27 percent in 1980, 2.77 percent in 1985, and 2.97 percent in 1990. Also consistent with these studies, ICF assumed that natural gas prices and oil prices would not be affected by the CAA. Coal prices were estimated to change in line with increases and decreases in demand for specific coal supplies (and consistent with ICF's detailed modeling of coal supply and demand). The average prices of all residual oils consumed were also estimated to change due to a greater use of more expensive lower sulfur residual oils under the CAA.

Coal, Nuclear, Hydro, and Oil/Gas Capacity

At EPA's direction, ICF's approach was based on the assumption that no changes in the amount of nuclear, coal, hydro, or oil/gas stream or combined cycle capacity would be built or in place in 1980, 1985, or 1990. Given that the driving factors associated with the actual decisions to build new baseload capacity were not based solely on economics but entailed financial, regulatory, and political factors as well, the actual effect of the CAA on these build decisions is very uncertain. To the extent that more coal-fired power plants would be built and fewer oil/gas-fired power plants constructed, the actual emissions reductions associated with the CAA would be greater than those estimated by ICF, while the estimated costs of the CAA would be greater (because fewer, lower-cost, coal-fired power plants would be on line under the CAA). However, the CAA had virtually no effect on the costs of constructing new coal-fired power plants that came on line prior to about 1975 and a relatively moderate cost effect on coal-fired power plants that came on line through the early 1980s (since these power plants were not required to install scrubbers). Since a large majority of coal-fired power plant capacity came on line prior to 1975, ICF concluded that the effect of the CAA on the amount of total coal-fired capacity was not expected to be very large.

Natural Gas Consumption

The analysis assumed that the amount of natural gas consumed under the no-control scenario could not exceed the actual amount of consumption in 1980, 1985, and 1990. In part, because of natural gas price regulation and the oil price shocks of the 1970s, natural gas was often unavailable to electric utilities in the early 1980s. Since the CAA is relatively unrelated to the questions of supply availability and price regulation of natural gas, ICF assumed that no additional gas supplies would be available if the CAA had never been adopted. It is possible, however, that in the absence of the CAA, industrial and commercial users of natural gas would have used more oil or coal. To the extent that this would have occurred, there would have been more natural gas supplies available to the electric utility sector. This increase in supply would have resulted in an increase in the estimated costs of the CAA, and a corresponding decrease in the estimated emission reductions. ICF concluded, however, that this effect would not be very significant.

State and Local Environmental Regulations

At EPA's direction, ICF assumed that there would be no State and local emission limits or other emission control requirements under the no-control scenario. Accordingly, ICF assumed that there would be no SO_2, NO_x, or TSP emission limits under the no-control scenario and that all scrubbers, NO_x controls, and ESPs/baghouses (at coal-fired power plants) were installed as a result of the CAA. (The more limited amount of particulate control equipment installed at oil-fired plants was assumed to have been installed prior to the passage of the CAA.) In the case of particulate control equipment, some ESPs and other equipment were installed at coal plants prior to the 1970 CAA. To the extent that this is the case, the estimates of the costs of meeting the CAA have been overstated. ICF concluded, however, that the amount of such capacity was not substantial.

Retirement Age

The analysis assumed that unit retirement age was constant between the control and no-controls scenarios. Adoption of this assumption might bias the emission reduction estimates upward to the extent turnover rates of older (and presumably higher-emitting) units may be slower under the control scenarios, because more significant CAA control requirements focused on new units. However the vast majority of existing coal and oil capacity was built after 1950 and it is generally acknowledged that a relatively short technical plant lifetime would be about 40 years. As such, even if the no-control scenarios resulted in no life-extension activity, there would be virtually no effect over the 1970 to 1990 timeframe of the analysis.

ICF 1975 Control Scenario Emissions

The 1975 emissions under both scenarios were calculated differently than emissions in 1980, 1985, and 1990. In calculating or estimating 1975 SO_2 emissions for the control scenario (i.e., "actual" 1975), the weighted average emission rates at the State level, in the year 1975 were estimated, based on plant level average sulfur content of fuel deliveries from Federal Energy Regulatory Commission (FERC) Form 423 and assumed AP-42 sulfur retention in ash. These weighted average emission rates were then applied to actual State-level electric utility fuel consumption in the year 1975 (DOE, 1991). In the case of NO_x emissions, first, an estimate of Statewide NO_x emissions in the year 1975 was derived based on the use of the same NO_x emission rates, by fuel type, as developed for the 1980 no-control scenario modeling runs. These emission rates were specific to the fuel type (coal, oil, or natural gas). These Statewide NO_x emission rates or factors were then applied to actual fuel consumed by electric utilities in the year 1975, in order to obtain estimated "actual" 1975 emissions. As before, the fuel consumption at a State level was derived from the *State Energy Data Report* (DOE, 1991). ICF calculated the weighted average heat content (BTU/lb) by State from the 1975 FERC Form 423 data and used these figures with the TSP emission factors (lbs/ton) to derive emission rates by State (lbs/MMBTU). These emission rates were then applied to 1975 fuel consumption estimates obtained from the *State Energy Data Report*. For the control scenario 1975 estimates, ICF used the 1975 factors.

For the remaining target years, ICF used the results of CEUM runs that provided fuel consumption figures in 1980, 1985, and 1990, respectively. Emissions were then calculated using the appropriate emission factors for each year.

ARGUS Modeling Assumptions

The portion of the electric utility sector analysis conducted by ANL with the ARGUS model is described in this subsection. ARGUS contains four major components: BUILD, DISPATCH, the Emissions and Cost Model, and the Coal Supply and Transportation Model (CSTM). An overview of ARGUS can be found in Veselka *et al* (1990). Only the DISPATCH and CSTM modules were used for the present analysis. A brief description of the ARGUS components used in this analysis is found in the following subsections.

DISPATCH Module

The DISPATCH module contains a probabilistic production-cost model called the Investigation of Costs and Reliability in Utility Systems (ICARUS). This module calculates reliability and cost information for a utility system. ICARUS represents detailed, unit-by-unit operating characteristics such as fuel cost, forced outage rate, scheduled maintenance, heat rate, and fixed and variable operating and maintenance (O&M) costs. These components are used to efficiently compute system reliability (such as loss-of-load probability and unserved energy) and production costs.

The input data required by ICARUS include monthly load duration curves, annual peak demands, and, for both new and existing units, unit sizes, capital costs, fixed and variable O&M costs, fuel types and costs, heat rates, scheduled maintenance, and equivalent forced outage rates. The output from ICARUS includes annual summaries of capacity, generation, cost, and reliability for the entire generating system.

CSTM Module

The CSTM module determines the least-cost combination, on a per BTU basis, of coal supply sources and transportation routes for each demand source. First, it estimates coal market prices based on regional demands for coal from all economic sectors. To generate market prices, CSTM estimates regional coal production patterns and coal transportation routes. The CSTM input data are grouped into three major categories: demand, supply, and transportation. CSTM uses supply curves from the Resource Allocation and Mine Costing (RAMC) Model (DOE, 1982). Every region has a separate curve for one or more of the 60 different coal types that may be produced in that region. CSTM modifies the original RAMC supply curve by dividing the single RAMC curve into two curves, one representing deep mines and the other representing surface mines, but still uses the same ranges for heating values and mine prices that define the supply curves in RAMC. Prices fluctuate as a result of different mining methods, size of mining operations, reserve characteristics, and depletion effects.

The transportation data defines the network that connects 32 coal supply origins with 48 demand centers. Transportation cost is affected by distance, terrain, congestion, variable fuel costs, cost escalators for fuels and facility upgrades, and competition. CSTM first computes the production cost for each coal supply region and coal type. It then matches supply sources with transportation routes to find the lowest delivered costs.

Coal demand for a particular region is based on the amount, geographic region, economic sector, and range of coal types. There are 44 domestic demand regions. CSTM allows demand to be met by one, or a combination of, different supply regions.

The ARGUS input data for existing units are based on the Argonne Power Plant Inventory (APPI). APPI is a data base of operating and planned generating units in the United States that was current through 1988 at the time of ANL's analysis. This data base is updated annually based on information in the regional North American Electric Reliability Council (NERC) reports, reports from the Energy Information Administration (EIA), and other sources. Unit operating characteristics (fixed O&M, variable O&M, heat rate, forced outage rate, and scheduled maintenance) are based on regional data as defined in the EPRI report on regional systems and other historic data (EPRI, 1981).

ANL used the 1988 inventory to generate a 1990 inventory. The 1990 inventory was then used to generate a separate unit inventory for the target years 1975, 1980 and 1985. The target year inventories were generated by removing units whose on-line year was greater than the target year, from their respective inventory. The regional capacity totals in these preliminary inventories were tabulated by major fuel category (nuclear, coal, oil and gas steam) and compared to the regional historic NERC totals. This review identified capacity differences, especially in 1975 and 1980 inventories. The original plan was to add phantom units to match the regional historic totals. However, based on the need for State-level emissions, it was decided that a more thorough review of the unit inventories was required.

ANL's detailed review included an examination of the nuclear and coal units greater than 100 megawatt equivalent (MWe) in each target year. Missing units, with the appropriate unit size and State code, were added so that the regional totals were comparable. The availability of coal units was based on the on-line year of the unit as reported in the EIA report *Inventory of Power Plants in the United States* (DOE, 1986). The coal units were also checked against the EIA Cost and Quality Report (EIA, 1985) to verify the existence of flue gas desulfurization (FGD) systems in each of the target years. The nuclear unit inventories were verified with the EIA report *An Analysis of Nuclear Power Plant Operating Costs* (DOE, 1988). The review also included oil and gas steam units greater than 100 MWe. The total capacity of the oil and gas steam units were compared because many units switched primary fuel from oil to gas during the relevant time period. The oil and gas units were compared to historic inventories based on information provided by Applied Economic Research. In addition to thermal generation, the hydro and exchange energy was reviewed. For each target year, the hydro generation and firm purchase and sale capacity data was adjusted to reflect the historic levels. These two components, hydro and firm purchase and sales, are accounted for first in the loading order. If these variables are overestimated, there will be less generation from coal units. Likewise, if they are underestimated, there will be too much coal generation. The hydro and firm purchases and sales can vary significantly from year to year because of weather conditions and other variables. Therefore, it was important that they be accurately represented.

No-control Scenario Emissions

In order to calculate utility emissions under the no-control scenario, inputs to both the CEUM and ARGUS models were adjusted to reflect no-control scenario conditions. The changes made to each model's base year input files are discussed separately in the following sections.

ICF Estimates of SO_2, TSP, and NO_x Emissions in the No-control Scenario

As described earlier, ICF utilized a different methodology to calculate 1975 emission estimates. Rather than relying on the use of detailed modeling runs, ICF based the 1975 emission estimation on historic fuel consumption and sulfur content data in 1975. This subsection first outlines the process used to calculate no-control scenario emissions in 1975 and then presents the methods used for the remaining target years.

1975 Utility SO_2, NO_x, and TSP Emissions

To develop State-level no-control scenario utility SO_2 emissions, ICF developed no-control scenario SO_2 emission rates. A reasonable surrogate for these emission rates is SO_2 rates just prior to the implementation of the SIPs under the CAA. ICF developed 1972 rates (based on the earliest year available for FERC Form 423) and compared these with 1975 rates. In each State, the greater of 1972 or 1975 rates was used in the calculation of SO_2 emissions in the absence of the CAA. To develop State-level no-control scenario SO_2 emissions, no-control scenario fuel consumption data were needed. ICF assumed that the demand for electricity in 1975 would be 2.73 percent higher than the actual energy sales in 1975. This assumption is identical to the no-control scenario electricity demand projections derived from the J/W projections. For the purpose of this analysis, it was further assumed that this increment in demand would have been met in 1975 from the oil and coal-fired plants in each State. The increase in consumption of these fuels was assumed to be in the same proportion as their share in the 1975 total energy mix for electricity generation in that State. It was assumed that the generation of nuclear, gas-fired, and other electricity generation would not change. A sensitivity case without an assumed electricity demand change was also calculated. (The sensitivity analysis results are presented later in this appendix.)

For NO_x emissions under the no-control scenario, it was also assumed that the 1975 electricity sales would have been 2.73 percent higher than was the case in 1975. No-control scenario TSP emissions in 1975 were based on national emission rate numbers from EPA that were converted to pounds per million BTU using the average energy content of fuels in each State. No-control scenario TSP emissions were calculated based on 1970 emission factors (Braine, Kohli, and Kim, 1993).

1980, 1985, and 1990 Utility Emissions

For 1980, 1985, and 1990, ICF calculated no-control scenario emissions based on fuel consumption figures from the CEUM runs, and 1970 emission factors from EPA.

Electric utility SO_2 emission estimates are approximately 10 million tons (or about 38 percent) lower by 1990 under the control scenario than under the no-control scenario. Most of this estimated difference results from the imposition of emission limits at existing power plants through the SIPs under the 1970 CAA. Most of these SIPs were effective by 1980 (with some not fully effective until 1985). Most of the additional reductions that occurred during the 1980s were the result of the electric utility NSPS, which required the installation of 70 to 90 percent SO_2 removal control equipment.

By contrast, electric utility NO_x emission estimates under the control scenario are only about 1.2 million tons, or 14 percent, lower than under the no-control scenario by 1990. This occurs because, under the implementation of the 1970 CAA, only a few existing power plants were subject to NO_x emission limits. Virtually all of the estimated reductions are the result of NO_x NSPS, which generally required moderate reductions at power plants relative to uncontrolled levels. In addition, electricity demand is estimated to be about 3 percent lower under the control scenario. This decrease reduces the utilization of existing power plants and also contributes to lower NO_x emissions (and other pollutants as well).

Electric utility annualized costs (levelized capital, fuel, and O&M) are estimated to be \$0.2 billion lower in 1980, \$1.5 billion higher in 1985, and \$1.9 billion higher in 1990 under the control scenario. Note, however, that this reflects the effects of two offsetting factors: (1) the *higher* utility compliance costs associated with using lower sulfur fuels, and the increased O&M and capital costs associated with scrubbers and particulate control equipment; and (2) *lower* utility generating costs (fuel, operating and capital costs) associated with lower electricity demand requirements. In 1980, the increase in fuel costs due to higher generation requirements (under the no-control scenario), was larger than the decrease in capital and O&M costs and thus yielded a cost increase over the control case.

However, lower electricity demand for the utility sector would translate into higher costs in other sectors (as electricity substitutes are used). This effect was captured to some extent by the original J/W macroeconomic modeling conducted for the present analysis.

Average levelized U.S. electricity rate estimates are approximately 3 percent higher under the control scenario during the 1980s. Note that year by year, electric utility revenue requirements and capital expenditures (not estimated by ICF) would be estimated to have increased by a greater percentage particularly in the 1970s and early 1980s as incremental capital expenditures for scrubbers and ESPs were brought into the rate base.

Significant shifts in regional coal production are estimated to have occurred between the control and no-control scenarios. High sulfur coal producing regions such as Northern Appalachia and the Midwest/ Central West are estimated to have lower production under the control scenario, while lower sulfur coal producing regions such as Central and Southern Appalachia are estimated to have higher coal production.[12]

ARGUS No-control Scenario

Regional fuel prices, for the thermal units, were based on historic information from the EIA Form 423 data for the year 1977, 1980 and 1985. The 1977 data was used for 1975. Fixed and variable O&M costs were adjusted from the 1988 level, and all cost data were converted to 1985 dollars.

The load data were based on regional historic NERC data for each of the target years. The shapes of the monthly load duration curves are the result of modifications based on the data in the EPRI report on regional systems (EPRI, 1981). The shapes were modified to match the projected 1988 monthly load factors for the NERC regions. These load shapes were held constant for all years.

The actual peak-loads were selected from historic information and used with the existing load duration curves. The system was dispatched so that the calculated generation could be compared with historic data. Discrepancies were resolved by adjusting the peak load so that the annual generation was on target. This procedure was repeated for each of the target years.

The electric utilities were expected to have an increase in generation as identified by the J/W data. Table B-11 identifies the increase in national level generation by year. The national level increase in generation was applied to each power pool.

Table B-11. J/W Estimates of Percentage Increases in National Electricity Generation Under No-control Scenario.

Year	Percentage Increase
1975	2.7%
1980	3.3%
1985	2.8%
1990	3.0%

In addition to load changes, coal units with FGD equipment were modified. These units had their FGD equipment removed along with a 3 percent decrease in heat rate, a 2 percentage point decrease in forced outage rate, and a 50 percent decrease in their fixed and variable O&M costs. These changes were incorporated into the ARGUS model for each of the target years. Model runs were then conducted to arrive at estimates of VOC and CO emissions in the no-control scenario.

Estimation of Lead Emissions from Utilities

In order to estimate lead emissions from electric utilities in each of the target years, data from three different sources were used. Energy use data for the control and no-control scenarios were obtained from the national coal use estimates prepared for the section 812 analysis by ICF (Braine and Kim, 1993). The *Trends* data base provided emission factors and control efficiencies, and the Interim 1990 Inventory identified utility characteristics. The ICF data bases provided the amount of coal consumed for both the control and no-control scenarios in each of the target years. A correspondence between the Interim Inventory and the ICF data base was achieved through the plant name variable. Using emission factors for lead and control efficiencies for electric utilities, estimates of lead emissions per plant per year were calculated. These factors were obtained from the *Trends* data base. It was assumed that pollution control on coal-burning power plants under the no-control scenario would be the same as the pollution control level in 1970. Therefore, the control efficiency from 1970 is used as the basis for the no-control case.

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[12] At EPA's direction, ICF's analysis did not estimate the effect of shifts in non-utility coal consumption on regional coal production, nor did it consider the possibility that fewer new coal powerplants might have been built due to the CAA as discussed earlier. Both of these factors could result in a greater estimated change in total U.S. coal production than estimated herein although the difference is not likely to be very significant.

CEUM Sensitivity Case

In addition to comparing actual (control scenario) historical costs and emissions with the higher electricity demand under the no-control scenario, ICF also evaluated emissions in a sensitivity case without the CAA (i.e., under the no-control scenario) with the same electricity demand (versus the no-control scenario with higher demand). The purpose of this sensitivity analysis was to isolate the incremental electric utility compliance costs and reductions in emissions associated with the CAA from the lower resulting generation costs and emissions due to lower estimated electricity demand under the CAA. The incremental effects of the CAA when compared with this case indicate:

- Estimated reductions in emissions due to the CAA are somewhat lower if measured against the sensitivity case without the CAA with the same electricity demand than the emissions without the CAA with lower demand. This occurs because lower electricity demand under the no-control scenario sensitivity results in lower utilization of existing coal and oil plants which, in turn, results in lower emissions. As noted above, in some sense, the changes in emissions represent the effects of electric utility compliance actions under the CAA, absent the effect of lower resultant demand for electricity.

- When measured against the sensitivity case without the CAA (with the same electricity demand), electric utility annualized costs are estimated to have increased by about $5 to $6 billion during the 1980 to 1990 period. This reflects the following cost factors: (1) higher annualized capital costs associated primarily with scrubbers and ESPs installed by electric utilities to comply with the CAA; (2) higher O&M costs associated with the additional air pollution control equipment; and (3) higher fuel costs associated with using lower sulfur coal and oil in order to meet the emission limit requirements of the CAA.

Commercial/Residential

The Commercial and Residential Simulation System (CRESS) model was developed by ANL as part of the Emissions and Control Costs Integrated Model Set and used in the NAPAP assessment (*Methods for Modeling Future Emissions and Control Costs, State of Science and Technology, Report 26*) (McDonald and South, 1984). CRESS is designed to project emissions for five pollutants: SO_x, NO_x, VOC, TSP, and CO. The CRESS output is aggregated into residential and commercial subsectors related to both economic activity and fuel use. The introductory material provided in this appendix about CRESS describes the base year as being 1985. It appears in this way because CRESS was originally developed to operate using the 1985 NAPAP Emission Inventory as its base year data set. For the five pollutants reported by CRESS, emission estimates are provided for the following sectors:

- ♦ Commercial/institutional
  - coal, including point and area categories of anthracite and bituminous boilers;
  - liquid fuel, including boiler and space heating uses of residual, distillate, LPG, and other fuels;
  - natural gas boilers, space heaters, and internal combustion engines;
  - wood used in boilers and space heaters; and
  - other mixed or unclassified fuel use.
- ♦ Residential
  - coal, including area sources of anthracite and bituminous;
  - liquid fuel, composed of distillate and residual oil;
  - natural gas; and
  - wood.
- ♦ Miscellaneous
  - waste disposal, incineration, and open burning; and
  - other, including forest fires, managed and agricultural burning, structural fires, cutback asphalt paving, and internal combustion engine testing.

In addition, VOC emissions are projected for these source categories:

- ♦ Service stations and gasoline marketing;
- ♦ Dry-cleaning point and area sources; and

- Other solvents, including architectural surface coating, auto-body refinishing, and consumer/commercial solvent use.

This section describes the use of CRESS to estimate control and no-control scenario emissions from the commercial/residential sector.

Control Scenario Emissions

For the NAPAP assessment, 1985 CRESS output corresponded to the 1985 NAPAP Inventory (EPA, 1989), which served as the benchmark for any projections. The design of CRESS is such that emissions by NAPAP SCC are input for each State, then projected to future years by scaling them to economic data such as energy demand. In estimating emissions, differences in emission controls associated with new, replacement, and existing equipment are taken into account where such differences are considered significant. The basic modeling approach is shown in the following equation:

$$Q_{t,b} = \left(\frac{Q_0}{E_0}\right), b \times \left(\frac{D_T}{D_0}\right) \times \sum^{j} (f_{t,j} \times E_{t,j}) \qquad (3)$$

where:

Q = emissions in year t or the base year, year 0

E = emission factor for the source category b in the base year, or for a subcategory j subject to controls in year t (this takes into account changes in emission rates that may occur as a result of emission regulations or technology changes)

D = driver data indicating activity levels in the base and future years

f = fraction of total activity in year t differentially affected by emission controls

The calculations are carried out in two subroutines, one for SO_2, NO_x, TSP and CO, and one for VOC.

Typically SO_2, NO_x, TSP, and CO emissions are projected by multiplying the 1985 NAPAP SCC data or base year data by the ratio of the driver data (activity level) value in the projection year to its value in the base year. Because there are few controls on SO_x or NO_x emissions from the sources covered by CRESS, projected emissions for most sectors are proportional to the expected activity levels. Thus,

$$Q_t = Q_0 \times \left(\frac{D_t}{D_0}\right) \qquad (4)$$

There are a few source types, such as commercial/institutional boilers, for which emission controls are mandated. These are modeled by multiplying the 1985 emission data by the ratio of the controlled emission factor to the base-year emission factor. Emission factors for each source type are weighted by the proportion of base year activity in each subsector to which controls are expected to apply.

$$Q_{t,b} = Q_0 \left[g_{t,b} + \left(\frac{E_{t,n}}{E_{0,b}}\right) \times (g_{t,r} + g_{t,n})\right] \qquad (5)$$

where:

g = the fraction of base-year activity accounted for by existing source b, replacement source r, or new source n in year t

The effective emission factor (Et,n) for the sector is calculated by weighing the portions of sectoral emissions subject to NSPS controls and those likely to continue at existing levels. An appropriate Internal Revenue Service-based rate at which new equipment replaces existing sources is applied to each sector in the model. This is done to estimate how emissions might change as older sources are retired and replaced by new sources that emit at lower rates.

The SO_x/NO_x/TSP/CO subroutine varies in new and replacement emission-source fractions subject to NSPS controls. These fractions are applied to the emission-source replacement rates. In addition, ratios for new source emission factors are varied by State. However, emission ratios for any pollutant/source type combination do not vary over the projection period.

The VOC estimation methodology is similar, but allows variation in emission factors over time. Emission ratios are calculated from files of replacement and existing source emission factors weighted by the replacement rate for each sector and new source factors by State. These are input for each 5-year projection interval. For most source categories, VOC controls are not envisioned, and the 1985 NAPAP emissions for the category are simply scaled proportionally to changes in the driver (activity level) data.

For sources to which controls apply, a variation on the following equation is employed:

$$Q_{t,b} = (\frac{Q_0}{E_0}, b) \times (E_{t,b} + g_{t,n} \times E_{t,n})] \qquad (6)$$

In equation 6, the emission factors for new and existing sources are effectively weighted by the proportion of total activity in year t to which controls apply.

In using CRESS for the CAA retrospective analysis, the base year was 1975. CRESS requires emissions information by State and NAPAP source category as input. Since detailed information on emission levels for 1975 by NAPAP source category were not available, the data were developed from a combination of sources. The procedure for calculating 1975 emissions based on the 1985 NAPAP inventory is described below. The emissions module uses these initial values in conjunction with activity estimates to project control and no-control scenario emissions.

Emissions Data

Since the starting point for the analysis was 1975, emissions data by State and SCC for SO_2, NO_x, VOC, TSP, and CO were required. Available emissions information for this year was not at the level of detail needed by CRESS. The 1985 NAPAP Inventory, which contains the necessary level of detail, in conjunction with information from EPA's *National Air Pollutant Emission Estimates, 1940-1990* (*Trends*) and ANL's MSCET, was used to construct an emissions inventory for 1975. The model then uses these emissions as a benchmark for the analysis.

The method for constructing the 1975 emissions data base was consistent for all pollutants; however, two different sources of emissions data were necessary in order to obtain time series information on all pollutants. MSCET contains monthly State-level emission estimates from 1975 to 1985 by emission source group for SO_2, NO_x, and VOC. Therefore, MSCET information was used for SO_2, NO_x, and VOC, while *Trends* data were used for TSP and CO. Emission source groups from MSCET were matched with 1985 NAPAP Inventory SCCs. The MSCET methodology is benchmarked to the 1985 NAPAP Inventory and uses time series information from *Trends* in conjunction with activity information to estimate State-level emissions for SO_2, NO_x, and VOC. Although the level of detail contained in the NAPAP Inventory could not be preserved because of the aggregation needed to match with MSCET emissions sources, MSCET provided the State-level spatial detail required by CRESS.

Once the 1985 emissions by SCC and State from the 1985 NAPAP Inventory were matched with emission source groups and States from the MSCET data base, an estimate of 1975 emissions was computed by multiplying the 1985 NAPAP Inventory emissions value by the ratio of 1975 MSCET emissions to 1985 MSCET emissions. Ratios were computed and applied for each combination of State, pollutant, and MSCET emission source group.

This method of constructing an emissions inventory for 1975 utilizes the State estimates from MSCET, thus capturing the spatial shifts that occurred over the analysis period. It is assumed that NAPAP provides the most reliable point and area source information in terms of the level of 1985 emissions (which is also the assumption of the MSCET methodology). Note that if there were a 1-to-1 correspondence between MSCET and NAPAP, this method would be equivalent to using the MSCET methodology directly for constructing 1975 emission levels.

A similar method was used for TSP and CO, but since these pollutants are not included in MSCET, the *Trends* ratio of 1975 to 1985 emissions for these two pollutants was used. Thus, for TSP and CO, all States were assumed to have experienced the same change in emissions as indicated by the national figures.

It should be noted that in addition to the loss in spatial detail, the *Trends* source groups generally spanned several NAPAP source categories. The strength in the *Trends* information is the consistency of emissions estimates over time. It is considered to be the most reliable data for tracking changes in emissions over the time period of the analysis, and was therefore chosen for developing 1975 estimates for TSP and CO.

The 15 source categories reported in *Trends* were matched with those in the 1985 NAPAP Inventory. The ratios of 1975 emissions to 1985 emissions by source category that were applied to the 1985 NAPAP emissions data are shown in B-12. The 1975 emis-

Table B-12. *Trends* Source Categories and (1975 to 1985) Scaling Factors for TSP and CO.

***Trends* Source Category**	**TSP***	**CO***
Commercial/Institutional Fuel Combustion:		
Coal	2.11	0.59
Natural Gas	1.00	0.91
Fuel Oil	2.35	1.43
Other	1.83	0.67
Residential Fuel Combustion:		
Coal	1.33	1.47
Natural Gas	1.17	1.00
Fuel Oil	1.11	1.76
Wood	0.49	0.49
Miscellaneous: Forest Fires	0.67	0.62
Solid Waste Disposal:		
Incineration	3.00	0.64
Open Burning	1.50	1.44
Miscellaneous Other Burning	1.00	1.33
Industrial Processes: Paving	2.71	0.56
Asphalt Paving and Roofing	2.71	0.56
Miscellaneous Other	1.83	0.67

Note: *These values are the ratios of 1985 *Trends* emissions to 1975 *Trends* emissions for each source category. For example, the commercial/ institutional fuel combustion: coal emission ratio of 2.11 is computed as the ratio of the 1975 TSP emissions of 40 gigagrams per year to the corresponding 1985 emissions of 19 gigagrams per year.

sions data estimated from the above procedure served as the benchmark and initial value for the CRESS emissions module for both scenarios.

CAA regulation of commercial/ residential emissions was limited and largely confined to fuel combustion sources (SO_2, NO_x, TSP), gasoline marketing (VOC), dry cleaning (VOC), and surface coating (VOC). NSPS regulations of small (over 29 MW capacity) fuel combustors were promulgated in 1984 and 1986. For purposes of emissions calculations, the stipulated NSPS for SO_2, NO_x, and TSP were incorporated into the control scenario for 1985 and 1990. Emission rates for source categories subject to VOC regulation were similarly adjusted.

Energy Data

Nearly 75 percent of the source categories in CRESS use energy consumption by State and sector as the driver for the emissions calculation. State-level energy consumption statistics are published by EIA in *State Energy Data Report, Consumption Estimates, 1960-1989,* and are electronically available as part of the State Energy Data System (SEDS) (DOE, 1991). The SEDS data base contains annual energy consumption estimates by sector for the various end-use sectors: residential, commercial, industrial and transportation, and electric utilities.

Seven fuel-type categories are used in CRESS: coal, distillate oil, residual oil, natural gas, liquid petroleum gas, wood, and electricity. The model assumes zero consumption of residual fuel oil in the residential sector and zero consumption of wood in the commercial sector. Energy consumption for each fuel-type was expressed in BTUs for purposes of model calculations. With the exception of wood consumption, all of the energy consumption statistics used in CRESS were obtained from SEDS.

Residential wood consumption estimates were derived from two data sources. State-level residential sector wood consumption estimates for 1975 and 1980 were obtained from *Estimates of U.S. Wood Energy Consumption from 1949 to 1981* (EIA, 1982). State-level wood consumption, however, was not available for 1985 and 1990, therefore, regional information from an alternative publication, *Estimates of U.S. Biofuels Consumption 1990* (EIA, 1990), was used to derive State-level residential wood use figures. Regional 1985 and 1990 wood consumption was distributed among States using 1981 State shares. All wood consumption figures were converted to BTU's using an average value of 17.2 million BTU per short ton.

Economic/Demographic Data

Emissions from slightly more than 25 percent of the CRESS source categories follow State-level economic and demographic activity variables. The demographic variables used by CRESS include State-level population, rural population, and forest acreage. State population is the activity indicator for six emissions source categories for SO_2, NO_x, TSP, and CO, and 13 VOC source categories. State population data were assembled from the SEDS data base. Rural population, which is the indicator of residential open burning activity, is computed as a fraction of total State population. Forest wildfires and managed open burning activity are related to 1977 State-level forest acreage. The demographic information is assumed to be invariant to CAA regulations and thus is the same in the control and no-control scenarios.

Car stock (or vehicle population), the driver variable for the auto body refinishing, is approximated by State motor vehicle registrations. *Highway Statistics*, an annual publication by the FHWA, was the source for data on State motor vehicle registrations. The three source categories connected with gasoline marketing are driven by State-level gasoline sales in gallons. State gasoline consumption was obtained from the SEDS data base. Housing starts and 10 percent of the existing housing stock were combined to form the activity indicator for architectural surface coating emissions. Housing data compiled by the U.S. Bureau of the Census were available in the *Statistical Abstract of the United States* (DOC, 1975; 1977; 1982; 1983; 1987; 1993). Regional-level data for 1975 was allocated to the States based on the 1980 State distribution.

No-control Scenario Emissions

Adjustments to control scenario emissions in each of the target years to reflect conditions under the no-control scenario were achieved through emission factors, energy input data, and economic/demographic data. The adjustments made to each of these variables to generate no-control scenario emissions are discussed individually in the following subsections.

Emissions Data

CAA regulation of the commercial/residential sector was minimal. For regulated source categories, emission factors were revised to reflect pre-regulation emission rates. Six commercial/residential source categories were regulated for VOC emissions: Service Stations Stage I Emissions, Service Stations Stage II Emissions, Dry Cleaning (perchloroethylene), Gasoline Marketed, Dry Cleaning (solvent), and Cutback Asphalt Paving. Commercial-Institutional boilers were regulated for SO_2 and TSP and internal combustion sources were regulated for NO_x emissions. All NSPS were removed for these sources to estimate no-control scenario emissions levels.

Energy Data

State-level energy demand for the residential and commercial sectors for the no-control scenario was estimated from the J/W model forecast. Final energy demand estimates for the household sector were calculated by an EPA contractor for the purposes of the no-control scenario analysis. State allocation of the national-level estimates was based on historic State shares, i.e., this assumes that there is no change in the distribution of energy demand across States as a result of removing regulations. In addition, the J/W model estimates an aggregate refined petroleum category and does not distinguish among liquid petroleum gas, distillate oil, and residual oil. The relative shares among these three categories of petroleum products remained constant between the control and no-control scenarios. The information on percentage change in energy demand by fuel type as provided by the J/W model is listed in Table B-13.

The differential for commercial sector final energy demand was calculated from the combination of four intermediate product flow categories from the J/W forecast. The National Income and Product Accounts (NIPA) for the commercial sector correspond to J/W SIC categories 32 through 35:

Table B-13. Percentage Change in Real Energy Demand by Households from Control to No-control Scenario.

Year	Coal	Refined Petroleum	Electric	Natural Gas
1975	1.48	4.76	3.62	2.42
1980	1.50	3.84	4.26	2.12
1985	1.98	3.90	3.88	2.41
1990	2.23	4.33	4.18	2.77

(32) Wholesale and Retail Trade;
(33) Finance, Insurance, and Real Estate;
(34) Other Services; and
(35) Government Services.

Percentage change information from the J/W forecast for energy cost shares, value of output, and energy prices was used to calculate the differential in commercial sector energy demand for the no-control scenario. The energy cost share is defined as the cost of energy input divided by the value of the output. In order to calculate the percentage change in commercial sector energy demand, the change in energy price was subtracted from the percentage change in energy cost, and added to the change in the value of output. Each of these variables was available from the J/W model results. This calculation was performed for each of the four energy types, and each of the four NIPA categories. The change in commercial sector energy demand was obtained by taking the weighted average of the four NIPA categories. Since data on relative energy demand for NIPA categories were not readily available, square footage was used as a proxy for calculating the weights. These data were taken from the *Nonresidential Buildings Energy Consumption Survey, Commercial Buildings Consumption and Expenditure 1986* (EIA, 1989). The resulting estimate for commercial sector changes in energy demand is provided in Table B-14.

Table B-14. Percentage Change in Commercial Energy Demand from Control to No-control Scenario.

Year	Coal	Refined Petroleum	Electric	Natural Gas
1975	-0.13	3.36	1.30	-0.80
1980	0.31	1.90	2.06	-0.82
1985	0.48	1.98	1.72	-0.40
1990	0.39	2.26	1.74	-0.22

The national-level change in commercial sector energy demand was allocated to the States using historic shares. Implicit is the assumption that removal of CAA regulations does not alter the State distribution of energy use.

Economic/Demographic Data

State population was assumed not to vary as a result of CAA regulations, thus only the economic variables were revised for the no-control scenario. No-control scenario housing starts and car stock were derived from J/W forecast information on construction and motor vehicles. The differential for categories 6 (construction) and 24 (motor vehicles and equipment) was applied to control scenario values to obtain no-control scenario levels. The percentage change from the J/W forecast is given in Table B-15.

State-level gasoline sales is one of the activities forecasted by the transportation sector model. The percentage change in gasoline sales calculated by the TEEMS model was used in the no-control scenario as a CRESS model input.

Table B-15. J/W Percent Differential in Economic Variables Used in CRESS.

Year	Construction	Motor Vehicles
1975	0.70	5.04
1980	0.14	4.79
1985	0.41	6.07
1990	0.29	6.25

Table B-16. TSP Emissions Under the Control and No-control Scenarios by Target Year (in thousands of short tons).

Sector	**With the CAA**				**Without the CAA**				**Difference in 1990 Emissions**
	1975	**1980**	**1985**	**1990**	**1975**	**1980**	**1985**	**1990**	
Transportation:									
Highway Vehicles	700	760	770	820	770	910	1,030	1,180	(30%)
Off-Highway Vehicles	270	280	270	280	260	270	260	270	5%
Stationary Sources:									
Electric Utilities	1,720	880	450	430	3,460	4,480	5,180	5,860	(93%)
Industrial Processes	5,620	3,650	3,040	3,080	11,120	12,000	11,710	12,960	(76%)
Industrial Boilers	740	480	250	240	780	550	360	400	(41%)
Commercial/Residential	2,020	2,510	2,680	2,550	2,020	2,520	2,700	2,560	(1%)
TOTAL*	11,070	8,550	7,460	7,390	18,410	20,730	21,250	23,230	(68%)

Notes: The estimates of emission levels *with and without the CAA* were developed specifically for this section 812 analysis using models designed to simulate conditions in the absence of the CAA. These numbers should not be interpreted as actual historical emission estimates.

*Totals may differ slightly from sums due to rounding.

Table B-17. SO_2 Emissions Under the Control and No-control Scenarios by Target Year (in thousands of short tons).

Sector	**With the CAA**				**Without the CAA**				**Difference in 1990 Emissions**
	1975	**1980**	**1985**	**1990**	**1975**	**1980**	**1985**	**1990**	
Transportation:									
Highway Vehicles	380	450	500	570	380	450	500	560	1%
Off-Highway Vehicles	370	530	410	390	360	530	400	390	1%
Stationary Sources:									
Electric Utilities	18,670	17,480	16,050	16,510	20,690	25,620	25,140	26,730	(38%)
Industrial Processes	4,530	3,420	2,730	2,460	5,560	5,940	5,630	6,130	(60%)
Industrial Boilers	3,440	3,180	2,660	2,820	3,910	4,110	4,020	4,610	(39%)
Commercial/Residential	1,000	800	590	690	1,000	810	610	710	(3%)
TOTAL*	28,380	25,860	22,950	23,440	31,900	37,460	36,310	39,140	(40%)

Notes: The estimates of emission levels *with and without the CAA* were developed specifically for this section 812 analysis using models designed to simulate conditions in the absence of the CAA. These numbers should not be interpreted as actual historical emission estimates.

*Totals may differ slightly from sums due to rounding.

Table B-18. NO$_x$ Emissions Under the Control and No-control Scenarios by Target Year (in thousands of short tons).

	With the CAA				**Without the CAA**				**Difference in 1990**
Sector	**1975**	**1980**	**1985**	**1990**	**1975**	**1980**	**1985**	**1990**	**Emissions**
Transportation:									
Highway Vehicles	8,640	9,340	8,610	8,140	9,020	11,060	13,160	15,390	(47%)
Off-Highway Vehicles	1,990	2,180	2,080	2,090	1,980	2,150	2,040	2,060	1%
Stationary Sources:									
Electric Utilities	5,540	6,450	6,660	7,060	5,740	7,150	7,780	8,300	(15%)
Industrial Processes	750	760	690	710	760	830	790	1,090	(35%)
Industrial Boilers	4,090	3,680	3,540	3,710	4,120	3,660	3,680	3,900	(5%)
Commercial/Residential	1,060	960	880	930	1,060	970	890	950	(2%)
TOTAL*	22,060	23,370	22,460	22,640	22,680	25,830	28,350	31,680	(29%)

Notes: The estimates of emission levels *with and without the CAA* were developed specifically for this section 812 analysis using models designed to simulate conditions in the absence of the CAA. These numbers should not be interpreted as actual historical emission estimates.

*Totals may differ slightly from sums due to rounding.

Table B-19. VOC Emissions Under the Control and No-control Scenarios by Target Year (in thousands of short tons).

	With the CAA				**Without the CAA**				**Difference in 1990**
Sector	**1975**	**1980**	**1985**	**1990**	**1975**	**1980**	**1985**	**1990**	**Emissions**
Transportation:									
Highway Vehicles	12,220	10,770	9,470	7,740	14,620	16,460	19,800	23,010	(66%)
Off-Highway Vehicles	1,380	1,370	1,340	1,410	1,390	1,420	1,390	1,490	(5%)
Stationary Sources:									
Electric Utilities	20	30	30	40	20	30	30	40	(7%)
Industrial Processes	5,910	6,780	6,230	5,630	6,130	7,930	7,290	6,810	(17%)
Industrial Boilers	150	150	150	150	150	150	140	150	0%
Commercial/Residential	4,980	5,480	5,820	5,870	4,980	5,700	6,080	6,130	(4%)
TOTAL*	24,660	24,580	23,030	20,840	27,290	31,680	34,730	37,630	(45%)

Notes: The estimates of emission levels *with and without the CAA* were developed specifically for this section 812 analysis using models designed to simulate conditions in the absence of the CAA. These numbers should not be interpreted as actual historical emission estimates.

*Totals may differ slightly from sums due to rounding.

Table B-20. CO Emissions Under the Control and No-control Scenarios by Target Year (in thousands of short tons).

Sector	With the CAA				Without the CAA				Difference in 1990 Emissions
	1975	**1980**	**1985**	**1990**	**1975**	**1980**	**1985**	**1990**	
Transportation:									
Highway Vehicles	83,580	79,970	72,490	65,430	90,460	105,530	131,420	149,280	(56%)
Off-Highway Vehicles	8,510	8,100	7,880	8,080	8,510	8,070	7,880	8,080	0%
Stationary Sources:									
Electric Utilities	240	280	290	370	250	290	300	380	(3%)
Industrial Processes	7,580	6,990	4,840	5,140	9,240	9,120	8,860	10,180	(49%)
Industrial Boilers	720	710	670	740	720	710	620	740	0%
Commercial/Residential	10,250	13,130	14,140	13,150	10,250	13,170	14,200	13,210	0%
TOTAL*	110,880	109,170	100,300	92,900	119,430	136,880	163,280	181,860	(49%)

Notes: The estimates of emission levels *with and without the CAA* were developed specifically for this section 812 analysis using models designed to simulate conditions in the absence of the CAA. These numbers should not be interpreted as actual historical emission estimates.

*Totals may differ slightly from sums due to rounding.

Table B-21. Lead (Pb) Emissions Under the Control and No-control Scenarios by Target Year (in thousands of short tons).

Sector	With the CAA				Without the CAA				Difference in 1990 Emissions
	1975	**1980**	**1985**	**1990**	**1975**	**1980**	**1985**	**1990**	
Transportation:									
Highway Vehicles	180	86	22	2	203	207	214	223	(99%)
Stationary Source:									
Industrial Processes	3	1	1	1	7	7	6	5	(87%)
Industrial Combustion	4	2	0	0	5	5	5	5	(96%)
Utilities	1	1	0	0	2	3	4	4	(95%)
TOTAL*	190	90	23	3	217	221	228	237	(99%)

Notes: The estimates of emission levels *with and without the CAA* were developed specifically for this section 812 analysis using models designed to simulate conditions in the absence of the CAA. These numbers should not be interpreted as actual historical emission estimates.

*Totals may differ slightly from sums due to rounding.

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Appendix C: Air Quality Modeling

Introduction

This appendix describes in greater detail the various methodologies used to translate differences in control and no-control scenario emission estimates into changes in air quality conditions. Summary characterizations of the results of the air quality modeling efforts for 1990 are provided here and in the main text. Further details and discussion of key analytical and modeling issues can be found in a number of supporting documents. These documents, which provide the analytical basis for the results presented herein, are:

- ICF Kaiser/Systems Applications International, *"Retrospective Analysis of Ozone Air Quality in the United States"*, Final Report, May 1995. (Hereafter referred to as "SAI Ozone Report (1995).")
- ICF Kaiser/Systems Applications International, "*Retrospective Analysis of Particulate Matter Air Quality in the United States"*, Draft Report, September 1992. (Hereafter referred to as "SAI PM Report (1992).")
- ICF Kaiser/Systems Applications International, "*Retrospective Analysis of Particulate Matter Air Quality in the United States"*, Final Report, April 1995. (Hereafter referred to as "SAI PM Report (1995).")
- ICF Kaiser/Systems Applications International, *"PM Interpolation Methodology for the section 812 retrospective analysis"*, Memorandum from J. Langstaff to J. DeMocker, March 1996. (Hereafter referred to as "SAI PM Interpolation Memo (1996).")
- ICF Kaiser/Systems Applications International, *"Retrospective Analysis of SO_2, NO_x and CO Air Quality in the United States"*, Final Report, November 1994. (Hereafter referred to as "SAI SO_2, NO_x and CO Report (1994).")
- ICF Kaiser/Systems Applications International, *"Retrospective Analysis of the Impact of the Clean Air Act on Urban Visibility in the Southwestern United States"*, Final Report, October 1994. (Hereafter referred to as "SAI SW Visibility Report (1994).")
- Dennis, Robin L., US EPA, ORD/NERL, *"Estimation of Regional Air Quality and Deposition Changes Under Alternative 812 Emissions Scenarios Predicted by the Regional Acid Deposition Model, RADM"*, Draft Report, October 1995. (Hereafter referred to as "RADM Report (1995).")

The remainder of this appendix describes, for each pollutant or air quality effect of concern, (a) the basis for development of the control scenario air quality profiles; (b) the air quality modeling approach used to estimate differences in air quality outcomes for the control and no-control scenario and the application of those results to the derivation of the no-control scenario air quality profiles; (c) the key assumptions, caveats, analytical issues, and limitations associated with the modeling approach used; and (d) a summary characterization of the differences in estimated air quality outcomes for the control and no-control scenarios.

Carbon Monoxide

Control scenario carbon monoxide profiles

As described in the preceding general methodology section, the starting point for development of control scenario air quality profiles was EPA's AIRS da-

Table C-1. Summary of CO Monitoring Data.

Year	Number of Monitors	Number of Counties	Percent Population Covered	Number of Samples	Mean Number of Samples per Monitor
1970	82	54	n/a	408,524	4,982
1975	503	246	n/a	2,667,525	5,303
1980	522	250	50 %	3,051,599	5,846
1985	472	232	n/a	3,533,286	7,486
1990	506	244	55 %	3,788,053	7,486

Data Source: SAI SO_2, NO_x and CO Report (1994).

tabase. Hourly CO air quality monitoring data were compiled for all monitors in the 48 contiguous states for the study target years of 1970, 1975, 1980, 1985, and 1990. Although the CO monitoring network was sparse in 1970, by 1990 506 monitors in 244 counties provided monitoring coverage for 55 percent of the population in the conterminous U.S. Table C-1 summarizes the CO monitoring data derived from AIRS. Additional data regarding the EPA Region location, land use category, location-setting category, and objective category of the monitors providing these data are described in the SAI SO_2, NO_x, and CO Report (1994).

The next step in constructing the control scenario air quality profiles was to calculate moving averages, for a variety of time periods, of the hourly CO data for each monitor. For CO, moving averages of 1, 3, 5, 7, 8, 12, and 24 hours were calculated. Daily maximum concentrations observed at each monitor for each of these averaging periods were then calculated. Finally, profiles were developed to reflect the average and maximum concentrations for each of the seven averaging periods. However, profiles were only developed for a given monitor when at least 10 percent of its theoretically available samples were actually available. The purpose of applying this cutoff was to avoid inclusion of monitors for which available sample sizes were too small to provide a reliable indication of historical air quality.

As discussed in the air quality modeling chapter of the main text, development of representative distributions for these profiles was then necessary to provide a manageable characterization of air quality conditions. Initially, two-parameter lognormal distributions were fitted to the profiles based on substantial evidence that such distributions are appropriate for modeling air quality data. However, given the relative importance of accurately modeling higher percentile observations (i.e., 90th percentile and higher), a three-parameter modeling approach was used to isolate the effect of observations equal, or very close, to zero. In this approach one parameter defines the proportion of data below a cutoff close to zero and the remaining two parameters describe the distribution of data above the cutoff value. Several other studies have already demonstrated good fit to air quality modeling data with a three-parameter gamma distribution, and both lognormal and gamma distributions using a three-parameter approach were developed for the present study. As documented in the SAI SO_2, NO_x, and CO Report (1994), a cutoff of 0.05 ppm was applied and both the three-parameter lognormal and three-parameter gamma distributions provided a good fit to the empirical data. For CO, the gamma distribution provided the best fit.

The control scenario air quality profiles are available on diskette. The filename for the CO Control Scenario profile database is COCAA.DAT, and adopts the format presented in Table C-2.

No-control scenario carbon monoxide profiles

To derive comparably configured profiles representing CO air quality in the no-control scenario, control scenario profile means and variances were adjusted in proportion to the difference in emissions estimated under the two scenarios. Specifically, for all control scenario air quality observations predicted by the three-parameter distributions falling above the “near-zero” cutoff level, comparable no-control estimates were derived by the following equation:

Table C-2. Format of Air Quality Profile Databases.

Columns	Format	Description
1 - 2	Integer	Year (70, 75, 80, 85, 90)
4 - 6	Integer	Averaging time (1, 3, 5, 7, 8, 12, 24 hours)
8 - 9	Integer	State FIPS code
11 - 13	Integer	County FIPS code
15 - 19	Integer	Monitor number (digits 6-10 of monitor id)
21 - 30	Real	Latitude
32 - 41	Real	Longitude
43 - 44	Integer	Latitude/longitude flag[a]
46 - 55	Real (F10.3)	Hourly intermittency parameter p[b]
56 - 65	Real (F10.3)	Hourly lognormal parameter μ[b]
66 - 75	Real (F10.3)	Hourly lognormal parameter σ[b]
76 - 85	Real (F10.3)	Hourly gamma parameter α[b]
86 - 95	Real (F10.3)	Hourly gamma parameter β[b]
96 - 105	Real (F10.3)	Daily max intermittency parameter p[b]
106 - 115	Real (F10.3)	Daily max lognormal parameter μ[b]
116 - 125	Real (F10.3)	Daily max lognormal parameter σ[b]
126 - 135	Real (F10.3)	Daily max gamma parameter α[b]
136 - 145	Real (F10.3)	Daily max gamma parameter β[b]

[a] Values for flag:
1 = actual latitude/longitude values
2 = latitude/longitude values from collocated monitor or previous monitor location (monitor parameter occurrence code 1)
-9 = latitude/longitude missing (county center substituted)

[b] Units of concentration are ppm for CO and ppb for SO_2, NO_2 and NO.

Source: SAI SO_2, NO_x and CO Report (1994).

$$X_{NC} = \left(\frac{E_{NC}}{E_C}\right)(X_C - b) + b \qquad (1)$$

where

X_{NC} = air quality measurement for the no-control scenario,
X_C = air quality measurement for the control scenario,
E_{NC} = emissions estimated for the no-control scenario,
E_C = emissions estimated for the control scenario, and
b = background concentration.

The adjustment for background concentration is made to hold ambient background concentrations of the pollutant constant between the control and no-control scenarios. To the extent background concentrations are affected by transport of anthropogenic pollutants from upwind sites, and to the extent upwind emissions may have been controlled under the control scenario, assuming a fixed background concentration represents a conservative assumption in this analysis. As discussed in the SAI SO_2, NO_x, and CO Report (1994), the CO background concentration used for this analysis was 0.2 ppm, which equals the lowest typical concentration observed in the lower 48 states.

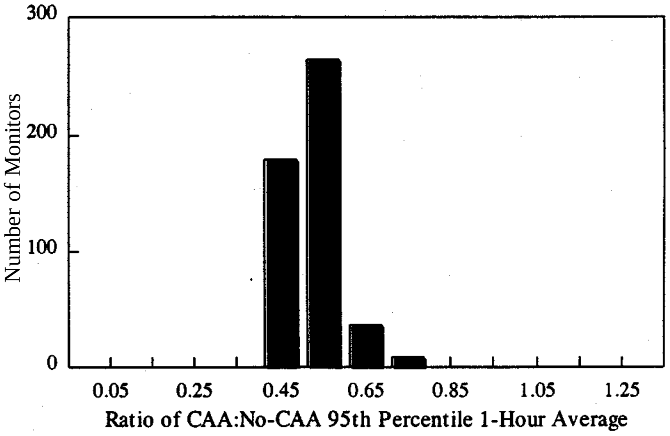
In the SAI SO_2, NO_x, and CO Report (1994) documenting the CO air quality modeling effort, reference is made to using county-level emission estimates as the basis for deriving the no-control profiles. Derivation of these county-level results is described in more detail in the appendix on emissions estimation. It is important to emphasize here, however, that the county-level CO emissions data were derived for both the control and no-control scenarios by simple population-weighted disaggregation of state-level emission totals. Although CO emission estimates were needed at the county level to support the ozone air quality modeling effort, differences in state-level emissions estimates are what drive the difference in the control and no-control air quality profiles for CO. In other words, the E_{NCAA} to E_{CAA} ratios used to derive the no-control profiles according to Equation (1) above are essentially based on state-level emissions estimates for CO.

As for the control scenario air quality profiles, the no-control scenario air quality profiles are available on diskette. The filename for the CO No-control Scenario profile database is CONCAA.DAT. The same data format described in Table C-2 is adopted.

Summary differences in carbon monoxide air quality

While the control and no-control scenario air quality profiles are too extensive to present in their entirety in this report, a summary indication of the difference in control and no-control scenario CO concentrations is useful. Figure C-1 provides this summary characterization. Specifically, the air quality indicator provided is the 95th percentile observation of 1990 CO concentrations averaged over a 1-hour period. The graph shows the number of monitors for which the ratio of 1990 control to no-control scenario 95th percentile 1-hour average concentrations falls within a particular range. The x-axis values in the graph represent the midpoint of each bin. The results indicate that, by 1990, CO concentrations under a no-control scenario would have been dramatically higher than control scenario concentrations.

Figure C-1. Frequency Distribution of Estimated Ratios for 1990 Control to No-control Scenario 95th Percentile 1-Hour Average CO Concentrations, by Monitor.



Key caveats and uncertainties for carbon monoxide

A number of important uncertainties should be noted regarding the CO air quality estimates used in this analysis. First and foremost, CO is a highly localized, “hot spot” pollutant. As such, CO monitors are often located near heavily-used highways and intersections to capture the peak concentrations associated with mobile sources. Since this analysis relies on state-level aggregate changes in CO emissions from all sources, the representativeness and accuracy of the predicted CO air quality changes are uncertain. There is no basis, however, for assuming any systematic bias which would lead to over- or under-estimation of air quality conditions due to reliance on state-wide emission estimates.

A second source of uncertainty is the extent to which the three-parameter distributions adequately characterize air quality indicators of concern. Appendix C of the SAI SO_2, NO_x, and CO Report (1994) presents a number of graphs comparing the fitted versus empirical data for one-hour and 12-hour averaging periods. In the case of CO, the gamma distribution appears to provide a very reasonable fit, though clearly some uncertainty remains.

Finally, a central premise of this analysis is that changes in CO emissions should be well-correlated with changes in CO air quality. Strong correlation between the state-level emissions estimates used in this analysis and empirical air quality measurements would not be expected due to inconsistencies between the state-level scale of modeled emissions versus the monitor-level scale of the air quality data, and between the modeled control scenario emissions inventories and actual historical air quality measurements. Under these circumstances, it is particularly important to focus on the primary objective of the current analysis, which is to estimate the difference in air quality outcomes between scenarios which assume the absence or presence of historical air pollution controls. In the process of taking differences, some of the uncertainties are expected to cancel out. No attempt is made in the overall analysis to predict historical air quality, or hypothetical air quality in the absence of the Clean Air Act, in absolute terms.

Table C-3. Summary of SO_2 Monitoring Data.

Year	Number of Monitors	Number of Counties	Percent Population Covered	Number of Samples	Mean Number of Samples per Monitor
1970	86	56	n/a	399,717	4,648
1975	847	340	n/a	4,280,303	5,053
1980	1,113	440	60 %	6,565,589	5,899
1985	926	401	n/a	6,602,615	7,130
1990	769	374	50 %	5,810,230	7,556

Data Source: SAI SO_2, NO_x and CO Report (1994).

Sulfur Dioxide

Sulfur dioxide (SO_2) emissions lead to several air quality effects, including secondary formation of fine particle sulfates, long range transport and deposition of sulfuric acid, and localized concentrations of gaseous sulfur dioxide. The first two effects are addressed later in this appendix, under the particulate matter and acid deposition sections. The focus of this section is estimation of changes in local concentrations of sulfur dioxide.

The methodology applied to estimation of local sulfur dioxide air quality is essentially identical to the one applied for carbon monoxide. As such, this section does not repeat the "roll-up" modeling methodological description presented in the CO section, but instead simply highlights those elements of the sulfur dioxide modeling which differ from carbon monoxide.

Control scenario sulfur dioxide profiles

Unlike the CO monitoring network, the number of monitors as well as the population coverage of the SO_2 monitoring network shrank during the 1980's. Table C-3 summarizes the SO_2 monitoring data used as the basis for development of the control scenario air quality profiles.

As for CO, air quality profiles reflecting average values and daily maxima for 1, 3, 5, 7, 8, 12, and 24 hour averages were compiled from AIRS for monitors in the lower 48 states which had at least 10 percent of their potential samples available. Applying a cutoff of 0.1 ppb to isolate the zero and near-zero observations, three-parameter lognormal and gamma distributions were fitted to these empirical profiles. In the case of SO_2, the three-parameter lognormal distribution was found to provide the best fit.

The control scenario SO_2 air quality profiles are available on diskette, contained in a file named SO2CAA.DAT. The same data format described in Table C-2 is adopted.

No-control scenario sulfur dioxide profiles

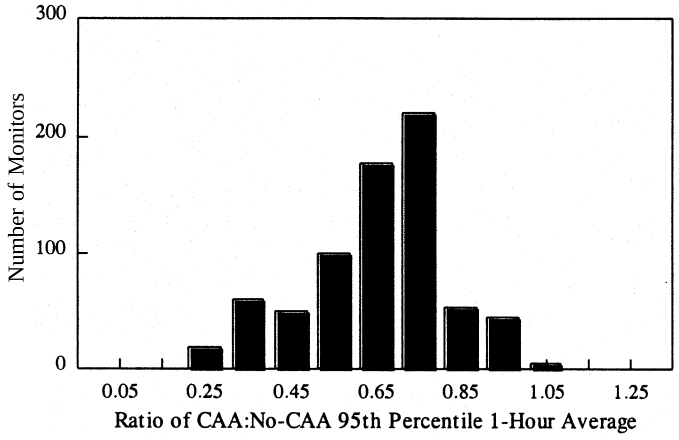
The no-control air quality profiles for SO_2 are derived using Equation 1, the same equation used for CO. For SO_2, the background concentration was assumed to be zero. Although anthropogenic emissions contribute only small amounts to total global atmospheric sulfur, measured background concentrations for the continental U.S. range from only 0.1 to 1.3 ppb. Background SO_2 is discussed in more detail in the supporting document SAI SO_2, NO_x, and CO Report (1994).[1]

The no-control scenario SO_2 air quality profiles are available on diskette, contained in a file named SO2NCAA.DAT. The data format is described in Table C-2.

Summary differences in sulfur dioxide air quality

As for CO, reporting differences in control and no-control scenario air quality projections for each monitor covered in the analysis is impractical due to the large amount of data involved. However, Figure C-2 provides an illustration of scenario differences similar to the one provided for CO. Specifically, the graph shows the distribution of 1990 control to no-control scenario 95th percentile 1-hour average concentrations ratios at SO_2 monitors. By 1990, SO_2 concentrations under the no-control scenario were substantially higher than those associated with the control scenario.

Figure C-2. Frequency Distribution of Estimated Ratios for 1990 Control to No-control Scenario 95th Percentile 1-Hour Average SO_2 Concentrations, by Monitor.



Key caveats and uncertainties for sulfur dioxide

The height of stacks used to vent flue gases from utility and industrial fossil fuel-fired boilers has a significant effect on the dispersion of sulfur dioxide and on the formation and long-range transport of secondary products such as particulate sulfates. Under a no-control scenario, it is conceivable that some sources might have built taller stacks to allow higher emission rates without creating extremely high ground-level concentrations of flue gases. On the other hand, it is also conceivable that, in the absence of post-1970 air pollution control programs, sources might have built shorter stacks to avoid incurring the higher costs associated with building and maintaining taller stacks. To the extent facilities would have adopted different stack height configurations under a no-control scenario, both local exposures to sulfur dioxides (and other emissions from fossil fuel combustion) and long-range transport, deposition, and exposure associated with secondary formation products may have been different. However, this analysis assumes that both the location of individual facilities and the height and configuration of emission stacks are constant between the two scenarios. If, in fact, stack heights were raised under the historical case due to CAA-related concerns, increases in local SO_2 concentrations under the no-control scenario may be overestimated. However, this same assumption may at the same time lead to underestimation under the no-control scenario of long-range transport and formation of secondary particulates associated with taller stacks. For stacks built lower under a no-control scenario, local SO_2 exposures would have been higher and long-range effects lower. Finally, the comments on uncertainties for carbon monoxide apply as well to SO_2.

Nitrogen Oxides

Similarly to sulfur dioxide, emissions of nitrogen oxides (NO_x) –including nitrogen dioxide (NO_2) and nitrous oxide (NO)– lead to several air quality effects. These effects include secondary formation of fine particle nitrates, formation of ground-level ozone, long range transport and deposition of nitric acid, and localized concentrations of both NO_2 and NO. The first three effects are addressed later in this appendix, under the particulate matter, ozone, and acid deposition sections. The focus of this section is estimation of changes in local concentrations of NO_2 and NO.

The methodology applied to estimation of local nitrogen oxides air quality is essentially identical to the one applied for carbon monoxide and sulfur dioxide. As such, this section does not repeat the "roll-up" modeling methodological description presented in the CO section, but instead simply highlights those elements of the nitrogen oxides modeling which differ from carbon monoxide.

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[1] SAI SO_2, NOx, and CO Report (1994), page 4-9.

Control scenario nitrogen oxides profiles

After peaking around 1980, the number of NO_2 and NO monitors, their county coverage, and their population coverage shrank between 1980 and 1990. Tables C-4 and C-5 summarize, respectively, the NO_2 and NO monitoring data used as the basis for development of the control scenario air quality profiles.

As for CO and SO_2, air quality profiles reflecting average values and maxima for 1, 3, 5, 7, 8, 12, and 24 hour NO_2 and NO averages were compiled from AIRS for monitors in the lower 48 states which had at least 10 percent of their potential samples available. Applying a cutoff of 0.5 ppb to both NO_2 and NO to isolate the zero and near-zero observations, three-parameter lognormal and gamma distributions were fitted to these empirical profiles. For NO_2 and NO, the three-parameter gamma distribution was found to provide the best fit.

The control scenario NO_2 and NO air quality profiles are available on diskette, contained in files named NO2CAA.DAT and NOCAA.DAT, respectively. The same data format described in Table C-2 is adopted.

Table C-4. Summary of NO_2 Monitoring Data.

Year	Number of Monitors	Number of Counties	Percent Population Covered	Number of Samples	Mean Number of Samples per Monitor
1970	45	32	n/a	275,534	6,123
1975	308	155	n/a	1,574,444	5,112
1980	379	205	45 %	1,984,128	5,235
1985	305	182	n/a	2,142,606	7,025
1990	346	187	40 %	2,456,922	7,101

Data Source: SAI SO_2, NO_x and CO Report (1994).

Table C-5. Summary of NO Monitoring Data.

Year	Number of Monitors	Number of Counties	Percent Population Covered	Number of Samples	Mean Number of Samples per Monitor
1970	39	28	n/a	246,262	6,314
1975	206	94	n/a	1,101,051	5,345
1980	224	124	30 %	1,023,834	4,571
1985	139	86	n/a	956,425	6,881
1990	145	81	15 %	999,808	6,895

Data Source: SAI SO_2, NO_x and CO Report (1994).

No-control scenario nitrogen oxides profiles

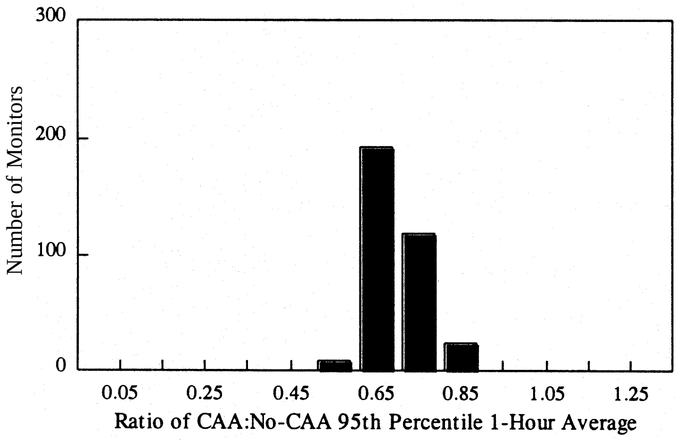
The no-control air quality profiles for NO_2 and NO are derived using Equation 1, the same equation used for CO and SO_2. As discussed in detail in the SAI SO_2, NO_x, and CO Report (1994),[2] nitrogen oxides are emitted almost entirely from anthropogenic sources and they do not have long atmospheric residence times. Therefore, global background concentrations are very low, on the order of 0.1 or 0.2 ppb. For the present analysis, background concentrations of NO_2 and NO were assumed to be zero.

The no-control scenario NO_2 and NO air quality profiles are available on diskette, contained in files named NO2NCAA.DAT and NONCAA.DAT, respectively. The data format is described in Table C-2.

Summary differences in nitrogen oxides air quality

Figure C-3 provides a summary indication of the differences in control and no-control scenario air quality for NO_2. As for CO and SO_2, the graph shows the distribution of 1990 control to no-control scenario 95th percentile 1–hour average concentration ratios at NO_2 monitors. These ratios indicate that, by 1990, no-control scenario NO_2 concentrations were significantly higher than they were under the control scenario. The changes for NO are similar to those for NO_2.

Figure C-3. Frequency Distribution of Estimated Ratios for 1990 Control to No-control Scenario 95th Percentile 1-Hour Average NO_2 Concentrations, by Monitor.



Key caveats and uncertainties for nitrogen oxides

A number of caveats and uncertainties specific to modeling NO_x should be noted. First, stack height and stack height control strategies likely to have influenced local concentrations of SO_2 may also have influenced local concentrations of NO_2 and NO. (For a fuller discussion of the stack heights issue, refer to the section "Key caveats and uncertainties for SO_2.") In addition, the earlier discussion of uncertainties resulting from the use of state-level emissions and the cancellation of uncertainties resulting from analyzing only differences or relative changes also applies to NO_x.

Acid Deposition

The focus of air quality modeling efforts described above for carbon monoxide, sulfur dioxide, and nitrogen oxides was to estimate the change in ambient concentrations of those pollutants as a result of changes in emissions. Particularly since the emissions modeling was driven by modeled macroeconomic conditions, rather than actual historical economic activity patterns, neither the emissions inventories nor the resultant air quality conditions developed for this analysis would be expected to match historical outcomes. The need to focus on relative changes, rather than absolute predictions, becomes even more acute for estimating air quality outcomes for pollutants subject to long-range transport, chemical transformation, and atmospheric deposition. The complexity of the relationships between emissions, air concentrations, and deposition is well-described in the following paragraph from the RADM report document developed by Robin Dennis of US EPA's National Exposure Research Laboratory in support of the present analysis:

> *"Sulfur, nitrogen, and oxidant species in the atmosphere can be transported hundreds to thousands of kilometers by meteorological forces. During transport the primary emissions, SO_2, NO_x, and volatile organic emissions (VOC) are oxidized in the air or in cloud-water to form new, secondary compounds, which are acidic, particularly sulfate and nitric acid, or which add to or subtract from the ambient levels of oxidants, such as ozone. The oxidizers, such as the hydroxyl radical, hydrogen peroxide and*

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[2] SAI SO_2, NO_x, and CO Report (1994), page 4-9.

> *ozone are produced by reactions of VOC and NO_x. The sulfur and nitrogen pollutants are deposited to the earth through either wet or dry deposition creating a load of pollutants to the earth's surface... However, the atmosphere is partly cleansed of oxidants through a number of physical processes including deposition (e.g., ozone is removed by wet and dry deposition). Dry deposition occurs when particles settle out of the air onto the earth or when gaseous or fine particle species directly impact land, plants, or water or when plant stomata take up gaseous species, such as SO_2. In wet deposition, pollutants are removed from the atmosphere by either rain or snow. In addition, fine particles or secondary aerosols formed by the gas- and aqueous-phase transformation processes scatter or absorb visible light and thus contribute to impairment of visibility."*[3]

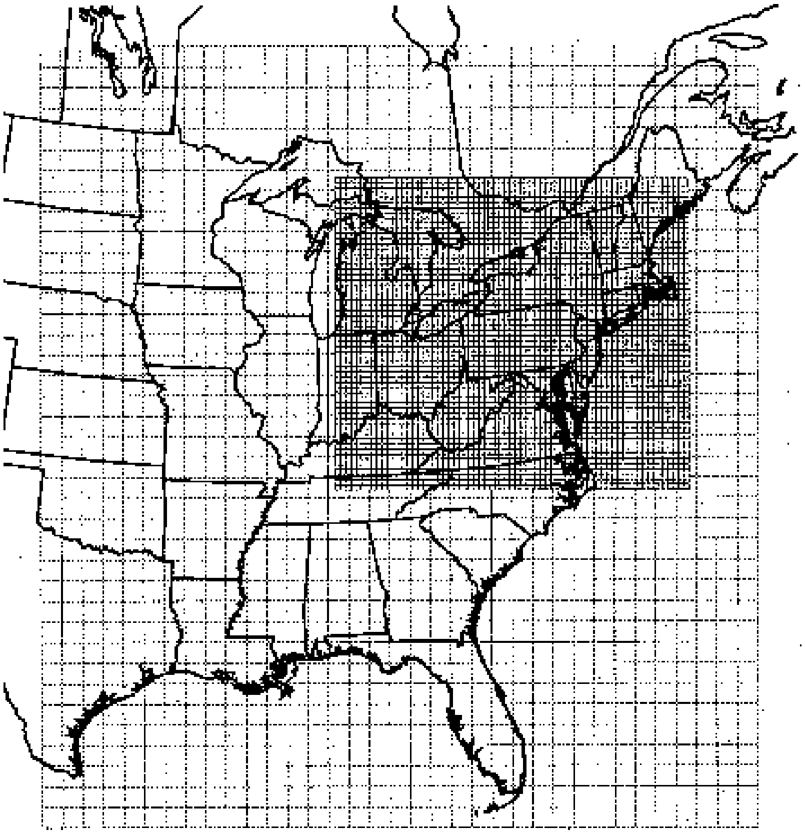
The complexity and nonlinearity of the relationships between localized emissions of precursors, such as SO_2 and VOC, and subsequent regional scale air quality and deposition effects are so substantial that the simple "roll-up" modeling methodology used for estimating local ambient concentrations of SO_2, NO_x, and CO is inadequate, even for a broad-scale, aggregate assessment such as the present study. For sulfur deposition, and for a number of other effects addressed in subsequent sections of this appendix, a regional air quality model was required. After careful review of the capabilities, geographic coverage, computing intensity, and resource requirements associated with available regional air quality models, EPA decided to use various forms of the Regional Acid Deposition Model (RADM) to estimate these effects.[4] Figure C-4 shows the geographic domain of the RADM.

Control scenario acid deposition profiles

The derivation of control scenario emission inventory inputs to the RADM model is succinctly described in this excerpt from the RADM Report (1995):

> *The RADM model requires a very detailed emissions inventory in both time and space. The emissions fields are also day-specific to account for the temperature effects on the volatile organics and the wind and temperature effects on the plume rise of the major point sources. At the time of the 812 retrospective study RADM runs, these inventories had been developed for 1985, using the 1985 NAPAP (National Acid Precipitation Assessment Program) inventory, and adjusted for point source*

Figure C-4. Location of the High Resolution RADM 20-km Grid Nested Inside the 80-km RADM Domain.



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[3] Dennis, R. RADM Report (1995), p. 1.

[4] For a detailed description of the various forms of the RADM and its evaluation history, see the Dennis, R. RADM Report (1995).

emissions to 1988 for the Eulerian Model Evaluation Field Study funded by NAPAP. These RADM emissions inventories had county-level and detailed SCC and species-level information incorporated into them to provide the 80- and 20-km detail. The 812 Study emissions are principally computed at the state level. While the 1985 812 Study emissions are close to the NAPAP inventory, they do not exactly match, nor do they have the spatial, nor economic sector, nor species detail within a state needed to run RADM. To connect the 812 Study emissions to the RADM emissions, the following approach was followed: An industry/commercial-level disaggregation (including mobile sources) was developed for the 812 emissions to allow different sectors in a state to change their emissions across time without being in lock step and the detailed NAPAP emissions for every 80- and 20-km RADM grid-cell were grouped by state to the same level of industry/commercial aggregation for an exact correspondence. Then it was assumed that the 812 Study 1985 control emissions were effectively the same as the 1985 NAPAP emissions. Relative changes in emissions between the 812 1985 control and any other scenario (e.g., 1985 no-control, or 1990 control, or 1980 no-control, etc.) were then applied to the 1985 NAPAP state-level industry/commercial groups in the appropriate 80- and 20-km grid cells. Thus, state-level emissions for each group would retain the same state-level geographic pattern in the different scenarios years, but the mix across groups could change with time. In this way, the more detailed emissions required by RADM were modeled for each scenario year using the 812 Study emissions data sets.[5]

Figure C-5. RADM-Predicted 1990 Total Sulfur Deposition (Wet + Dry; in kg/ha) Under the Control Scenario.

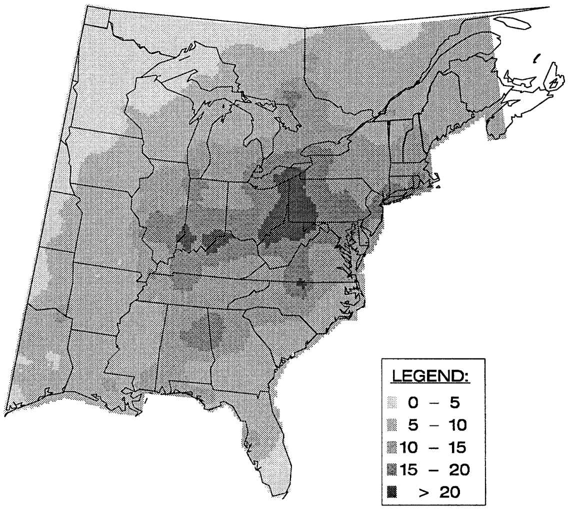
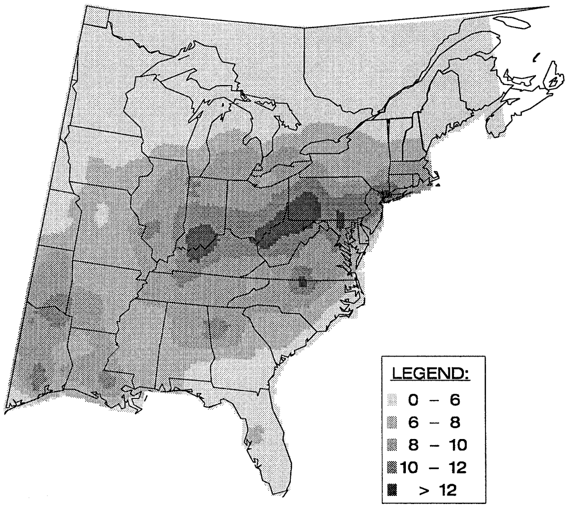


Figure C-6. RADM-Predicted 1990 Total Nitrogen Deposition (Wet + Dry; in kg/ha) Under the Control Scenario.



Although the focus of the present analysis is to estimate the differences between the control and no-control scenarios, it is useful to illustrate the absolute levels of acid deposition associated with the two scenarios. It is particularly important to demonstrate the initial deposition conditions to preclude possible misinterpretations of the maps showing percent change in deposition. A relatively high percentage change in a particular region, for example, may occur when initial deposition is low, even when the change in deposition is also modest. The RADM-modeled 1990 control scenario wet and dry sulfur deposition pattern is shown in Figure C-5. A comparable map for nitrogen deposition is presented in Figure C-6. Maps of the RADM-predicted 1990 no-control scenario sulfur and nitrogen deposition are presented in Figures C-7 and C-8, respectively.

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[5] Dennis, R. RADM Report (1995).

Figure C-7. RADM-Predicted 1990 Total Sulfur Deposition (Wet + Dry; in kg/ha) Under the No-control Scenario.

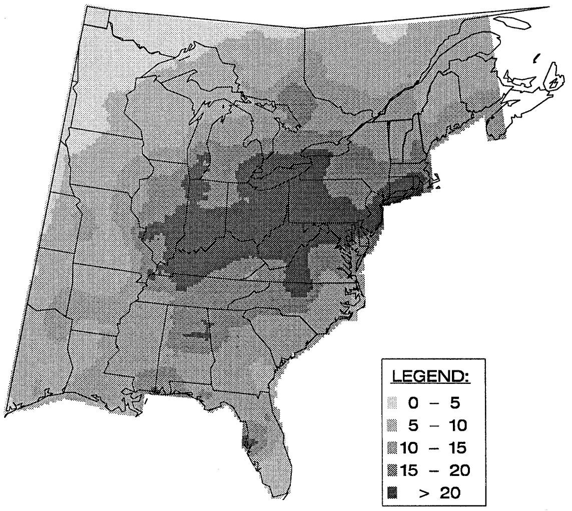
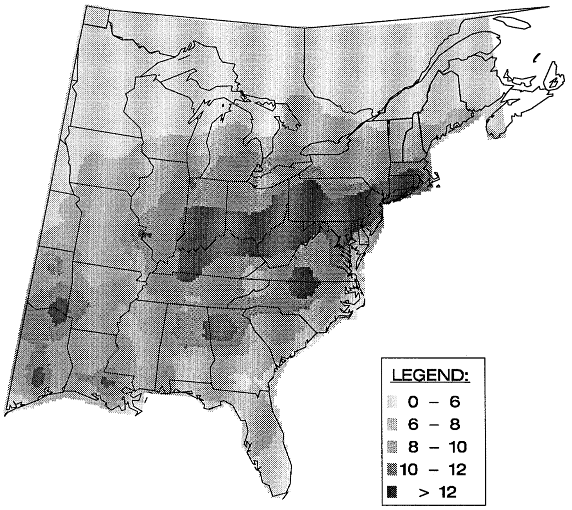


Figure C-8. RADM-Predicted 1990 Total Nitrogen Deposition (Wet + Dry; in kg/ha) Under the No-control Scenario.



No-control scenario acid deposition profiles

Configuration of the RADM model for the present analysis —including allocation of emission inventories to model grid cells, design of meteorological cases, treatment of biogenic versus anthropogenic emissions, and temporal, spatial, and species allocation of emissions— are described in detail in the RADM Report (1995). The remainder of this section provides a summary description of the acid deposition modeling effort.

For sulfur deposition, the RADM Engineering Model (RADM/EM), which focuses on sulfur compounds, was used to derive annual average total (wet plus dry) deposition of sulfur in kilograms sulfur per hectare (kg-S/ha) under both the control and no-control scenarios. The relative changes in annual average total sulfur deposition for each of the 80-km RADM/EM grid cells for 1975, 1980, 1985, and 1990 were then compiled.

Nitrogen deposition was calculated in a different manner. Since nitrogen effects are not included in the computationally fast RADM/EM, nitrogen deposition had to be derived from the full-scale, 15-layer RADM runs. Because of the cost and computational intensity of the 15-layer RADM, nitrogen deposition estimates were only developed for 1980 and 1990. As for sulfur deposition, the relative changes in annual average total (wet plus dry) nitrogen deposition, expressed as kg-N/ha, were calculated for each 80-km grid cell and for each of the two scenarios. It is important to note that ammonia depositin contributes significantly to total nitrogen deposition. However, the activities of sources associated with formation and deposition of ammonia, such as livestock farming and wildlife, were essentially unaffected by Clean Air Act-related control programs during the 1970 to 1990 period of this analysis. Therefore, ammonia deposition is held constant between the two scenarios.

Figure C-9. RADM-Predicted Percent Increase in Total Sulfur Deposition (Wet + Dry; in kg/ha) Under the No-control Scenario.

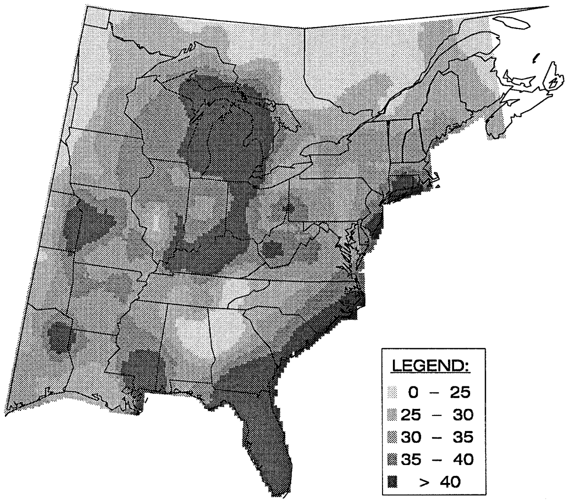
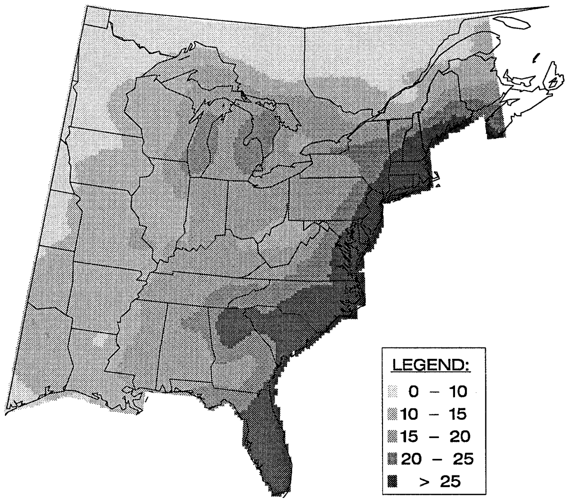


Figure C-10. RADM-Predicted Percent Increase in Total Nitrogen Deposition (Wet + Dry; in kg/ha) Under the No-control Scenario.



Summary differences in acid deposition

Figure C-9 is a contour map showing the estimated percent increase in sulfur deposition under the no-control scenario relative to the control scenario for 1990. Figure C-10 provides comparable information for nitrogen deposition. These maps indicate that by 1990 acid deposition would have been significantly higher across the RADM domain under the no-control scenario.

Examination of the percent change sulfur deposition map indicates relatively large percentage changes in the upper Great Lakes and the Florida-Southeast Atlantic Coast areas. This result may appear somewhat surprising to readers familiar with the historical patterns of acid deposition. However, a review of the emission data and the control scenario sulfur deposition map reveal the reasons for this result.

First, Figure C-5 shows that control scenario deposition rates are relatively low. As described above, even a small absolute increase in deposition leads to a large percentage increase in areas with low initial rates of deposition. Second, the scenario differences in SO_x emission rates for these areas were substantial. For example, 1990 no-control scenario total SO_x emissions for Michigan were approximately 1.8 million tons but control scenario emissions for the same year were less than 600,000 tons; a reduction of over two-thirds. Similarly, 1990 no-control scenario emissions for Florida were over 2.3 million tons, compared to approximately 800,000 tons under the control scenario; also a reduction of about two-thirds. Almost 1 million tons of the Michigan reduction and approximately 1.3 million tons of the Florida reduction were associated with utilities. Emission reductions of these magnitudes would be expected to yield significant reductions in rates of acid deposition.

Key caveats and uncertainties for acid deposition

Regional-scale oxidant and deposition modeling involves substantial uncertainty. This uncertainty arises from uncertainties in modeling atmospheric chemistry, incomplete meteorological data, normal seasonal and temporal fluctuations in atmospheric conditions, temporal and spatial variability in emissions, and many other factors. Uncertainties specific to the RADM model, and this particular exercise, are discussed in detail in the RADM Report (1995). It is important, however, to highlight some of the potential sources of modeling uncertainty unique to this analysis.

The first source of uncertainty specific to this analysis is associated with the spatial and geographic disaggregation of emissions data. As discussed in the RADM Report, the RADM model requires emission inventory inputs which are highly disaggregated over both time and space. The ideal emissions inventory fed into the RADM model includes day-specific emissions to account for temperature effects on VOCs and the significance of localized meteorological conditions around major point sources. Given the broad-scale, comprehensive nature of the present study, such detailed emissions inventories were not available. However, the industry/commercial-level disaggregation approach developed for the present analysis would not be expected to introduce any systematic bias, and the contribution of this disaggregation of emissions would not be expected to contribute significantly to the overall uncertainty of the larger analysis.

The acid deposition estimates included in the present analysis are limited in that only the eastern 31 of the 48 coterminous states are covered. Although acid deposition is a problem primarily for the eastern U.S., acid deposition does occur in states west of the RADM domain. The magnitude of the benefits of reducing acid deposition in these western states is likely to be small, however, relative to the overall benefits of the historical Clean Air Act.

Particulate Matter

Developing air quality profiles for particulate matter is significantly complicated by the fact that “particulate matter” is actually an aggregation of different pollutants with varying chemical and aerodynamic properties. Particulate species include chemically inert substances, such as wind-blown sand, as well as toxic substances such as acid aerosols; and include coarse particles implicated in household soiling as well as fine particles which contribute to human respiratory effects. In addition, emissions of both primary particulate matter and precursors of secondarily-formed particulates are generated by a wide variety of mobile and stationary sources, further complicating specification of particulate air quality models. Finally, particulate air quality models must take account of potentially significant background concentrations of atmospheric particles.

Modeling multiple species and emission sources, however, is not the only major challenge related to particulate matter which is faced in the present study. Over the 1970 to 1990 period being analyzed, understanding of the relative significance of fine versus coarse particles evolved significantly. Up until the mid-1980s, particulate air quality data were collected as Total Suspended Particulates (TSP). However, during the 1980s, health scientists concluded that small, respirable particles, particularly those with an aerodynamic diameter of less than or equal to 10 microns (PM_{10}), were the component of particulate matter primarily responsible for adverse human health effects. As of 1987, federal health-based ambient air quality standards for particulate matter were revised to be expressed in terms of PM_{10} rather than TSP. Starting in the mid-1980s, therefore, the U.S. began shifting away from TSP monitors toward PM_{10} monitors. As a result, neither TSP nor PM_{10} are fully represented by historical air quality data over the 1970 to 1990 period of this analysis. Furthermore, a large number of U.S. counties have no historical PM monitoring data at all, making it difficult to estimate changes in ambient concentrations of this significant pollutant for areas containing roughly 30 percent of the U.S. population.

Given the relative significance of particulate matter to the bottom-line estimate of net benefits of the historical Clean Air Act, it was important to develop methodologies to meet each of these challenges. The methodologies developed and data used are described primarily in the two supporting documents SAI PM Report (1992) and SAI PM Report (1995).[6] To summarize the overall approach, historical TSP data were broken down into principal component species, including primary particulates, sulfates, nitrates, organic particulates, and background particulates. Historical data were used for the control scenario. To derive the no-control profiles, the four non-background components were scaled up based on corresponding no-control to control scenario ratios of emissions and/or modeled atmospheric concentrations. Specifically, the primary particulate component was scaled up by the ratio of no-control to control emissions of PM.

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[6] In addition, SAI memoranda and reports which supplement the results and methodologies used in this analysis are included in the references.

Organic constituents were scaled up by the ratio of no-control to control VOC emissions. In the eastern 31 states where RADM sulfate and nitrate data were available, values for SO_4 and NO_3 from an appropriate RADM grid cell were assigned to the relevant county and used to scale these components of PM. For the western states not covered by RADM, sulfates were scaled up by the change in SO_2 emissions and nitrates were scaled up the change in NO_x emissions. No-control scenario profiles were then constructed by adding these scaled components to background concentrations.

To resolve the problem of variable records of TSP and PM_{10} data, both TSP and PM_{10} profiles were generated for the entire 20 year period. Missing early year data for PM_{10} were derived by applying region-specific, land use category-specific PM_{10} to TSP ratios to the historical TSP data. Missing recent year TSP data were derived for those areas where PM_{10} monitors replaced TSP monitors by applying the reciprocal of the relevant PM_{10} to TSP ratio. The methodology is described in detail in the SAI PM Report (1995).

In addition, to increase the geographic coverage of estimates of air quality, an interpolation methodology[7] was developed to predict air quality for the control scenario in counties without measured data. PM concentrations were estimated by first estimating the components of PM (i.e., sulfate, nitrate, and organic particulate, and primary particulate). The methodology for developing the concentrations of components within a county differed depending upon whether the county was within or outside the RADM domain.

For those counties within the RADM domain, the RADM modeled concentrations for 1980 and 1990 were used to predict sulfate air quality. Relationships based on linear regressions that related 1980 and 1990 RADM sulfate concentrations to estimated sulfate particulate concentrations were calculated for counties with AIRS data. Sulfate particulate concentrations were then calculated for all counties in the domain by applying the regression results to the RADM grid cell concentration located over the county center. Statewide average nitrate, VOC, and primary particulate concentrations were calculated from measured ambient TSP and PM_{10} to describe these constituents in counties without data. Control scenario PM profiles were developed by adding the RADM-estimated sulfate particulate levels to the statewide average nitrate, VOC, and primary particulate levels, and background.

For counties outside the RADM domain, an alternate procedure was used. Using the primary and secondary particulate estimates for counties with data, statewide average sulfate, nitrate, VOC, and primary particulate concentrations were determined. Control scenario PM_{10} was predicted by adding the statewide averages of all primary and secondary particulate, and background. Using this method, all counties that did not have monitors and are in the same state are assigned the same PM concentration profiles. These interpolated results are clearly less certain than results based on actual historical monitoring data and are therefore presented separately.

Control scenario particulate matter profiles

The number of TSP monitors peaked in 1977 and declined throughout the 1980s. Table C-6 summarizes the daily (i.e., 24-hour average) TSP monitoring data used as the basis for development of the control scenario air quality profiles. Most of the TSP and PM_{10} monitors collected samples every six days (i.e., 61 samples per year).

Daily PM_{10} data were also collected for each year between 1983 and 1990. Table C-7 summarizes the daily PM_{10} monitoring data used for the control scenario air quality profiles.

Table C-6. Summary of TSP Monitoring Data.

Year	Number of Monitors	Number of Counties	Number of Samples	Mean Number of Samples per Monitor
1970	751	245	56,804	76
1975	3,467	1,146	221,873	64
1980	3,595	1,178	234,503	65
1985	2,932	1,018	189,344	65
1990	923	410	59,184	64

Data Source: SAI PM Report (1995).

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[7] The interpolation methodology is described in detail in SAI, 1996. Memo from J. Langstaff to J. DeMocker. PM Interpolation Methodology for the section 812 retrospective analysis. March 1996.

Table C-7. Summary of PM_{10} Monitoring Data.

Year	Number of Monitors	Number of Counties	Number of Samples	Mean Number of Samples per Monitor
1985	303	194	22,031	73
1990	1,249	556	98,904	79

Data Source: SAI PM Report (1995).

Further speciation of TSP and PM_{10} air quality data serves two purposes in the present analysis. First, speciation of TSP into PM_{10} and other fractions allows derivation of PM_{10}:TSP ratios. Such ratios can then be used to estimate historical PM_{10} for those years and monitors which had TSP data but no PM_{10} data. The reciprocal ratio is also applied in this analysis to expand 1985 and 1990 TSP data to cover those areas which monitored PM_{10} but not TSP. The second purpose served by speciation of particulate data is, as described earlier, to provide a basis for scaling up concentrations of each species to derive no-control scenario TSP and PM_{10} profiles.

To break the TSP and PM_{10} data down into component species, speciation factors were applied to the PM fractions with aerodynamic diameters below 2.5 microns ($PM_{2.5}$) and from 2.5 to 10 microns (PM_{10}). The $PM_{2.5}$ speciation factors were drawn from a National Acid Precipitation Assessment Program (NAPAP) report on visibility which reviewed and consolidated speciation data from a number of studies.[8] These factors are presented in Table C-8. In the table, fine particle concentrations are based on particle mass measured after equilibrating to a relative humidity of 40 to 50 percent; and organics include fine organic carbon.

To develop speciation factors for coarser particles (i.e., in the $PM_{2.5}$ to PM_{10} range), SAI performed a review of the available literature, including Conner et al. (1991), Wolff and Korsog (1989), Lewis and Macias (1980), Wolff et al. (1983), Wolff et al. (1991), and Chow et al. (1994).[9] These speciation factors are summarized in Table C-9. Data were too limited to allow differentiation between urban and rural locations for coarser particles.

The TSP and PM_{10} control scenario profiles developed based on this methodology are available on diskette, under the filenames listed in Table C-10.

No-control scenario particulate matter profiles

To derive the no-control TSP and PM_{10} air quality profiles, individual component species were adjusted to reflect the relative change in emissions or, in the case of sulfates and nitrates in the eastern U.S., the relative change in modeled ambient concentration. The following excerpt from the SAI PM Report (1995) describes the specific algorithm used:[10]

> *"For the retrospective analysis, the no-CAA scenario TSP and PM_{10} air quality was estimated by means of the following algorithm:*
>
> - *Apportion CAA scenario TSP and PM_{10} to size categories and species;*
> - *Adjust for background concentrations;*
> - *Use a linear scaling to adjust the non-background portions of primary particulates, sulfate, nitrate, and organic components based on emissions ratios of PM, SO_2, NO_x and VOC, and Regional Acid Deposition Model (RADM) annual aggregation results for SO_4 and NO_3;*
> - *Add up the scaled components to estimate the no-CAA scenario TSP and PM_{10} concentrations."*

The specific procedures and values used for the linear rollback, speciation, fine to coarse particle ratio, scaling, and background adjustment steps are described in detail in the SAI PM report (1995).[11] Table C-11 lists the names of the electronic data files containing the TSP and PM_{10} profiles for the no-control scenario.

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[8] J. Trijonis, "Visibility: Existing and Historical Conditions--Causes and Effects," NAPAP Report 24, 1990.

[9] This literature review, and complete citations of the underlying studies, are presented in the SAI PM Report (1995), pp. 4-2 to 4-6 and pp. R-1 to R-2, respectively.

[10] SAI PM Report (1995), p. 5-1.

[11] SAI PM Report (1995), pp. 5-2 to 5-15.

Table C-8. Fine Particle ($PM_{2.5}$) Chemical Composition by U.S. Region.

Component	**Units**	**Number of Data Sets**	**Arithmetic Mean**	**Range of Values**
RURAL EAST				
Fine particle concentration	µg/m$_3$	19	18	6 - 46
Ammonium sulfate	% Fine particles	19	52	41 - 66
Ammonium nitrate	% Fine particles	3	1	1
Organics	% Fine particles	5	24	9 - 34
URBAN EAST				
Fine particle concentration	µg/m$_3$	3	36	29 - 43
Ammonium sulfate	% Fine particles	3	55	53 - 57
Ammonium nitrate	% Fine particles	2	1	1
Organics	% Fine particles	2	24	15 - 32
RURAL WEST				
Fine particle concentration	µg/m$_3$	25	5	1 - 11
Ammonium sulfate	% Fine particles	25	35	15 - 56
Ammonium nitrate	% Fine particles	17	4	1 - 17
Organics	% Fine particles	25	27	14 - 41
URBAN WEST				
Fine particle concentration	µg/m$_3$	16	35	13 - 74
Ammonium sulfate	% Fine particles	16	16	3 - 35
Ammonium nitrate	% Fine particles	14	15	2 - 37
Organics	% Fine particles	16	42	25 - 79

Data Sources: SAI PM Report (1995); and J. Trijonis, *"Visibility: Existing and Historical Conditions--Causes and Effects,"* NAPAP Report 24, 1990.

Summary differences in particulate matter air quality

Figure C-11 provides one indication of the estimated change in particulate matter air quality between the control and no-control scenarios. Specifically, the graph provides data on the estimated ratios of 1990 control to no-control scenario annual mean TSP concentrations in monitored counties. The X-axis values represent the mid-point of the ratio interval bin, and the Y-axis provides the number of counties falling into each bin. Figure C-11 indicates that annual average TSP concentrations would have been substantially higher in monitored counties under the no-control scenario.

Key caveats and uncertainties for particulate matter

There are several important caveats and uncertainties associated with the TSP and PM_{10} air quality profiles developed for this study. Although further

Table C-9. Coarse Particle ($PM_{2.5}$ to PM_{10}) Chemical Composition by U.S. Region.

Component	Units	Number of Data Sets	Arithmetic Mean	Range of Values
EAST				
Coarse particle concentration	µg/m^3	1	5.5	5.5
Ammonium sulfate	% Coarse particles	3	3	1 - 4
Ammonium nitrate	% Coarse particles	1	4	4
Organics	% Coarse particles	2	10	7 - 13.8
WEST				
Coarse particle concentration	µg/m^3	18	24	7.7 - 56.7
Ammonium sulfate	% Coarse particles	18	6	2.1 - 10.39
Ammonium nitrate	% Coarse particles	18	18	2.33-28.52
Organics	% Coarse particles	18	14	8.41-25.81

Data Source: SAI PM Report (1995).

Table C-10. PM Control Scenario Air Quality Profile Filenames.

Component	Indicator	Filename
TSP	Annual Mean	TSPCMEAN.DAT
TSP	2nd Highest Daily	TSPCHI2.DAT
TSP	(X)th Percentile	TSPC(X).DAT
PM_{10}	Annual Mean	PM10CMEA.DAT
PM_{10}	2nd Highest Daily	PM10CHI2.DAT
PM_{10}	(X)th Percentile	PM10C(X).DAT

Note: "(X)" refers to percentiles from 5 to 95, indicating 19 percentile data files available for TSP and 19 files available for PM_{10}; for example, the filename for the 50th percentile TSP air quality data profile for the control scenario is named TSPC50.DAT.

Table C-11. PM No-Control Scenario Air Quality Profile Filenames.

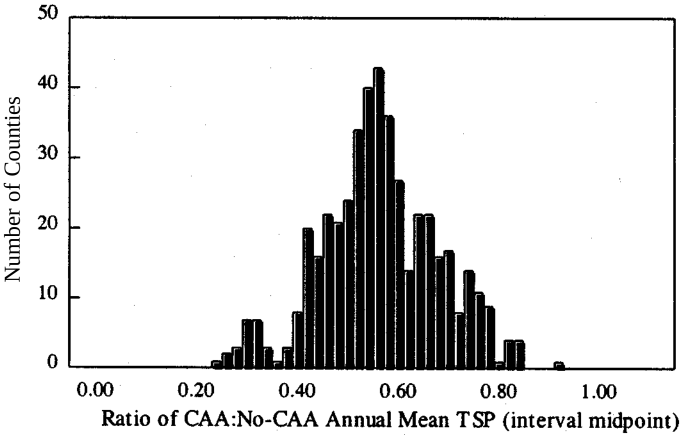
Component	Indicator	Filename
TSP	Annual Mean	TSPCNMEA.DAT
TSP	2nd Highest Daily	TSPNCHI.DAT
TSP	(X)th Percentile	TSPNC(X).DAT
PM$_{10}$	Annual Mean	PM10NCME.DAT
PM$_{10}$	2nd Highest Daily	PM10NCHI.DAT
PM$_{10}$	(X)th Percentile	PM10NC(X).DAT

Note: "(X)" refers to percentiles from 5 to 95, indicating 19 percentile-based data files available for TSP and 19 similar files available for PM$_{10}$; for example, the filename for the 50th percentile TSP air quality data profile for the no-control scenario is named TSPNC50.DAT.

reductions in these uncertainties were not possible for this study given time and resource limitations, the relative importance of particulate matter reduction contributions towards total benefits of the Clean Air Act highlights the importance of these uncertainties.

A number of uncertainties were introduced in the process of speciating and rolling up individual components of particulate matter. First, temporal and spatial variability in the size and chemical properties of particulate emissions are substantial. These characteristics change from day to day at any given location. Second, using changes in proxy pollutant emissions, such as using SO_2 as a surrogate for SO_4 in the western states, to roll up individual PM components may introduce significant uncertainty. Third, even assuming a satisfactorily high degree of correlation between target and surrogate pollutants, relying on predicted changes in emissions at the state level further compounds the uncertainty. Finally, and perhaps most important, using PM_{10} to TSP ratios derived from late 1980s monitoring data may lead to significant underestimation of reductions in fine particulates achieved in earlier years. This is because historical Clean Air Act programs focused extensively on controlling combustion sources of fine particulates. As a result, the share of TSP represented by PM_{10} observed in the late 1980s would be lower due to implementation of controls on combustion sources. This would lead, in turn, to underestimation of baseline PM_{10} concentrations, as a share of TSP, in the 1970s and early 1980s. If baseline PM_{10} concentrations in these early years are underestimated, the reductions in PM_{10} estimated by linear scaling would also be underestimated.[12]

Figure C-11. Distribution of Estimated Ratios for 1990 Control to No-control Annual Mean TSP Concentrations, by Monitored County.



Ozone

Nonlinear formation processes, long-range atmospheric transport, multiple precursors, complex atmospheric chemistry, and acute sensitivity to meteorological conditions combine to pose substantial difficulties in estimating air quality profiles for ozone. Even in the context of an aggregated, national study such as this, the location-specific factors controlling ozone formation preclude the use of roll-up modeling based on proxy pollutants or application of state-wide or nation-wide average conditions. Such simplifications would yield virtually meaningless results for ozone.

Ideally, large-scale photochemical grid models — such as the Urban Airshed Model (UAM)— would be used to develop control and no-control scenario estimates for ozone concentrations in rural and urban areas. Such models provide better representations of the effects of several important factors influencing air quality projections such as long-range atmospheric transport of ozone. However, the substantial computing time and data input requirements for such models precluded their use for this study.[13] Instead, three sepa-

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[12] See SAI PM Report (1995), p. 5-9.

[13] For a description of the extensive data inputs required to operate UAM, see SAI Ozone Report (1995), p. 1-1.

Table C-12. Urban Areas Modeled with OZIPM4.

Albany, NY
Albuquerque, NM
Allentown, PA-NJ
Altoona, PA
Anderson, IN
Appleton, WI
Asheville, NC
Atlanta, GA
Atlantic City, NJ
Auburn, ME
Augusta, GA-SC
Austin, TX
Baltimore, MD
Baton Rouge, LA
Beaumont, TX
Bellingham, WA
Billings, MT
Birmingham, AL
Boston, MA
Boulder, CO
Canton, OH
Cedar Rapids, IA
Champaign, IL
Charleston, SC
Charleston, WV
Charlotte, NC
Chattanooga, TN-GA
Chicago, IL
Cincinnati, OH
Cleveland, OH
Colorado Springs, CO
Columbia, SC
Columbus, GA-AL
Columbus, OH
Corpus Christi, TX
Cumberland, MD-WV
Dallas, TX
Davenport, IA-IL
Decatur, IL
Denver, CO
Detroit, MI
El Paso, TX
Erie, PA
Eugene, OR
Evansville, IN
Fayetteville, NC
Flint, MI
Fort Collins, CO
Fort Smith, AR-OK
Fort Wayne, IN
Grand Rapids, MI
Greeley, CO
Green Bay, WI
Greensboro, NC
Greenville, SC
Harrisburg, PA
Hartford, CT
Houston, TX
Huntington, WV-KY
Huntsville, AL
Indianapolis, IN
Iowa City, IA
Jackson, MS
Jacksonville, FL
Janesville Rock Co, WI
Johnson City, TN-VA
Johnstown, PA
Kansas City, MO
Knoxville, TN
Lafayette, IN
Lafayette, LA
Lake Charles, LA
Lancaster, PA
Lansing, MI
Las Cruces, NM
Las Vegas, NV
Lexington, KY
Lima, OH
Little Rock, AR
Longview, TX
Los Angeles, CA
Louisville, KY
Lynchburg, VA
Medford, OR
Memphis, TN
Miami, FL
Minneapolis, MN-WI
Mobile, AL
Monroe, LA
Montgomery, AL
Nashville, TN
New Orleans, LA
New York, NY
Norfolk, VA
Oklahoma City, OK
Omaha, NE-IA
Orange Co, CA
Orlando, FL
Owensboro, KY
Parkersburg, WV
Pascagoula, MS
Pensacola, FL
Peoria, IL
Philadelphia, PA
Phoenix, AZ
Portland, OR
Portsmouth, NH
Raleigh, NC
Reading, PA
Reno, NV
Richmond, VA
Roanoke, VA
Rochester, NY
Rockford, IL
Sacramento, CA
Salt Lake City, UT
San Antonio, TX
San Diego, CA
San Francisco, CA
San Joaquin Valley, CA
Santa Barbara, CA
Sarasota, FL
Scranton, PA
Seattle, WA
Sheboygan, WI
Shreveport, LA
South Bend, IN
Springfield, IL
Springfield, MO
Springfield, OH
St Louis, MO
Steubenville, OH-WV
Syracuse, NY
Tallahassee, FL
Tampa, FL
Terre Haute, IN
Toledo, OH
Tucson, AZ
Tulsa, OK
Utica-Rome, NY
Ventura County, CA
Victoria, TX
Washington, DC
Wheeling, WV-OH
Wichita, KS
York, PA
Youngstown, OH-PA

rate modeling efforts were conducted to provide urban and rural ozone profiles for those areas of the lower 48 states in which historical ozone changes attributable to the Clean Air Act may be most significant.

First, for urban areas the Ozone Isopleth Plotting with Optional Mechanisms-IV (OZIPM4) model was run for 147 urban areas. Table C-12 lists the urban areas modeled with OZIPM4. Although it requires substantially less input data than UAM, the OZIPM4 model provides reasonable evaluations of the relative reactivity of ozone precursors and ozone formation mechanisms associated with urban air masses.[14] Three to five meteorological episodes were modeled for each of the 147 urban areas; and for each of these, four model runs were performed to simulate the 1980 and 1990 control and no-control scenarios. The outputs of these model runs were peak ozone concentrations for each of the target year-scenario combinations. The differentials between the control and no-control scenario outputs were averaged over meteorological episodes and then applied to scale up historical air quality at individual monitors to obtain no-control case profiles. As for the other pollutants, the control scenario profiles were derived by fitting statistical distributions to actual historical data for individual monitors.

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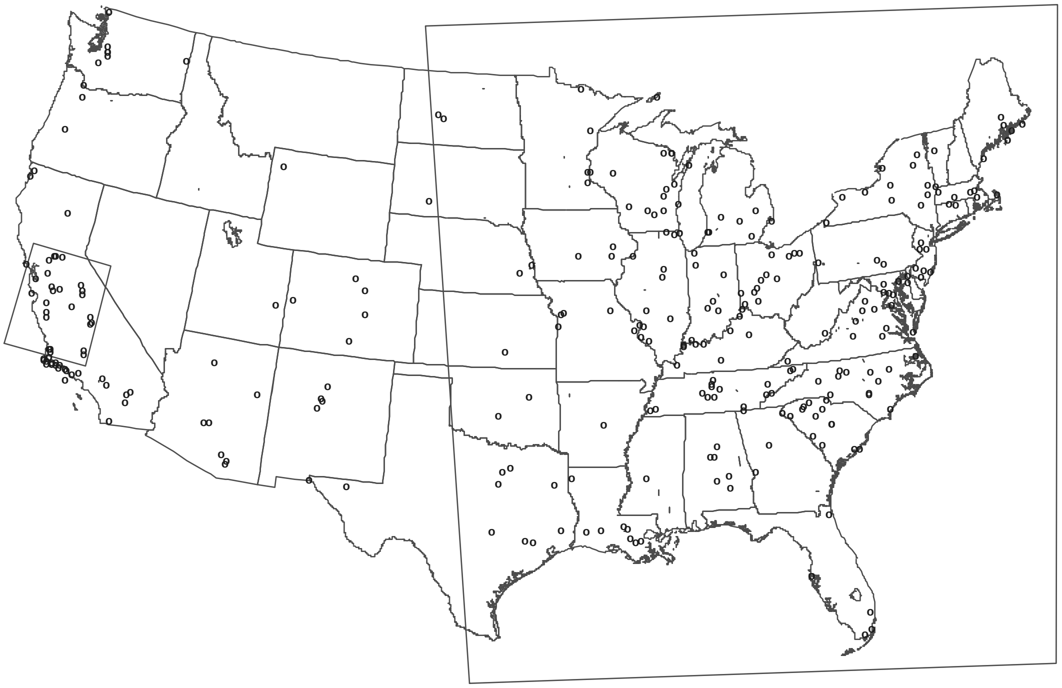
[14] See SAI Ozone Report (1995), p. 1-1.

Second, the 15-layer RADM runs for 1980 and 1990 were used to estimate the relative change in rural ozone distributions for the eastern 31 states. In addition, a limited number of 20-km grid cell high-resolution RADM runs were conducted to benchmark the 15-layer, 80-km RADM median ozone response and to estimate high ozone response. The relative changes in modeled median and 90th percentile rural ozone were then assumed to be proportional to the changes in, respectively, the median and 90th percentile ozone concentrations. The domain of the high-resolution RADM is shown in Figure C-4 and the general RADM domain is shown in Figure C-12.

Finally, the SARMAP Air Quality Model (SAQM) was run for EPA by the California Air Resources Board (CARB) to gauge the differences in peak ozone concentrations in key California agricultural areas for 1980 and 1990. No-control profiles were developed for ozone monitors in these areas by assuming the relative change in peak ozone concentration also applies to the median of the ozone distribution. The domain of the SAQM is shown in Figure C-12.

Figure C-12. RADM and SAQM Modeling Domains, with Rural Ozone Monitor Locations.



Control scenario ozone profiles

For ozone, air quality profiles were developed from historical AIRS data and calculated for individual monitors based on 1, 2, 6, 12, and 24 hour averaging times. Profiles based on the daily maximum concentrations for these averaging times were also calculated. Given the significance of seasonal and diurnal ozone formation, twelve separate profiles of hourly ozone distributions were also developed for six 2-month periods and for daytime and nighttime hours. The 2-month periods are January-February, March-April, and so forth. The diurnal/nocturnal profiles are divided at 7 A.M. and 7 P.M. Local Standard Time. All of these profiles are based on constructing 1, 2, 6, 12, and 24-hour moving average profiles from the hourly ozone data from each monitor.[15] A two-parameter gamma distribution is then fitted to characterize each of these air quality profiles.[16] The functional form of the gamma distribution, the basis for deriving the monitor-specific values for mean and variance, and an analysis of the goodness of fit to the data are presented in the SAI Ozone Report (1995).

Table C-13 summarizes the ozone monitoring data used as the basis for the control scenario profiles. The distribution of these monitors among urban, suburban, and rural locations is presented in Table C-2 of the SAI Ozone Report (1995).

Table C-13. Summary of Ozone Monitoring Data.

Year	Number of Monitors	Number of Counties
1970	1	1
1975	467	240
1980	791	415
1985	719	415
1990	834	477

Data Source: SAI Ozone Report (1995).

Given the substantial number of alternative air quality profiles for ozone, approximately 20 high-density disks are required to hold the profiles, even in compressed data format. Resource limitations therefore preclude general distribution of the actual profiles. As discussed in the caveats and uncertainties subsection below, however, the substantial uncertainties associated with model results for any given area preclude application of these profiles in contexts other than broad-scale, aggregated assessments such as the present study. The historical ozone monitoring data used as the basis for this study are, nevertheless, available through EPA's Aerometric Information Retrieval System (AIRS).

No-control scenario ozone profiles

The specific modeling methodologies for the OZIPM4 runs —including emissions processing, development of initial and boundary conditions, meteorological conditions, simulation start and end times, organic reactivity, and carbon fractions— are described in detail in the SAI Ozone Report (1995). Assumptions and modeling procedures not otherwise described in the SAI report were conducted in accordance with standard EPA guidance.[17]

Similarly, the RADM modeling methodology used to estimate changes in day-time rural ozone distributions in the eastern 31 states are described in detail in the RADM Report (1995). The referenced report also provides complete citations of the literature associated with development, standard application procedures, and evaluation of RADM by the National Acid Precipitation Assessment Program (NAPAP).

To derive the no-control scenario results for key California agricultural areas, the California Air Resources Board and US EPA's Region 9 office agreed to conduct three runs of the SAQM. For the 1990 control scenario, the 1990 SARMAP base case scenario adopted for California State Implementation Plan modeling was adopted.[18] Derivation of 1990 no-control and 1980 control and no-control scenarios was based on adjusting the aggregate mobile, point, and area source VOC and NO_x emissions associated with each of these cases. For example, the 1980 no-control results were derived by, first, multiplying the 1990 SARMAP base case mobile source VOC emissions by the ratio of 1980 no-control scenario to 1990 control scenario mobile source VOC emissions derived for the present study. Similar adjustments were made for point and area sources, and for NO_x. The SAQM was then re-run holding fixed all other conditions associated with the 1990 SARMAP base case, including meteorology, activity patterns, and other conditions. The specific emission ratios used to modify the 1990 SARMAP base case are presented in Table C-14. The ratios themselves were derived by adding on-highway and off-highway emissions to represent the mobile source category; adding utility, industrial process, and industrial combustion emissions to represent point sources; and using commercial/residential emissions to represent area sources. The no-control scenarios were then derived by adjusting the peak and median of the control scenario ozone distribution based on the ratio of SARMAP-predicted peak ozone concentrations under the control and no-control scenarios.

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[15] For the nighttime profiles, only 1, 2, 6, and 12-hour averaged concentrations are derived.

[16] Normal and lognormal distributions were also developed and tested for goodness of fit; however, the gamma distribution provided a better representation of the concentration distribution. See SAI Ozone Report (1995), page 4-2.

[17] US EPA, Office of Air Quality Planning and Standards, "*Procedures for Applying City-Specific EKMA*," EPA-450/4-89-012, 1989.

[18] Documentation of the SARMAP Air Quality Model and the SARMAP 1990 base case can be found in the SAQM references listed at the end of this appendix.

The relative results of the control and no-control scenario runs of the OZIPM4, RADM, and SAQM models were then used to derive the no-control case air quality profiles. For the urban monitors relying on OZIPM4 results, only ozone-season daytime concentrations could be calculated directly from OZIPM4 results. This is because OZIPM4 provides only the maximum hourly ozone concentration. However, to estimate all the various physical consequences of changes in ambient ozone concentrations, the current study requires estimation of the shift in the entire distribution of ozone concentrations. Since it is daytime ozone season concentrations which are most sensitive to changes in VOC and NO_x emissions, the predicted shifts in the most important component of the ozone concentration distribution are reasonably well-founded. The method adopted for this analysis involved applying the no-control to control peak concentration ratio to all concentrations in the distribution down to a level of 0.04 ppm. The 0.04 ppm level is considered at the high end of hypothetical ambient ozone concentrations in the absence of all anthropogenic ozone precursor emissions. A ratio of 1.0 is used for ozone concentrations at or near zero. The methodology is described in more detail in the SAI Ozone Report (1995) on page 4-6.

Table C-14. Apportionment of Emissions Inventories for SAQM Runs.

	Source Category	1980 Control to 1990 Control Ratio	1980 No-Control to 1990 Control Ratio	1990 No-Control to 1990 Control Ratio
VOC	Mobile	1.344	1.955	3.178
	Area	0.820	0.901	1.106
	Point	1.284	1.439	1.232
NO_x	Mobile	1.042	1.148	1.677
	Area	0.731	0.738	1.058
	Point	0.987	1.339	1.159

Estimating changes in rural ozone concentrations is required primarily for estimating effects on agricultural crops, trees, and other vegetation. For this reason, only the differences in daytime, growing season ozone concentrations are derived for the present study. As described in detail in the SAI Ozone Report (1995) on page 4-7, the no-control rural ozone profiles are calculated by, first, taking the ratio of the average daytime growing season ozone concentrations simulated by RADM or SAQM (whichever is relevant for that monitor). The ratio of no-control to control scenario average ozone concentration is then applied to all the hourly concentrations from that monitor.

Profiles based on 1, 2, 6, 12, and 24-hour averages are then calculated for the control case; and averages for daytime hours are calculated for the no-control case.[19] Even though the control and no-control scenario off-season profiles are held constant, profiles for the no-control scenario are developed for all months of the year since the ozone season varies throughout the country.

Summary differences in ozone air quality

Figure C-13 presents a summary of the results of the 1990 OZIPM4 results for all 147 of the modeled urban areas. Specifically, the graph depicts a frequency distribution of the ratio of control to no-control scenario peak ozone. While the vast majority of simulated peak ozone concentration ratios fall below 1.00, eight urban areas show lower simulated peak ozone for the no-control scenario than for the control scenario. For these eight urban areas, emissions of precursors were higher under the no-control scenario; however, the high proportion of ambient NO_x compared to ambient non-methane organic compounds (NMOCs) in these areas results in a decrease in net ozone production when NO_x emissions increase. Figures C-14 and C-15 present frequency distributions for control to no-control ratios of average ozone-season daytime ozone concentrations at rural monitors as simulated by RADM and SAQM, respectively.

These figures indicate that, by 1990, no-control scenario ozone concentrations in the modeled areas would have been generally higher in both urban and rural areas. Rural area concentrations differences are not as great as urban area differences due to (a) the differentially greater effect of CAA emission controls in high population density areas, and (b) potential differences in the models used for urban and rural areas.

Ozone reductions in both rural and urban areas projected in this analysis are not as proportionally large as the estimated reductions in emissions of ozone precursors for at least four reasons. First, current knowledge of atmospheric photochemistry suggests that ozone reductions resulting from emissions changes will be proportionally smaller than the emissions reductions. Second, biogenic emissions of VOCs, an important ozone precursor, are significant and are held constant for the control and no-control scenarios of this analysis. Biogenic emissions are important because they contribute roughly half of the total

Figure C-13. Distribution of Estimated Ratios for 1990 Control to No-control OZIPM4-Simulated 1-Hour Peak Ozone Concentrations, by Urban Area.

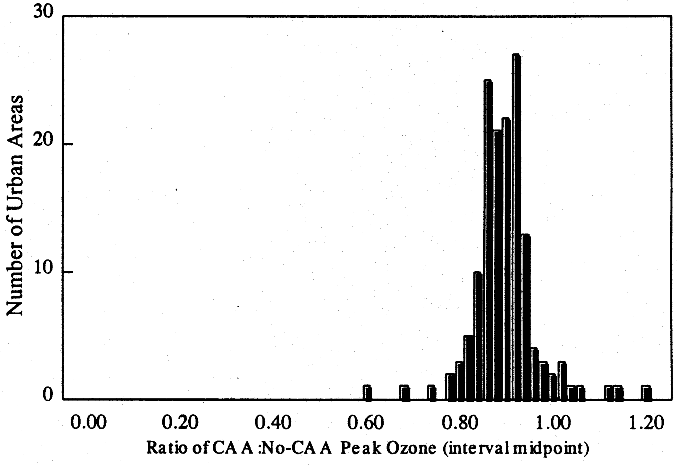


Figure C-14. Distribution of Estimated Ratios for 1990 Control to No-control RADM-Simulated Daytime Average Rural Ozone Concentrations, by RADM Grid Cell.

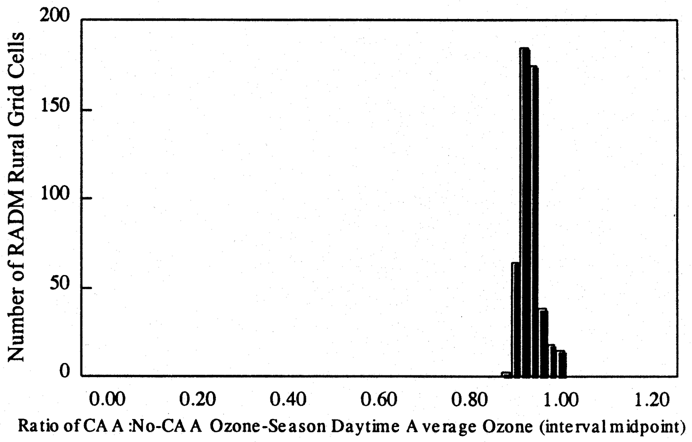
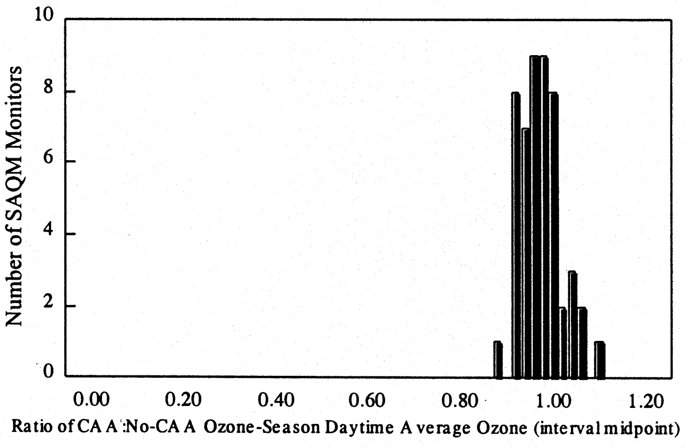


Figure C-15. Distribution of Estimated Ratios for 1990 Control to No-control SAQM-Simluated Daytime Average Ozone Concentrations, by SAQM Monitor.



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[19] The no-control scenario nighttime profiles are assumed to be the same as the control scenario profiles.

(manmade plus natural) VOC emissions nationwide. Due to this abundance of VOC loading and the inherent nonlinearity of the ozone-precursor response system,[20] historical reductions in anthropogenic VOC emissions can yield minimal reductions in ozone, especially in rural environments. Third, this rural effect also influences urban areas receiving substantial ozone transported in from surrounding areas. Consequently, the effect of emission controls placed in urban areas often is reduced since much of the urban area ozone is imported. Thus, the problem is truly regionalized given the importance of transport, biogenic emissions and associated urban-rural interactions, all contributing toward a relatively non-responsive atmospheric system.[21] Finally, physical process characterizations within OZIPM4 are severely limited and incapable of handling transport, complex flow phenomena, and multi-day pollution events in a physically realistic manner. Consequently, it is possible that the OZIPM4 method used herein produces negative bias tendencies in control estimations. Additional discussion of uncertainties in the ozone air quality modeling is presented in the following section.

Key caveats and uncertainties for ozone

There are a number of uncertainties in the overall analytical results of the present study contributed by the ozone air quality modeling in addition to the potential systematic downward bias discussed above. First, there are substantial uncertainties inherent in any effort to model ozone formation and dispersion. These uncertainties are compounded in the present study by the need to perform city-specific air quality modeling using OZIPM4, which is less sophisticated than an Eulerian model such as the Urban Airshed Model. However, while the absolute ozone predictions for any given urban area provided by OZIPM4 may be quite uncertain, the process of aggregating results for a number of cities and meteorological episodes should significantly reduce this uncertainty.[22] Urban areas for which ozone changes may be overpredicted are offset to some degree by urban areas for which the change in ozone concentrations may be underpredicted. In weighing the significance of this source of uncertainty, it is important to consider the central purpose of the present study, which is to develop a reasonable estimate of the overall costs and benefits of all historical Clean Air Act programs. All analyses are based on relative modeled results, and ratios of the model predictions for the control and no-control scenarios, rather than the absolute predictions. As a result of this, the effect of any bias in the model predictions is greatly reduced due to partial cancellation.

Additional uncertainty is contributed by other limitations of the models, the supporting data, and the scope of the present analysis. Relying on linear interpolation between 1970 and modeled 1980 results to derive results for 1975, and between modeled results for 1980 and 1990 to derive results for 1985, clearly adds to the uncertainty associated with the RADM-based rural ozone estimates. Assuming that changes in peak concentration predicted by OZIPM4 and SAQM can be applied to scale hourly ozone values throughout the concentration distribution also contributes to uncertainty. Resource and model limitations also required that night-time ozone concentrations be held constant between the scenarios. This leads to an underestimation of the night-time component of ozone transport. Finally, changes in rural ozone in areas not covered by RADM or SAQM could not be estimated. As a result, potentially significant changes in ambient ozone in other major agricultural areas, such as in the mid-west, could not be developed for this analysis. The Project Team considered using an emissions scaling (i.e., a roll-back) modeling strategy to develop crude estimates of the potential change in rural ozone concentrations in monitored areas outside the RADM and SAQM domains. However, the Project Team concluded that such estimates would be unreliable due to the nonlinear effect on ozone of precursor emission changes. Furthermore, the team concluded that baseline levels of ozone and changes in precursor emissions in these areas are relatively low. The decision not to spend scarce project resources on estimating ozone changes in these rural areas is further supported by the relatively modest change in rural ozone concentrations estimated within the RADM and SAQM domains.

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[20] Nonlinear systems are those where a reduction in precursors can result in a wide range of responses in secondary pollutants such as ozone. Ozone response often is “flat” or nonresponsive to reductions of VOCs in many rural areas with significant natural VOC emissions. Also, ozone can increase in response to increases in NO_x emissions in certain localized urban areas.

[21] Both the 1990 CAA and EPA’s and the National Academy of Science’s Section 185B Report to Congress recognized the consequences of biogenics, transport and the need to conduct regionalized assessments, as reflected in organizational structures such as the Ozone Transport Commission and the North American Research Strategy for Tropospheric Ozone (NARSTO).

[22] Note that aggregating individual urban area results may reduce the effect of uncertainty in individual city projections (i.e., overestimated cities would offset underestimated cities). However, aggregation of individual urban area results would not reduce potential errors caused by systematic biases which arise due to, for example, misestimated emissions inventories.

Visibility

Two separate modeling approaches were used to estimate changes in visibility degradation in the eastern and southwestern U.S. These are the two regions of the coterminous U.S. for which Clean Air Act programs were expected to have yielded the most significant reductions in visibility degradation. Visibility changes in the eastern 31 states were estimated based on the RADM/EM results for sulfates; and changes in visibility in 30 southwestern U.S. urban areas were calculated using a linear emissions scaling approach. Despite the potential significance of Clean Air Act-related visibility changes in southwestern U.S. Class I areas, such as National Parks, resource limitations precluded implementation of the analysis planned for these areas.

The RADM/EM system includes a post-processor which computes various measures of visibility degradation associated with changes in sulfate aerosols.[23] The basic approach is to allocate the light extinction budget for the eastern U.S. among various aerosols, including particulate sulfates, nitrates, and organics. The change in light extinction from sulfates is provided directly by RADM, thereby reflecting the complex formation and transport mechanisms associated with this most significant contributor to light extinction in the eastern U.S. Nitrates are not estimated directly by RADM. Instead, RADM-estimated concentrations of nitric acid are used as a surrogate to provide the basis for estimating changes in the particulate nitrate contribution to light extinction. The organic fractions were held constant between the two scenarios. Standard outputs include daylight distribution of light extinction, visual range, and DeciViews[24] for each of RADM's 80-km grid cells. For the present study, the RADM visibility post-processor was configured to provide the 90th percentile for light extinction and the 10th percentile for visual range to represent worst cases; and the 50th percentile for both of these to represent average cases. More detailed documentation of the RADM/EM system and the assumptions used to configure the visibility calculations are presented in the RADM Report (1995).

To estimate differences in control and no-control scenario visibility in southwestern U.S. urban areas, a modified linear rollback approach was developed and applied to 30 major urban areas with population greater than 100,000.[25] For each of the 30 urban centers, seasonal average 1990 air quality data was compiled for key pollutants, including NO_2 and PM_{10}, contributing to visibility degradation in southwestern U.S. coastal and inland cities. PM_{10} was then speciated into its key components using city-specific annual average PM_{10} profile data. After adjusting for regional — and for some species, city-specific— background levels, concentrations of individual light-attenuating species were scaled linearly based on changes in emissions of that pollutant or a proxy pollutant.[26] Using the same approach used for the 1993 EPA Report to Congress on effects of the 1990 Clean Air Act Amendments on visibility in Class I areas, light extinction coefficients for each of these species were then multiplied by their respective concentrations to derive a city-specific light extinction budget.[27] This process was repeated for pre-1990 control and all no-control scenarios by scaling 1990 results by the relative change in annual county-level emissions of SO_x, NO_x, and PM. Based on the city-specific light extinction budget calculations, measures for total extinction, visual range, and DeciView were calculated for each scenario and target year.

Control scenario visibility

Unlike the other air quality conditions addressed in the present study, modeled visibility conditions are used as the basis for the control scenario rather than actual historical conditions. However, like the other air quality benefits of the historical Clean Air Act, it is the differences between modeled visibility outcomes for the control and no-control scenarios which are used to estimate visibility benefits. Nevertheless, 1990 absolute levels of eastern U.S. visibility predicted by RADM under the control scenario are presented in Figure C-16 to provide a sense of initial visibility conditions.

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[23] A complete discussion, including appropriate references to other documents, of the RADM and RADM/EM modeling conducted for the present study is presented in the subsection on acid deposition earlier in this appendix.

[24] The DeciView Haze Index (dV) is a relatively new visibility indicator aimed at measuring visibility changes in terms of human perception. It is described in detail in the SAI SW Visibility Report (1994), pp. 4-2 to 4-3. See also Pitchford and Malm (1994) for the complete derivation of the DeciView index.

[25] Complete documentation of the linear scaling modeling, speciation methodologies, spatial allocation of emissions, and other data and assumptions are provided by the SAI SW Visibility Report (1994).

[26] For example, sulfate (SO_4) concentrations were scaled based on changes in sulfur oxide (SO_x) emissions.

[27] The term "light extinction budget" refers to the apportionment of total light attenuation in an area to the relevant pollutant species.

For the southwestern urban areas, 1990 control scenario annual average light extinction budget, visual range, and DeciView conditions are listed in Table C-15. These 1990 results are presented to give the reader a sense of the initial visibility conditions in absolute, albeit approximate, terms.

No-control scenario visibility

The no-control scenario visibility results for the eastern U.S. area covered by RADM are presented in Figure C-17. No-control scenario 1990 outcomes for the 30 southwestern U.S. urban areas are presented in Table C-16.

Summary differences in visibility

DeciView Haze Index

The DeciView Haze Index (dV) has recently been proposed as an indicator of the clarity of the atmosphere that is more closely related to human perception than visual range (VR) or total extinction (b_{ext}) (Pitchford and Malm, 1994). It is defined by the equation:

$$dV = 10\ 1\text{ne}\ (\frac{b_{ext}}{10}) \qquad (2)$$

where:

b_{ext} = total extinction in inverse megameters (Mm^{-1})

This index has the value of approximately 0 when the extinction coefficient is equal to the scattering coefficient for particle-free air (Rayleigh scattering) and increases in value by approximately one unit for each 10 percent increase in b_{ext}. Since the apparent change in visibility is related to the percent change in b_{ext} (Pitchford et al., 1990), equal changes in dV correspond to approximately equally perceptible changes in visibility. Recent research indicates that, for most observers, a "just noticeable change" in visibility corresponds to an increase or decrease of about one to two dV units.

Figure C-16. RADM-Predicted Visibility Degradation, Expressed in Annual Average DeciView, for Poor Visibility Conditions (90th Percentile Under the Control Scenario.

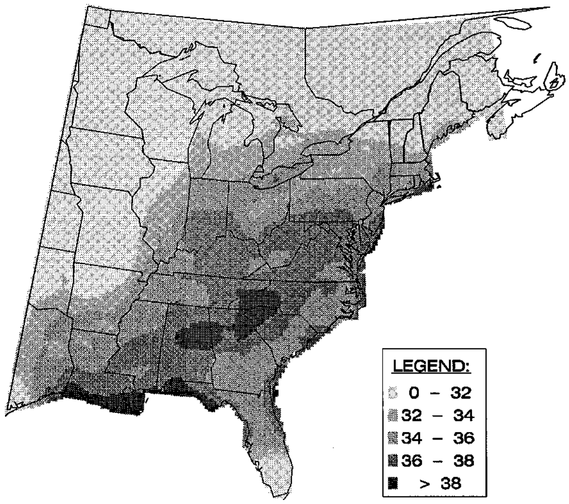


Figure C-17. RADM-Predicted Visibility Degradation, Expressed in Annual Average DeciView, for Poor Visibility Conditions (90th Percentile Under the No-control Scenario.

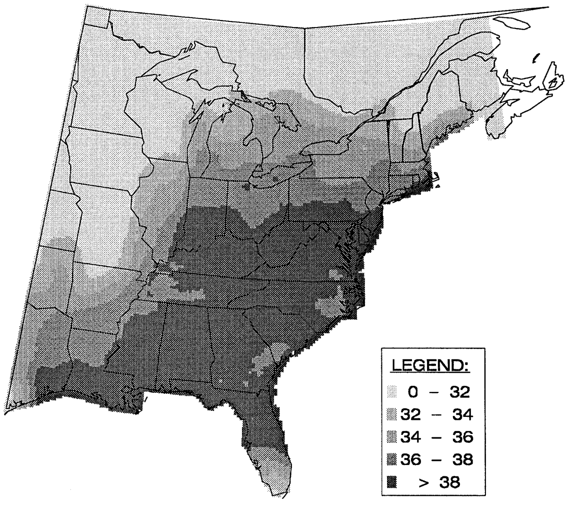


Table C-15. 1990 Control Scenario Visibility Conditions for 30 Southwestern U.S. Cities.

City	Light Extinction Budget (b, Mm^{-1})	Visual Range (km)	Deci View (dV)
Los Angeles, CA	197.6	15.2	29.8
San Bernardino, CA	201.7	14.9	30.0
Riverside, CA	208.3	14.4	30.4
Anaheim, CA	170.1	17.6	28.3
Ventura, CA	113.3	26.5	24.3
San Diego, CA	126.9	23.6	25.4
Santa Barbara, CA	112.8	26.6	24.2
Bakersfield, CA	215.1	13.9	30.7
Fresno, CA	211.7	14.2	30.5
Modesto, CA	148.8	20.2	27.0
Stockton, CA	153.1	19.6	27.3
San Francisco, CA	120.8	24.8	24.9
Oakland, CA	117.5	25.5	24.6
San Jose, CA	154.6	19.4	27.4
Monterey, CA	84.7	35.4	21.4
Sacramento, CA	119.1	25.2	24.8
Redding, CA	83.2	36.1	21.2
Reno, NV	147.4	20.3	26.9
Las Vegas, NV	157.9	19.0	27.6
Salt Lake City, UT	117.5	25.5	24.6
Provo, UT	107.8	27.8	23.8
Fort Collins, CO	80.7	37.2	20.9
Greeley, CO	84.2	35.6	21.3
Denver, CO	153.4	19.6	27.3
Colorado Springs, CO	83.3	36.0	21.2
Pueblo, CO	88.1	34.1	21.8
Albuquerque, NM	91.1	32.9	22.1
El Paso, TX	109.3	27.5	23.9
Tucson, AZ	85.6	35.0	21.5
Phoenix, AZ	125.3	23.9	25.3

Data Source: SAI SW Visibility Report (1994).

Table C-16. 1990 No-control Scenario Visibility Conditions for 30 Southwestern U.S. Cities.

City	Light Extinction Budget (b, Mm^{-1})	Visual Range (km)	DeciView (dV)
Los Angeles, CA	333.4	9.0	35.1
San Bernardino, CA	337.3	8.9	35.2
Riverside, CA	343.2	8.7	35.4
Anaheim, CA	286.3	10.5	33.5
Ventura, CA	194.8	15.4	29.7
San Diego, CA	210.1	14.3	30.4
Santa Barbara, CA	183.2	16.4	29.1
Bakersfield, CA	356.4	8.4	35.7
Fresno, CA	349.0	8.6	35.5
Modesto, CA	240.1	12.5	31.8
Stockton, CA	248.1	12.1	32.1
San Francisco, CA	197.3	15.2	29.8
Oakland, CA	188.6	15.9	29.4
San Jose, CA	253.0	11.9	32.3
Monterey, CA	141.4	21.2	26.5
Sacramento, CA	189.2	15.9	29.4
Redding, CA	128.6	23.3	25.5
Reno, NV	416.6	7.2	37.3
Las Vegas, NV	643.8	4.7	41.6
Salt Lake City, UT	185.8	16.1	29.2
Provo, UT	159.0	18.9	27.7
Fort Collins, CO	191.2	15.7	29.5
Greeley, CO	117.0	25.6	24.6
Denver, CO	284.4	10.5	33.5
Colorado Springs, CO	175.8	17.1	28.7
Pueblo, CO	299.9	10.0	34.0
Albuquerque, NM	175.8	17.1	28.7
El Paso, TX	276.3	10.9	33.2
Tucson, AZ	272.2	11.0	33.0
Phoenix, AZ	429.5	7.0	37.6

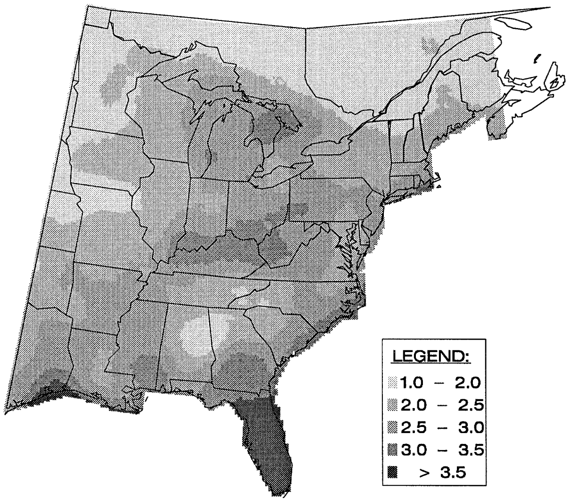
Data Source: SAI SW Visibility Report (1994).

Both VR and dV are measures of the value of b_{ext} at one location in the atmosphere. Both are unaffected by the actual variability of the compositions and illumination of the atmosphere, so neither is closely linked to the human perception of a particular scene. The isolation of these parameters from site-specific variations and temporal fluctuations of the atmospheric illumination increases their usefulness for comparing the effects of air quality on visibility across a range of geographic locations for a range of time periods. Each parameter attempts to scale the b_{ext} data so that changes in air quality can be used to provide an indication of changes in the human perception of a scene.

Modeling Results

The differences in modeled 1990 control and no-control scenario visibility conditions projected by the RADM/EM for the eastern U.S. are presented in Figure C-18. The map shows the percent increase in modeled annual average visibility degradation under poor conditions for 1990 when moving from the control to the no-control scenario. The results indicate perceptible differences in visibility between the control and no-control scenario throughout the RADM domain. The relatively large increase in visibility impairment in the Gulf Coast area is a reflection of the significant increases in 1990 sulfate concentrations associated with the no-control scenario. (See the earlier discussion of effects in this region in the sections dealing with acid deposition.)

Figure C-18. RADM-Predicted Increase in Visibility Degradation, Expressed in Annual Average DeciView, for Poor Visibility Conditions (90th Percentile) Under the No-control Scenario.



The differences in modeled 1990 control and no-control scenario visibility conditions in the 30 southwestern U.S. urban areas projected by linear rollback modeling are presented in Table C-17. When reviewing these visibility degradation differentials for the 30 southwestern U.S. urban areas, it is important to consider that while estimated differences in visual range were in many cases very large, changes in the DeciView Haze Index (dV) may be relatively small. This is because the perception of visibility degradation measured by dV may be small when baseline visibility is high.[28] Even so, the results indicate that, by 1990, visibility in southwestern U.S. urban areas would be noticeably worse under the no-control scenario.

Key caveats and uncertainties for visibility

There are several sources of uncertainty in the RADM and southwestern U.S. linear scaling model analyses. For RADM, the use of nitric acid as a surrogate for estimating changes in light-attenuating nitrate particles ignores the interaction effects of nitrates, sulfates, and ammonia. As a result, increases in nitrates may be overestimated by the model when both sulfates and nitric acid increase. However, the significance of this potential overestimation is mitigated to some extent by the relative insignificance of nitrate-related visibility degradation relative to sulfates which prevails in the eastern U.S.

Several important uncertainties in the southwestern U.S. urban area visibility analysis are described in detail in the SAI SW Visibility Report (1994). First, the need to use seasonal average conditions leads to underestimation of extreme visibility impairment episodes associated with high humidity, since particle growth due to water absorption is highly nonlinear. Second, although the use of city-specific light extinction and PM speciation data is significantly better than reliance on regional averages, uncertainties in city-specific data may contribute to overall uncertainty in the estimates. However, overall uncertainty associated with these factors will be reduced to some extent since overestimation of visibility degradation in some cities will be offset by underestimations in other cities. Finally, the linear scaling used to estimate the pre-1990 control scenarios and the no-control scenarios was based on changes in county-wide or air basin emissions. Uncertainties associated with apportionment of state-wide emission changes to individual counties or air basins may contribute significantly to overall uncertainty in the visibility change estimates. Such apportionment is particularly difficult for SO_x emission changes, since emission reductions achieved by the Clean Air Act tended to be at relatively remote utility and smelter plants. However, sulfates are a relatively minor source of light attenuation in western urban areas.

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[28] See SAI SW Visibility Report (1994), page 5-3.

An important overall limitation of the visibility analysis conducted for the present study is that only southwestern urban areas and the eastern 31 states were included. The Clean Air Act may have contributed toward significant reductions in visibility degradation in other areas. For example, Clean Air Act programs to reduce ambient particulate matter may have motivated reductions in silvicultural burning in some northwestern states. Perhaps the greatest deficiency in geographic coverage by the present study is the omission of visibility changes in Class I areas in the west.

Table C-17. Summary of Relative Change in Visual Range and DeciView Between 1990 Control and No-control Scenario Visibility Conditions for 30 Southwestern U.S. Cities.

City	Visual Range (%)	DeciView (dV)
Los Angeles, CA	69	-5
San Bernardino, CA	67	-5
Riverside, CA	65	-5
Anaheim, CA	68	-5
Ventura, CA	72	-5
San Diego, CA	65	-5
Santa Barbara, CA	62	-5
Bakersfield, CA	66	-5
Fresno, CA	65	-5
Modesto, CA	61	-5
Stockton, CA	62	-5
San Francisco, CA	63	-5
Oakland, CA	61	-5
San Jose, CA	64	-5
Monterey, CA	67	-5
Sacramento, CA	59	-5
Redding, CA	55	-4
Reno, NV	183	-10
Las Vegas, NV	308	-14
Salt Lake City, UT	58	-5
Provo, UT	48	-4
Fort Collins, CO	137	-9
Greeley, CO	39	-3
Denver, CO	85	-6
Colorado Springs, CO	111	-7
Pueblo, CO	240	-12
Albuquerque, NM	93	-7
El Paso, TX	153	-9
Tucson, AZ	218	-12
Phoenix, AZ	243	-12

Data Source: SAI SW Visibility Report (1994).

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Appendix D: Human Health and Welfare Effects of Criteria Pollutants

Introduction

In responding to the mandate of section 812, EPA conducted a comprehensive benefits analysis to identify and estimate the quantifiable health and welfare benefits enjoyed by Americans due to improved air quality resulting from the CAA. Health benefits resulted from avoidance of air pollution-related health effects, such as mortality, respiratory illness, and heart disease. Welfare benefits accrued where improved air quality averted damage to ecological health and measurable resources, such as agricultural production, building materials, and visibility.

This appendix presents an overview of EPA's approach for modeling human health and welfare effects. It provides an outline of the principles used to guide the benefits analysis, details methods used to quantify criteria air pollutant exposure nationwide across the study period (1970 to 1990), and discusses several critical conceptual and implementation issues for using health and welfare effect information. Modeling results, estimates of avoided incidences of adverse health and welfare effects, are then presented. Ecological and agricultural benefits are examined in more detail in Appendices E and F, respectively. Appendix I details the approach used to translate health and welfare effects into monetary benefits.

Principles for the Section 812 Benefits Analysis

Estimating the effects of even modest shifts in environmental releases involves complex chemical, environmental, biological, psychological and economic processes. The task of estimating the broad changes associated with adoption and implementation of the Clean Air Act challenges the limits of scientific knowledge and modeling capability to synthesize available information and techniques into a practical framework. A pragmatic plan for a comprehensive assessment must fairly reflect the complexities and uncertainties, but still produce a policy-relevant analysis in a timely fashion. In order to achieve this ambitious goal, the following principles have been used to guide the section 812 benefits assessment.

Comprehensiveness: The assessment should include as many benefit categories as are reasonably believed to be affected by implementation of the Clean Air Act. Comprehensiveness requires assessing effects with which greater levels of scientific confidence are associated, as well as less well-understood effects. The degree of relative certainty among effects must be carefully described in order to fairly present a broad portrayal of the physical and social benefits accruing to the nation from implementing the Act. In addition, section 812 of the 1990 CAA Amendments explicitly directs a comprehensive benefits coverage that prohibits a default assumption of zero value for identified benefits unless a zero value is supported by specific data.

Quantification Where Feasible: The central goal of the present study is to evaluate and compare the benefits and costs of historical CAA-related programs. Effective comparison of the variety of human health, welfare, and ecological benefits with the associated compliance costs requires that these consequences be measured in terms of a common metric. Expressing the value of these various effects in economic terms is the most efficient way to accomplish this objective, and is consistent with standard practices associated with economic benefit-cost analysis. Expressing these effects in economic terms requires quantifying and presenting estimated effects in both physical and monetized economic terms. Pursuant to this paradigm, the emphasis in the present study is largely on categories having direct and perceptible effects on human health. That is, the emphasis of the analysis is on categories such as symptoms and diseases rather than on physical changes (such as cell level changes) that do not directly result in a decreased health status noticeable to the individual.

Efficient Use of Previous Research Results: Significant research effort has been spent to understand and quantify the complex relationships between air pollution and human health. The present study has relied as much as possible on available research results, making adjustments as necessary to apply the existing results to the current analysis.

Incorporate Uncertainty: To properly convey the results of any benefits assessment, it is important to include an evaluation and characterization of how much confidence the analysts have in the estimates. Ideally this would include a formal quantitative assessment of the potential for error, and the sources, directions, and potential significance of any resultant biases. A method for considering and reporting uncertainty must be built into the fundamental design of the assessment. Such a framework was developed and applied in the present study, and was supplemented where necessary by expert judgment regarding the sources and potential significance of errors in each analytical step.

General Modeling Approach

Consistent with these principles, the EPA developed an approach for quantifying the effects of reduced pollutant exposure, with particular focus on those effect categories for which monetary benefits could be estimated. As described previously, the study design adopted for the section 812 assessment links a sequence of analytical models. The macroeconomic modeling (Appendix A) estimated economy-wide effects of CAA expenditures. These effects provided a basis for the modeling of criteria pollutant emissions under the two scenarios considered (the factual control scenario and the hypothetical no-control scenario), as documented in Appendix B. The emissions estimates were used as input to the air quality models (Appendix C). Ambient pollutant concentrations estimated by the air quality models were used as inputs to the health and welfare benefits model, the focus of this appendix.

The approach developed to model health and welfare benefits is known as a "reduced form" or "embedded model" approach. The concept of a reduced form model is to use simplified versions of previously constructed complex models to characterize the impact of a series of linked physical and socioeconomic processes. The health and welfare benefits model is characterized as a reduced form model because it relies on *summaries* of the data output from the air quality models, which rely on emissions summaries and summaries of macroeconomic conditions, successively. Although results of the independent models are used in series, the models themselves have not been integrated into the health and welfare benefits model.

In general, the reduced form health and welfare benefits model relies on two fundamental inputs: (1) nationwide changes in pollutant exposures across the study period, and (2) the association between changes in exposure and expected changes in specific health and welfare effects. These inputs are discussed below.

Quantifying Changes in Pollutant Exposures

Estimating changes in pollutant exposures requires characterization of nationwide air quality improvements across the study period, as well as the populations exposed to the different levels of improvement.

Air Quality

As discussed in Appendix C, the section 812 analysis estimated ambient concentrations for both the control and no-control scenarios for the following pollutants and air quality parameters:

- Particulate matter, less than 10 microns in diameter (PM_{10})
- Ozone (O_3)
- Nitrogen dioxide (NO_2)
- Sulfur dioxide (SO_2)
- Carbon monoxide (CO)
- Visibility measures (light extinction and DeciView)[1]
- Lead (Pb)

Generally, this analysis adopted actual historical air pollution monitoring data to represent control scenario air quality. No-control scenario profiles were derived by running the control and no-control scenario emissions inventories through a suite of air quality models and then using the differences in these modeled outcomes to adjust the historical profiles. Since lead was treated differently than the other pollutants, the analysis of the CAA impacts on atmospheric lead concentrations is documented in Appendix G.

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[1] While the visibility measures listed are not criteria air pollutants, they provide important measures of a significant welfare effect resulting from air pollution, visibility degradation. Light extinction (which is related to DeciView, a haziness index) results from light scattered by fine particles in the atmosphere, especially sulfates and ammonium nitrates. As atmospheric concentrations of such particles increase, light is attenuated and visibility diminishes.

With respect to the distribution of air quality data across the two decades considered, it should be noted that both the number and location of monitors tracking air quality changed over time. Table D-1 depicts the number of monitors for each pollutant across the period of this analysis. The number of monitors generally increased throughout the 1970s and leveled off or declined at varying points during the 1980s, depending on the pollutant.

Table D-1. Criteria Air Pollutant Monitors in the U.S., 1970 - 1990.

	Pollutant				
Year	PM_{10}	O_3	NO_2	SO_2	CO
1970	245	1	43	86	82
1975	1,120	321	303	827	494
1980	1,131	546	375	1,088	511
1985	970	527	305	916	458
1990	720	627	345	753	493

For the section 812 modeling, the non-lead pollutants have been characterized as either county-level or monitor-level pollutants. The distinction was important for quantifying the population exposed to different levels of air quality improvements, as discussed below. PM_{10} is considered a county-level pollutant, since historical concentrations in monitored counties have been synthesized into a single concentration for each county.[2] In contrast, O_3, NO_2, NO, SO_2, and CO were reported at specific monitor locations, given by latitude/longitude coordinates. Finally, visibility was treated as a county-level pollutant in the western U.S. and a monitor-level pollutant in the eastern U.S.[3] Air quality data for PM_{10} and ozone were reported for each year of the study period; data for the remaining pollutants were reported only for 1975, 1980, 1985, and 1990.

In order to reduce the volume of air quality data necessary to describe pollutant concentrations for two scenarios nationwide over twenty years, annual concentration profiles were reduced to frequency distributions. That is, annual pollutant concentrations for a variety of averaging times (e.g., 1-hour, 6-hour, daily) were summarized as a distribution of values across the year. This approach reduced data management requirements significantly, while adequately capturing air quality improvements between the control and no-control scenarios.

Population Distribution

Health and some welfare benefits resulting from air quality improvements are distributed to populations in proportion to the reduction in exposure each enjoys. Predicting population exposures, then, is a necessary step in estimating health effects. Doing so for the section 812 analysis required not only an understanding of where air quality improved as a result of the CAA, but also how many individuals were affected by varying levels of air quality improvements. Thus, a critical component of the benefits analysis required that the distribution of the U.S. population nationwide be described in a manner compatible with the air quality data. Described below is the method used to allocate U.S. Census data to a symmetrical grid overlying the country.

Census Data

Three years of U.S. Census data were used to represent the geographical distribution of U.S. residents: 1970, 1980, and 1990. Population data were supplied at the census block group level, with approximately

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[2] Two different measures of ambient concentrations of particulate matter were used in the United States during the period 1970 to 1990. Prior to 1987, the indicator for the National Ambient Air Quality Standard for PM was total suspended particulates (TSP). In 1987, the indicator was changed to PM_{10} (particles less than 10 µM in diameter). Widespread PM_{10} monitoring did not begin until 1985; prior to that only TSP data is available. Because the recent scientific literature reports primarily the relationship between PM_{10} and adverse health and welfare effects, PM_{10} data is preferred, if available. Where only TSP is available, PM_{10} concentrations were estimated using PM_{10}:TSP ratios that vary by area of the country and the urban/rural characterization of the area.

[3] In the western U.S., visibility was modeled using a linear-rollback model and extinction budget approach for 30 major urban centers (SAI, 1994). The modeling results, reported in DeciView, were applied to the counties in the vicinity of the urban centers and considered to share a common air basin. In the eastern U.S., Regional Acid Deposition Model (RADM) runs provided visibility estimates in terms of light extinction coefficients. These were modeled across a 60 km. X 60 km. grid, approximately covering the eastern half of the country. Since the extinction coefficients were reported at the grid cell centroids, for which the coordinates were known, visibility in the east was treated as a monitor-level pollutant.

290,000 block groups nationwide. Allocating air quality improvements to the population during intermediate years necessitated interpolation of the three years of population data. Linear interpolation was performed at the block group level in order to preserve the variability in growth rates throughout the country.

Gridding U.S. Population

To ease computational burden, block group population estimates were aggregated to a rectangular grid structure. The grid, comprised of ten kilometer by ten kilometer gridcells, spanned the entire area of the contentional United States. This grid size generated 46,885 populated gridcells throughout the U.S.

The entire population of each block group was assumed to reside at the geographical centroid of the block group area, the coordinates of which were available from the U.S. Bureau of the Census. Block group populations were aggregated to gridcells according to the block group centroids encompassed by each cell. In addition to the population of each gridcell, the state and county names for each gridcell were retained, permitting aggregation of data at the state and county level, as well as nationwide.

Allocating Exposure Estimates to the Population

Two alternative modeling strategies were used to allocate air quality improvements to the U.S. population. They differed in terms of both the certainty of the estimates and the geographic coverage:

Table D-2. Population Coverage in the "Within 50 km" Model Runs (percent of continental U.S. population).

	1975	1980	1985	1990
CO	67.4%	67.9%	68.4%	70.4%
EXT	73.2%	72.3%	72.3%	72.2%
NO_2	53.3%	58.8%	60.8%	61.5%
O_3	55.5%	70.5%	71.5%	74.4%
PM_{10}	78.5%	79.5%	75.8%	67.8%
SO_2	64.7%	73.3%	73.0%	70.6%
Pb	100%	100%	100%	100%

Method One

Air quality improvements (difference between control and no-control scenarios) were applied to individuals living in the vicinity of air quality monitors. For pollutants with monitor-level data, it was assumed that the individuals in a gridcell were exposed to air quality changes estimated at the nearest monitor, as long as the monitor was within 50 kilometers. Likewise, for PM_{10} (for which data was available at the county level) the population of each monitored county was assumed to be exposed to the air quality changes reported for that county.[4] The remainder of the population was excluded from the analysis.

Unfortunately, by limiting the quantitative analysis to populations within 50 km of a monitor (or within a monitored county, for PM), a significant portion of the U.S. population was left out of the analysis (see Table D-2). For most pollutants in most years (excepting lead), less than three-quarters of the population lived within 50 km of a monitor (or within a PM-monitored county). Clearly, an analysis that excluded 25 percent of the population from the benefits calculations (thus implicitly assuming that the CAA had no impact on that population) would understate the physical effects of the CAA. Conversely, ascribing air pollution reduction benefits to persons living great distances from air quality monitors is a speculative exercise, and could overstate benefits.

Method Two

As an alternative modeling strategy, air quality improvements were applied to almost all individuals nationwide. Where monitor data were not available within 50 kilometers, data from the closest monitor, regardless of distance, were used. Similarly, PM_{10} concentrations were extrapolated using regional air quality models to all counties (even those for which monitoring data was unavailable) and applied to the populations of those counties.

Although subject to less certain air quality data, the second alternative extrapolates pollutant exposure estimates to almost the entire population using the closest monitoring data available (see Table D-3).[5] This second alternative was chosen as the preferred approach in the benefits analysis. The sensitivity of the benefits estimate to the extrapolation of air quality data beyond monitored areas is explored in Appendix I.

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[4] Since the lead (Pb) analysis, which was handled separately from that of the other criteria pollutants, did not require air quality modeling data, the issue of proximity to monitors is irrelevant. The Pb analysis extended to 100 percent of the population.

[5] While this alternative captures the vast majority of the U.S. population, it does not model exposure for everyone. To improve computational efficiency, those gridcells with populations less than 1,000 were not modeled; these cells account for less than five percent of the U.S. population.

Table D-3. Population Coverage for "Extrapolated to All U.S." Model Runs (percent of continental U.S. population).

	1975	1980	1985	1990
CO	97.2%	97.2%	98.7%	100.0%
EXT	75.6%	74.8%	74.7%	74.7%
NO_2	97.2%	97.2%	98.7%	100.0%
O_3	96.6%	97.2%	98.7%	100.0%
PM_{10}	95.9%	95.8%	97.2%	98.5%
SO_2	95.4%	95.6%	97.0%	98.4%
Pb	100%	100%	100%	100%

Estimating Human Health Effects of Exposure

It is impossible to estimate all of the physical effects that would have occurred without the Clean Air Act. While scientific information is available that makes it possible to estimate certain effects, many other, potentially very important, health and welfare effects cannot be estimated at this time. Other physical effects can be quantified, but it is impossible to assess the economic value of those endpoints based on the current economics literature. Table D-4 shows the health and welfare effects for which quantitative analysis has been prepared, as well as some of the health effects that have not been quantified in the analysis.

In order to translate the reductions in pollutant exposure estimated to result from the CAA into health benefits, it is necessary to quantify the relationship between such exposures and adverse health effects. As indicated below, this analysis relies on concentration-response relationships published in the scientific literature which provide estimates of the number of fewer individuals that incur an adverse health effect per unit change in air quality. Such relationships are combined with the air quality improvement and population distribution data to estimate changes in the incidence of each health endpoint. By evaluating each concentration-response function for every gridcell throughout the country, and aggregating the resulting incidence estimates, it was possible to generate national estimates of avoided incidence.

It should be noted that a slightly different approach was used to compute health effects associated with exposure to gasoline lead. Instead of relating health outcomes to ambient pollutant concentrations, the concentration-response functions for lead-induced effects link changes in health effects directly to changes in the population's mean blood lead level. This value is directly related to the concentration of lead in gasoline in a particular year. Appendix G documents both the methods used to characterize mean blood lead levels and the approach for estimating human health effects from lead exposure.

The discussion below outlines the types of health studies considered for this analysis, and issues critical to selecting specific studies appropriate for use in the section 812 context. Next, details regarding use of the results of the studies are explored. Finally, the concentration-response functions used to model health benefits from reductions in non-lead criteria pollutants are outlined.

Types of Health Studies

Scientific research about air pollution's adverse health impacts uses a broad array of methods and procedures. The research methods used to investigate the health effects of air pollution have become considerably more sophisticated over time, and will continue to evolve in the future. This progress is the result of better available research techniques and data, and the ability to focus further research more sharply on key remaining issues based on the contributions of earlier work.

The available health effects studies that could potentially be used as the basis of the section 812 assessment are categorized into epidemiology studies and human clinical studies. Epidemiological research in air pollution investigates the association between exposure to air pollution and observed health effects in the study population. Human clinical studies involve examination of human responses to controlled conditions in a laboratory setting. Research has been conducted on health effects from exposure to pollution using each approach, and studies using these techniques have been considered in various formal regulatory proceedings. Each type of study (as it is used

Table D-4. Human Health Effects of Criteria Pollutants.

Pollutant	Quantified Health Effects	Unquantified Health Effects	Other Possible Effects
Ozone	Mortality* Respiratory symptoms Minor restricted activity days Respiratory restricted activity days Hospital admissions Asthma attacks Changes in pulmonary function Chronic Sinusitis & Hay Fever	Increased airway responsiveness to stimuli Centroacinar fibrosis Inflammation in the lung	Immunologic changes Chronic respiratory diseases Extrapulmonary effects (e.g., changes in structure, function of other organs)
Particulate Matter/ TSP/ Sulfates	Mortality* Bronchitis - Chronic and Acute Hospital admissions Lower respiratory illness Upper respiratory illness Chest illness Respiratory symptoms Minor restricted activity days All restricted activity days Days of work loss Moderate or worse asthma status (asthmatics)	Changes in pulmonary function	Chronic respiratory diseases other than chronic bronchitis Inflammation in the lung
Carbon Monoxide	Hospital Admissions - congestive heart failure Decreased time to onset of angina	Behavioral effects Other hospital admissions	Other cardiovascular effects Developmental effects
Nitrogen Oxides	Respiratory illness	Increased airway responsiveness	Decreased pulmonary function Inflammation in the lung Immunological changes
Sulfur Dioxide	In exercising asthmatics: Changes in pulmonary function Respiratory symptoms Combined responses of respiratory symptoms and pulmonary function changes		Respiratory symptoms in non-asthmatics Hospital admissions
Lead	Mortality Hypertension Non-fatal coronary heart disease Non-fatal strokes IQ loss effect on lifetime earnings IQ loss effects on special education needs	Health effects for individuals in age ranges other than those studied Neurobehavioral function Other cardiovascular diseases Reproductive effects Fetal effects from maternal exposure Delinquent and anti-social behavior in children	

* This analysis estimates excess mortality using PM_{10} as an indicator of the pollutant mix to which individuals were exposed.

for air pollution research) is described below, and the relative strengths and weaknesses for the purposes of the section 812 assessment are examined.

Epidemiological Studies

Epidemiological studies evaluate the relationship between exposures to ambient air pollution and health effects in the human population, typically in a "natural" setting. Statistical techniques (typically variants of multivariate regression analysis) are used to estimate quantitative concentration-response (or exposure-response) relationships between pollution levels and health effects.

Epidemiology studies can examine many of the types of health effects that are difficult to study using a clinical approach. Epidemiological results are well-suited for quantitative benefit analyses because they provide a means to estimate the incidence of health effects related to varying levels of ambient air pollution without extensive further modeling effort. These estimated relationships implicitly take into account at least some of the complex real-world human activity patterns, spatial and temporal distributions of air pollution, synergistic effects of multiple pollutants and other risk factors, and compensating or mitigating behavior by the subject population. Suspected relationships between air pollution and the effects of both long-term and short-term exposure can be investigated using an epidemiological approach. In addition, observable health endpoints are measured, unlike clinical studies which often monitor endpoints that do not result in observable health effects (e.g. forced expiratory volume). Thus, from the point of view of conducting a benefits analysis, the results of epidemiological studies, combined with measures of ambient pollution levels and the size of the relevant population, provide all the essential components for associating measures of ambient air pollution and health status for a population in the airshed being monitored.

Two types of epidemiological studies are considered for dose-response modeling: individual level cohort studies and population level ecological studies. Cohort-based studies track individuals that are initially disease-free over a certain period of time, with periodic evaluation of the individuals' health status. Studies about relatively rare events such as cancer incidence or mortality can require tracking the individuals over a long period of time, while more common events (e.g., respiratory symptoms) occur with sufficient frequency to evaluate the relationship over a much shorter time period. An important feature of cohort studies is that information is known about each individual, including other potential variables correlated to disease state. These variables, called confounders, are important to identify because if they are not accounted for in the study they may produce a spurious association between air pollution and health effect.

A second type of study used in this analysis is a population-level ecological study. The relationship between population-wide health information (such as counts for daily mortality, hospital admissions, or emergency room visits) and ambient levels of air pollution are evaluated. One particular type of ecological study, time-series, has been used frequently in air-pollution research. An advantage of the time-series design is that it allows "the population to serve as its own control" with regard to certain factors such as race and gender. Other factors that change over time (tobacco, alcohol and illicit drug use, access to health care, employment, and nutrition) can also affect health. However, since such potential confounding factors are unlikely to vary over time in the same manner as air pollution levels, or to vary over periods of months to several years in a given community, these factors are unlikely to affect the magnitude of the association between air pollution and variations in short-term human health responses.

Drawbacks to epidemiological methods include difficulties associated with adequately characterizing exposure, measurement errors in the explanatory variables, the influence of unmeasured variables, and correlations between the pollution variables of concern and both the included and omitted variables. These can potentially lead to spurious conclusions. However, epidemiological studies involve a large number of people and do not suffer extrapolation problems common to clinical studies of limited numbers of people from selected population subgroups.

Human Clinical Studies

Clinical studies of air pollution involve exposing human subjects to various levels of air pollution in a carefully controlled and monitored laboratory situation. The physical condition of the subjects is measured before, during and after the pollution exposure. Physical condition measurements can include general biomedical information (e.g., pulse rate and blood pressure), physiological effects specifically affected by the pollutant (e.g., lung function), the onset of symptoms (e.g., wheezing or chest pain), or the ability of the individual to perform specific physical or cognitive tasks (e.g., maximum sustainable speed on a treadmill). These studies often involve exposing the individuals to pollutants while exercising, increasing the amount of pollutants that are actually introduced into the lungs.

Clinical studies can isolate cause-effect relationships between pollutants and certain human health effects. Repeated experiments altering the pollutant level, exercise regime duration and types of participants can potentially identify effect thresholds, the impact of recovery (rest) periods, and the differences in response among population groups. While cost considerations tend to limit the number of participants and experimental variants examined in a single study, clinical studies can follow rigorous laboratory scientific protocols, such as the use of placebos (clean air) to establish a baseline level of effects and precise measurement of certain health effects of concern.

There are drawbacks to using clinical studies as the basis for a comprehensive benefits analysis. Clinical studies are appropriate for examining acute symptoms caused by short-term exposure to a pollutant. While this permits examination of some important health effects from air pollution, such as bronchoconstriction in asthmatic individuals caused by sulfur dioxide, it excludes studying more severe effects or effects caused by long term exposure. Another drawback is that health effects measured in some well-designed clinical studies are selected on the basis of the ability to measure precisely the effect, for example forced expiratory volume, rather than a larger symptom. The impact of some clinically measurable but reversible health effects such as lung function on future medical condition or lifestyle changes are not well understood.

Ethical limits on experiments involving humans also impose important limits to the potential scope of clinical research. Chronic effects cannot be investigated because people cannot be kept in controlled conditions for an extended period of time, and because these effects are generally irreversible. Participation is generally restricted to healthy subjects, or at least to exclude people with substantial health conditions that compromise their safe inclusion in the study. This can cause clinical studies to avoid providing direct evidence about populations of most concern, such as people who already have serious respiratory diseases. Ethical considerations also limit the exposures to relatively modest exposure levels, and to examining only mild health effects that do no permanent damage. Obviously for ethical reasons human clinical evidence cannot be obtained on the possible relationship between pollution and mortality, heart attack or stroke, or cancer.

One potential obstacle to using dose-response information from clinical research methods in a benefits assessment is the need for an exposure model. The dose-response functions developed from clinical research are specific to the population participating in the study and the exposure conditions used in the laboratory setting. It is therefore difficult to extrapolate results from clinical settings to daily exposures faced by the whole population. For example, many clinical studies evaluate effects on exercising individuals. Only a small portion of the population engages in strenuous activity (manual labor or exercise) at any time. Reflecting these fundamental differences between the laboratory setting and the "real world" imposes a formidable burden on researchers to provide information about human activity patterns, exercise levels, and pollution levels. This requirement adds an additional step in the analytical process, introducing another source of uncertainty and possible error.

To apply the clinical results to model the general population, two decisions must be made. First, how far can the conditions in the clinical setting be expanded? For example, if the subjects in the clinical study were healthy male college students, should the results be applied to the entire population, including children? Second, how many people in the general population are exposed to conditions similar to those used in the clinical setting? Frequently, clinical studies are conducted at relatively high exercise levels (increasing the dose, or the quantity of pollutants actually delivered to the lungs). In the general population few people experience these conditions very often, and people do not reach these exercise levels with equal frequencies during the day and night.

In addition, the analyst must determine the number of people that are exposed to the levels of ambient conditions seen in the laboratory. Air quality varies throughout a city and is typically reported by data from monitors located at various places throughout the city. However, people are not exposed to the conditions at any one monitor all day. As people move around in the city, they are exposed to ambient air quality conditions represented by different monitors at different times during the day. To further compound the problem, air quality also varies between indoors and outdoors, within a car or garage, and by such factors as proximity to a roadway or major pollution source (or sink). The exposure model must account for the ambient conditions in the "microenvironments" that the population actually experiences.

The issues of study subjects, exercise and microenvironments can influence the choice of clinical studies selected for the section 812 assessment. Clinical studies that use exposure regimes and exercise levels more similar to what larger groups of the population see are easier to apply in a benefits model than are more narrow studies. Similarly, studies that use a diverse group of subjects are easier to apply to the general population than are more narrow studies.

Given the major advantages of epidemiological studies—exposures do not need to be modeled and health effects are observed in a large, more heterogeneous population—epidemiological studies are used as the basis for determining the majority of health effects and dose-response curves. The diverse activity patterns, microenvironments, and pollution levels are already considered in the aggregate through the concentration-response functions derived from epidemiological studies. Clinical studies are used if there are health effects observed in clinical studies not observed in epidemiological studies.

Issues in Selecting Studies To Estimate Health Effects

A number of issues arise when selecting and linking the individual components of a comprehensive benefits analysis. The appropriate procedure for handling each issue must be decided within the context of the current analytical needs, considering the broader analytical framework. While more sophisticated or robust studies may be available in some circumstances, the potential impact on the overall analysis may make using a simpler, more tractable approach the pragmatic choice. In considering the overall impact of selecting a study for use in the section 812 assessment, important factors to consider include the likely magnitude the decision will have on the overall analysis, the balance between the overall level of analytical rigor and comprehensiveness in separate pieces of the analysis, and the effect on the scientific defensibility of the overall project.

This section discusses ten critical issues in selecting health information for use in the section 812 assessment: use of peer-reviewed research, confounding factors, uncertainty, the magnitude of exposure, duration of exposure, threshold concentrations, the target population, statistical significance of relationships, relative risks, and the need for baseline incidence data. The previous discussion about the types of research methods available for the health information alluded to some of these issues, as they are potentially important factors in selecting between studies using different methods. Other issues address how scientific research is used in the overall analytical framework.

Peer-Review of Research

Whenever possible, peer reviewed research rather than unpublished information has been relied upon. Research that has been reviewed by the EPA’s own peer review processes, such as review by the Clean Air Science Advisory Committee (CASAC) of the Science Advisory Board (SAB), has been used whenever possible. Research reviewed by other public scientific peer review processes such as the National Academy of Science, the National Acidic Precipitation Assessment Program, and the Health Effects Institute is also included in this category.

Research published in peer reviewed journals but not reviewed by CASAC has also been considered for use in the section 812 assessment, and has been used if it is determined to be the most appropriate available study. Research accepted for publication by peer reviewed journals (“in press”) has been considered to have been published. Indications that EPA intends to submit research to the CASAC (such as inclusion in a draft Criteria Document or Staff Paper) provide further evidence that the journal-published research should be used.

Air pollution health research is a very active field of scientific inquiry, and new results are being produced constantly. Many research findings are first released in University Working Papers, dissertations, government reports, non-reviewed journals and conference proceedings. Some research is published in abstract form in journals, which does not require peer review. In order to use the most recent research findings and be as comprehensive as possible, unpublished research was examined for possible use in the section 812 assessment. Any unpublished research used is carefully identified in the report, and treated as having a higher degree of uncertainty than published results. The peer review of the section 812 assessment by the Advisory Council on Clean Air Compliance Analysis provides one review process for all components of the assessment, as well as for the way in which the components have been used.

Confounding Factors

Confounding can occur when the real cause of disease is associated with a number of factors. If only one contributing factor is evaluated in an epidemiological study, a false association may occur. For example, in epidemiology studies of air pollution, it is important to take into account weather conditions, because weather is associated with both air pollution and health outcomes. If only air pollution is evaluated, a false association between air pollution and health could result; one may incorrectly assume that a reduction in air pollution is exclusively responsible for a reduction in a health outcome. Potential confounders include weather-related variables, age and gender mix of the subject population, and pollution emissions other than those being studied. Studies that control for a broad range of likely confounders can offer a more robust conclusion about an individual pollutant, even if the statistical confidence interval is larger due to the inclusion of more variables in the analysis.

In many cases, several pollutants in a "pollutant mix" are correlated with each other—that is, they tend to occur simultaneously. Therefore, although there may be an association between a health effect and each of several pollutants in the mix, it may not be clear which pollutant is causally related to the health effect (or whether more than one pollutant is causally related). This analysis includes epidemiological modeling of the health effects that have been associated with exposure to a number of pollutants. In most cases where the health effect is being modeled for the several correlated pollutants of interest, regression coefficients based on PM as a surrogate for the mixture were chosen in preference to multiple pollutant models and single pollutant models. The most important example of this occurs in estimating mortality effects. There is substantial evidence that exposure to criteria pollutants, either individually or collectively, is significantly associated with excess mortality. Generally, this association is related to particulate matter. Therefore, even though particulate matter cannot be shown to be the sole pollutant causing pollution-related excess mortality, it can be used as an indicator of the pollutant mixture which appears to result in excess mortality. This analysis estimates excess mortality (for all criteria pollutants other than lead) using PM as an indicator of the pollutant mix to which individuals were exposed. This issue is discussed further below, where details on estimating mortality effects are explored.

The one exception to the use of single pollutant regression models is estimating hospital admissions. Both PM and ozone are generally found to have a statistically significant and separate association with hospital admissions. Using separate regressions (from single pollutant models) for each pollutant may overstate the number of effects caused by each pollutant alone. On the other hand, using PM as a single indicator of the pollutant mix could underestimate the total hospital admissions caused by different mechanisms. Separate PM and ozone coefficients for hospital admissions are selected from regression models that consider the effects of both pollutants simultaneously.

Uncertainty

The stated goal of the section 812 assessment is to provide a comprehensive estimate of benefits of the Clean Air Act. To achieve this goal, information with very different levels of confidence must be used. Benefit categories are not to be omitted simply because they are highly uncertain or controversial, but those benefit categories that are reasonably well understood must be distinguished from those which are more tentative.

The ideal approach to characterizing uncertainty is to conduct a formal quantitative uncertainty analysis. A common approach develops an estimated probability distribution for each component of the analysis. A Monte Carlo procedure draws randomly from each of these distributions to generate an estimate of the result. Evaluating the result for many such random combinations, creates a distribution of results that reflects the joint uncertainties in the analysis.

The most serious obstacle to preparing a formal quantitative uncertainty analysis is identifying all the necessary distributions for each component of the analysis. The Monte Carlo procedure requires that all components of the model be rerun many times. However, the section 812 project links the outputs from independent modeling activities. It would be impractical to simultaneously rerun the macroeconomic, emissions, air quality, and exposure models because of the diverse origins of the models. Therefore, instead of a complete formal uncertainty analysis, the section 812 assessment includes a less rigorous analysis of the inherent uncertainties in the modeling effort. The uncertainty analysis combines quantitative and qualitative elements designed to sufficiently describe the implications of the uncertainties. A primary goal of the sensitivity/uncertainty analysis is to identify the health effects that make a sizable contribution to the overall assessment of the monetary benefits. There may be situations where there are significant differences in the available information used to predict the incidence of a particular health effect (i.e., the uncertainty bounds are large). It is important to alert the reader to situations where using the lower incidence estimates may portray the health effect as only modestly contributing to the overall total benefits, but using reasonable alternative higher estimated incidence figures (or higher monetized values) would substantially impact not only the monetized value of the individual health effect, but actually make a noticeable difference in the total benefits assessment.

Consideration of the overall uncertainties inherent in the section 812 assessment has several important implications for health study selection. It was important to carefully examine the balance between the level of uncertainties in the analysis and the need for comprehensive coverage of all benefit categories. There were frequently situations in which a direct tradeoff existed between more comprehensive coverage and the restriction of the analysis to more certain information. Also, the relationship between the uncertainty in other parts of the analysis and the uncertainty for each particular health effect was carefully considered.

Magnitude of Exposure

One component of the section 812 analysis estimates the air pollution levels that would have occurred in the absence of the Clean Air Act. These estimates are larger than currently observed levels of U.S. air pollution, and perhaps even levels currently observed elsewhere in the world. This aspect of the analysis poses difficulties for the application of concentration-response functions that have been based on exposures at much lower pollution levels. The shape of the concentration-response function much above observed exposures levels is unknown. It is possible that biological mechanisms affecting response that are unimportant at low levels of exposure may dominate the form of response at higher levels, introducing nonlinearity to the mathematical relationship. In general, studies that include exposure levels spanning the range of interest in the section 812 assessment are preferable to studies at levels outside of the range, or that only include a narrow part of the range. A possible drawback to this approach is that studies which fit this criterion have often been conducted outside the U.S. The application of foreign studies to U.S. populations introduces additional uncertainties regarding the representativeness of the exposed population and the relative composition of the air pollution mix for which the single pollutant is an indicator. These difficult issues were considered in selecting studies for the benefits analysis.

Duration of Exposure

Selection of health studies for the section 812 assessment must consider the need to match the health information to the air quality modeling conducted for the assessment. For example, information on the health effects from short term (five minute) exposure to sulfur dioxide cannot be readily combined with information on average daily sulfur dioxide levels. In selecting studies for the benefits analysis, preference was shown for studies whose duration of exposure matched one of the averaging times of the air quality data.

Thresholds

Exposure-response relationships are conceptualized as either exhibiting a threshold of exposure below which adverse effects are not expected to occur, or as having no response threshold, where any exposure level theoretically poses a non-zero risk of response to at least one segment of the population. The methods employed by health researchers to characterize exposure-response relationships may or may not explicitly analyze the data for the existence of a threshold. Studies may analyze relationships between health and air pollution without considering a threshold. If a threshold for population risk exists but is not identified by researchers, then Clean Air Act benefits could be overestimated if CAA levels are below the threshold, because the risk reduction from the no-control scenario could be overstated. On the other hand, if a threshold is artificially imposed where one does not exist, the relative benefits of the Clean Air Act may be underestimated. In general, those studies that explicitly consider the question of a threshold (whether a threshold is identified or not) provide stronger evidence; consideration of this question is a positive feature when selecting studies for this analysis.

Target Population

Many of the studies relevant to quantifying the benefits of air pollution reductions have focused on specific sensitive subpopulations suspected to be most susceptible to the effects of the pollutant. Some of these effects may be relevant only for the studied subpopulation; effects on other individuals are either unknown, or not expected to occur. For such studies, the challenge of the analysis is to identify the size and characteristics of the subpopulation and match its occurrence to exposure. Other studies have examined specific cohorts who may be less susceptible than the general population to health effects from air pollution (e.g., healthy workers), or who differ in age, gender, race, ethnicity or other relevant characteristics from the target population of the benefits analysis. Extrapolating results from studies on nonrepresentative subpopulations to the general population introduces uncertainties to the analysis, but the magnitude of the uncertainty and its direction are often unknown. Because of these uncertainties, benefit analyses often limit the application of the dose-response functions only to those subpopulations with the characteristics of the study population. While this approach has merit in minimizing uncertainty in the analysis, it can also severely underestimate benefits if, in fact, similar effects are likely to occur in other populations. For these reasons, studies that examine broad, representative populations are preferable to studies with narrower scope because they allow application of the functions to larger numbers of persons without introducing additional uncertainty.

Many studies included in the section 812 analysis focus on a particular age cohort of the population for the identification of health effects. The choice of age group is often a matter of convenience (e.g., extensive Medicare data may be available for the elderly population) and not because the effects are, in reality, restricted to the specific age group (even though their incidence may vary considerably over the life span). However, since no information is available about effects beyond the studied population, this analysis applies the given concentration-response relationships only to those age groups corresponding to the cohorts studied. Likewise, some studies were performed on individuals with specific occupations, activity patterns, or medical conditions because these traits relate to the likelihood of effect. In these cases, application of dose-response functions has been restricted to populations of individuals with these same characteristics.

Statistical Significance of Exposure-Response Relationships

The analysis includes as many studies related to a given health effect as possible, except for studies inapplicable to the current analysis. For some endpoints, the group of adequate studies yielded mixed results, with some showing statistically significant responses to pollutant concentrations and others with insignificant associations. Unless study methods have been judged inadequate, dose-response functions with both statistically significant and insignificant coefficients have been included to characterize the possible range of risk estimates. Excluding studies exclusively on the basis of significance could create an upward bias in the estimates by not reflecting research that indicates there is a small, or even zero, relationship between pollution and specific health effects. It should be noted, however, that some studies that found insignificant effects for a pollutant could not be used because they did not report the insignificant coefficient values.

In some cases, a single study reported results for multiple analyses, yielding both significant and nonsignificant results, depending on the nature of the input parameters (e.g., for different lag periods or concurrent exposures). In these cases, only significant results were included.

Relative Risks

Many studies reported only a relative risk value (defined as the ratio of the incidence of disease in two groups exposed to two different exposure levels). The analysis required conversion of these values to their corresponding regression coefficients when the coefficients were not reported. When converting the relative risk to a coefficient value, the analysis used the functional form of the regression equation reported by the authors of the study.

The coefficients from a number of studies measured the change in the number of health effects for the study population rather than a change per individual. These coefficients were divided by the size of the study population to obtain an estimate of change per individual. The coefficient could then be multiplied by the size of the population modeled in the current analysis to determine total incidence of health effects.

Baseline Incidence Data

Certain dose-response functions (those expressed as a change relative to baseline conditions) require baseline incidence data associated with ambient levels of pollutants. Incidence data necessary for the calculation of risk and benefits were obtained from national sources whenever possible, because these data are most applicable to a national assessment of benefits. The National Center for Health Statistics provided much of the information on national incidence rates. However, for some studies, the only available incidence information come from the studies themselves; in these cases, incidence in the study population is assumed to represent typical incidence nationally.

Studies were excluded if health endpoints could not be defined in the U.S. population. For example, in Pope and Dockery (1992) the authors developed a unique definition of symptomatic children in Utah which has no correlation in the incidence data bases which were available; consequently, the results could not be applied to the general population.

Estimating Mortality Effects

Using PM as an Indicator

There is substantial evidence that exposure to criteria pollutants, either individually or collectively, is significantly associated with excess mortality. This association is most closely and consistently related to the ambient air concentrations of PM.

Several studies have found small but statistically significant relationships between ozone and mortality, while other studies have not found a significant relationship. There is inconclusive evidence whether ozone has an effect independent of the effect of other pollutants (e.g., PM or CO), has a synergistic effect in combination with other effects, or is a confounder in the relationship between mortality and other pollutants. For example, in a recent study HEI (1996) found a significant and relatively stable ozone coefficient for most of the model specifications presented in the study. However, the measured ozone effect was largest and most significant in the winter and autumn, when ozone levels are low.

This analysis estimates excess mortality (for all criteria pollutants other than lead) using PM as an indicator of the pollutant mix to which individuals were exposed. Even if particulate matter exposure cannot be shown to be an independent causal factor of excess mortality, it is, at a minimum, a good indicator measure of the exposure to the pollutant mixture that has been shown to be related to excess mortality. Because PM is used as an indicator, the concentration-response functions from single pollutant models (i.e., statistical models including PM as the only pollutant) are preferred. To the extent that ozone is correlated with PM, the effect of ozone, either as an independent association or acting in combination with other pollutants, will be captured by this approach.

Estimating the Relationship Between PM and Premature Mortality

Long-term exposure versus short-term exposure studies and the degree of prematurity of mortality. Both long-term exposure (cohort) studies and short-term exposure (longitudinal or time-series) studies have estimated the relationship between exposure to PM and premature mortality. While there are advantages and disadvantages to each type of study (as discussed above), the long-term studies may capture more of the PM-related premature mortality, as well as premature mortality that is more premature, than the short-term studies.

The degree of prematurity of pollution-related death may be an important uncertainty in the effort to estimate the benefits of reducing pollution concentrations, as discussed in Appendix I. The willingness to pay to save a few days of life may be significantly less than the willingness to pay to save a few, or many, years of life. Evidence concerning the degree of prematurity of pollution-related death would, in this case, be crucial. Such evidence is, however, still scarce. There is some limited evidence that the relative risk of mortality from exposure to PM is higher for older individuals than for younger individuals. This, combined with the fact that the baseline incidence of mortality consists disproportionately of people 65 and over, suggests that PM-related mortality is disproportionately among older individuals. The extent to which prematurity of death among older individuals is on the order of days or weeks versus years, however, is more uncertain. The short-term exposure studies can provide little information on this. It is possible that premature deaths on high pollution days would have occurred only days later, if the individuals were sick and therefore particularly susceptible. The fact that the long-term exposure mortality studies found substantially larger relative risks, however, suggests that not all of the premature mortality is on the order of days or even weeks. Shortening of life of such a small duration would not be detectable in a long-term epidemiology study, ensuring that the effects detected in such studies must represent longer periods of life shortening. This suggests that at least some of the premature mortality associated with exposure to PM may reduce lifespans by substantially longer amounts of time.

Even if an individual's PM-related premature mortality is of very short duration, on the order of days, however, it may be misleading to characterize such a PM-related loss as only those few days if the individual's underlying susceptibility was itself exacerbated by chronic exposure to elevated levels of pollution. Suppose, for example, that long-term exposure to elevated PM levels compromises the cardiopulmonary system, making the individual more susceptible to mortality on peak PM days than he otherwise would have been. If this is the case, then the underlying susceptibility would itself be either caused by chronic exposure to elevated PM levels or exacerbated by it. Characterizing the individual's loss as a few days could, in this case, be a substantial underestimate.

In addition, the long-term studies estimate significantly more PM-related mortality than the annual sum of the daily estimates from the short-term studies, suggesting that the short-term studies may be missing a component of PM-related mortality that is being observed in the long-term studies. For example, if chronic exposure to elevated PM levels causes premature mortality that is not necessarily correlated with daily PM peak levels, this type of mortality would be detected in the long-term studies but not necessarily in the short-term studies. Two of the long-term exposure studies suggest, moreover, that the association between ambient air pollution and mortality cannot be explained by the confounding influences of smoking and other personal risk factors.

Uncertainties surround analyses based on epidemiological studies of PM and mortality. In addition to the uncertainty about the degree of prematurity of mortality, there are other uncertainties surrounding estimates based on epidemiological studies of PM and mortality. Although epidemiological studies are generally preferred to human clinical studies, there is nevertheless uncertainty associated with estimates of the risk of premature mortality (and morbidity) based on studies in the epidemiological literature. Considering all the epidemiological studies of PM and mortality, both short-term and long-term, there is significant interstudy variability as well as intrastudy uncertainty. Some of the difference among estimates reported by different studies may reflect only sampling error; some of the difference, however, may reflect actual differences in the concentration-response relationship from one location to another. The transferability of a concentration-response function estimated in one location to other locations is a notable source of uncertainty.

Although there may be more uncertainty about the degree of prematurity of mortality captured by short-term exposure studies than by long-term exposure studies, certain sources of uncertainty associated with long-term exposure studies require mention. Although studies that are well-executed attempt to control for those factors that may confound the results of the study, there is always the possibility of insufficient or inappropriate adjustment for those factors that affect long-term mortality rates and may be confounded with the factor of interest (e.g., PM concentrations). Prospective cohort studies have an advantage over ecologic, or population-based, studies in that they gather individual-specific information on such important risk factors as smoking. It is always possible, however, that a relevant, individual-specific risk factor may not have been controlled for or that some factor that is not individual-specific (e.g., climate) was not adequately controlled for. It is therefore possible that differences in mortality rates that have been ascribed to differences in average PM levels may be due, in part, to some other factor or factors (e.g., differences among communities in diet, exercise, ethnicity, climate, industrial effluents, etc.) that have not been adequately controlled for.

Another source of uncertainty surrounding the prospective cohort studies concerns possible historical trends in PM concentrations and the relevant period of exposure, which is as yet unknown. TSP concentrations were substantially higher in many locations for several years prior to the cohort studies and had declined substantially by the time these studies were conducted. If this is also true for PM_{10} and or $PM_{2.5}$, it is possible that the larger PM_{10} and or $PM_{2.5}$ coefficients reported by the long-term exposure studies (as opposed to the short-term exposure studies) reflect an upward bias. If the relevant exposure period extends over a decade or more, then a coefficient based on PM concentrations at the beginning of the study or in those years immediately prior to the study could be biased upward if pollution levels had been decreasing markedly for a decade or longer prior to the study.

On the other hand, if a downward trend in PM concentrations continued throughout the period of the study, and if a much shorter exposure period is relevant (e.g., contained within the study period itself), then characterizing PM levels throughout the study by those levels just prior to the study would tend to bias the PM coefficient downward.

The relevant exposure period is one of a cluster of characteristics of the mortality-PM relationship that are as yet unknown and potentially important. It is also unknown whether there is a time lag in the PM effect. Finally, it is unknown whether there may be cumulative effects of chronic exposure — that is, whether the relative risk of mortality actually increases as the period of exposure increases.

Estimating the relationship between PM and premature mortality. The incidence of PM-related mortality used for estimating the benefits of the CAA is based on the concentration-response relationship reported by one of the two recent long-term exposure (prospective cohort) studies (Pope et al., 1995, and Dockery et al., 1993). Because it is based on a much larger population and many more locations than Dockery et al. (1993), the concentration-response function from Pope et al. (1995) was used in this analysis. The results of Pope et al. are consistent with those of Dockery et al., which reported an even larger response, but in only six cities. Moreover, Pope et al. is also supported by several ecological cross-sectional studies of annual mortality based on 1960 and 1970 census data (using either TSP or sulfate as indicators of PM), including the work of Lave and Seskin (1977) and Lipfert (1984).

Numerous short-term exposure (time series) studies have also reported a positive and statistically significant relationship between PM and mortality. Of the fourteen studies that estimated the relationship between daily PM_{10} concentrations and daily mortality listed in Table 12-2 of the PM Criteria Document, twelve reported positive and statistically significant findings (Pope et al., 1992; Pope and Kalkstein, 1996; Dockery et al., 1992; Schwartz, 1993a; Ozkaynak et al., 1994; Kinney et al., 1995: Ito et al., 1995; Ostro et al., 1996; Saldiva et al., 1995; Styer et al., 1995; Ito and Thurston, 1996; Schwartz et al., 1996). While these studies lend substantial support to the hypothesis that there is a relationship between PM_{10} and mortality, they may be capturing only the portion of that relationship involving short-term effects. For this reason, they are considered in this analysis only as supporting evidence to the results of the study by Pope et al.

The Pope et al. study has several further advantages. The population followed in this study was largely white and middle class, decreasing the likelihood that interlocational differences in premature mortality were due in part to differences in socioeconomic status or related factors. In addition, the generally lower mortality rates and possibly lower exposures to pollution among this group, in comparison to poorer minority populations, would tend to bias the PM coefficient from this study downward, counteracting a possible upward bias associated with historical air quality trends discussed above.

Another source of downward bias in the PM coefficient in Pope et al. is that intercity movement of cohort members was not considered in this study. Migration across study cities would result in exposures of cohort members being more similar than would be indicated by assigning city-specific annual average pollution levels to each member of the cohort. The more intercity migration there is, the more exposure will tend toward an intercity mean. If this is ignored, differences in exposure levels, proxied by differences in city-specific annual median PM levels, will be exaggerated, resulting in a downward bias of the PM coefficient (because a given difference in mortality rates is being associated with a larger difference in PM levels than is actually the case).

In summary, because long-term exposure studies appear to have captured more of the PM-related premature mortality, as well as premature mortality that is more premature, they are preferable to the short-term exposure studies. Among the long-term exposure studies, the Pope et al. study has several advantages, as discussed above, which are likely to reduce the possibility of a key source of confounding and increase the reliability of the concentration-response function from that study. For these reasons, the concentration-response function estimated in this study is considered the most reasonable choice for this analysis.

Matching PM Indices in the Air Quality Profiles and Concentration-Response Function. The Pope et al. study examined the health effects associated with two indices of PM exposure: sulfate particles and fine particles ($PM_{2.5}$). The reported mortality risk ratios are slightly larger for $PM_{2.5}$ than for sulfates (1.17 versus 1.15 for a comparison between the most polluted and least polluted cities). The $PM_{2.5}$ relationship is used in this analysis because it is more consistent with the PM_{10} air quality data selected for the analysis. Estimated changes in $PM_{2.5}$ air quality must be matched with the $PM_{2.5}$ mortality relationship. However, only PM_{10} profiles were used for the entire 20 year period. Therefore, the same regional information about the PM_{10} components (sulfate, nitrate, organic particulate and primary particulate) used to develop the PM_{10} profiles were used to develop regional $PM_{2.5}/PM_{10}$ ratios. Although both urban and rural ratios are available, for computational simplicity, only the regional urban ratios were used to estimate the $PM_{2.5}$ profiles from the PM_{10} profiles used in the analysis. This reflects the exposure of the majority of the modeled population (i.e., the urban population), while introducing some error in the exposure changes for the rural population. In the east and west, where the rural ratio is larger than the urban ratio, the change in $PM_{2.5}$ exposure will be underestimated for the rural population.

In the central region the $PM_{2.5}$ change will be overestimated. These ratios were used in each year during 1970-1990, introducing another source of uncertainty in the analysis. Table D-5 summarizes the $PM_{2.5}/PM_{10}$ ratios used in this analysis.

Table D-5. $PM_{2.5}/PM_{10}$ Ratios Used to Estimate $PM_{2.5}$ Data Used With Pope et al. (1995) Mortality Relationship.

	East	**Central**	**West**	**National**
Urban	0.59	0.58	0.48	0.55
Rural	0.68	0.53	0.49	0.57

Prematurity of Mortality: Life-Years Lost as a Unit of Measure

Perhaps the most important health effect that is examined in this analysis is mortality. Although this analysis does not take into account the degree of prematurity of death (that is, the ages of those individuals who die prematurely from exposure to PM are not considered), considerable attention has been paid to this issue and, in particular, to life-years lost as an alternative to lives lost as a measure of the mortality-related effects of pollution.

Because life-years lost is of potential interest and because there is a substantial potential for confusion in understanding apparently disparate estimates of life-years lost from pollution exposure, this section attempts to present a clear discussion of the various possible measures of life-years lost, what they depend on, and how they are related to each other.

Because the actual number of years any particular individual is going to live cannot be known, “life-years lost” by an individual actually refers to an *expected* loss of years of life by that individual. The expected loss of years of life by an individual depends crucially on whether the expectation is contingent on the individual only having been exposed to PM or on the individual actually having died from that exposure.

An *ex ante* estimate of life-years lost per individual is contingent not on the individual having died prematurely but only on the individual having been exposed. Suppose, for example, that a 25 year old has a life expectancy of 50 more years in the absence of exposure and only 49 more years in the presence of exposure. Given (chronic) exposure from the age of 25 on, the 25 year old exposed to (some elevated level of) PM might expect a shortening of life expectancy of one year, for example. That is one expected life-year lost due to chronic exposure. This is the life-years lost that can be expected by every *exposed* individual.

An *ex post* estimate of life-years lost per individual is contingent on the individual actually having died from exposure to PM. When an individual dies of exposure to PM, he is said to have lost the number of years he would have been expected to live, calculated, for example, from age- and gender-specific life expectancy tables. Suppose that the life expectancy of 25 year olds is 75 — that is, a 25 year old can expect to live 50 more years. A 25 year old who dies from exposure to PM has therefore lost 50 expected years of life. This is the life-years lost that can be expected by every 25 year old *affected* individual (i.e., every 25 year old who actually dies from exposure to PM).

Estimates of the total life-years lost by a population exposed to PM depend on several factors, including the age distribution and the size of the exposed population, the magnitude of the change (or changes) in PM being considered, the relative risk assumed to be associated with each change in PM, and the length of time exposure (i.e., the change in PM) is presumed to occur. A population chronically exposed to a given increase in PM will lose more life-years than a population exposed to the same increase in PM for only a year or two.[6] A population that is generally older will lose fewer life-years, all else equal, than one that is generally younger, because older individuals have fewer (expected) years of life left to lose. And a population exposed to a greater increase in PM will lose more life-years than if it were exposed to a smaller increase in PM. Finally, the life-years lost by the population will increase as the relative risk associated with the increase in PM increases.

Life-years lost are usually reported as averages over a population of individuals. The population being averaged over, however, can make a crucial difference in the reported average life-years lost, as noted above. The average life-years lost *per exposed individual* (the *ex ante* estimate) is just the total life-years lost by the population of exposed individuals divided by the number of exposed individuals. This average will depend on all the factors that the total life-years lost depends on except the size of the exposed population. The average life-years lost by an exposed individual is a statistical expectation. It is the average of the numbers of life-years actually lost by each member of the exposed population. Alternatively, it can be thought of as a weighted average of possible numbers of years lost, where the weights are the proportions of the population that lose each number of expected years of life. Although those individuals who do die prematurely from exposure to PM may lose several expected years of life, most exposed individuals do not actually die from exposure to PM and therefore lose zero life-years. The average life-years lost per exposed individual in a population, alternatively referred to as the average decrease in life expectancy of the exposed population, is therefore heavily weighted towards zero. The average number of life-years lost *per individual who dies of exposure to PM* (the *ex post* measure of life-years lost) is an average of the numbers of expected years of life lost by individuals who actually died prematurely because of PM. Because everyone who dies prematurely from exposure to PM loses some positive number of expected years of life, this average, by definition, does not include zero.

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[6] Even in the absence of cumulative effects of exposure, exposure of a population for many years will result in a greater total number of pollution-related deaths than exposure for only a year or two, because the same relative risk is applied repeatedly, year after year, to the population, rather than for only a year or two.

An example of an *ex ante* measure of life-years lost is given by a study in the Netherlands (WHO, 1996), which considered a cohort of Dutch males, aged 25-30, and compared the life expectancy of this cohort to what it would be in a hypothetical alternative scenario in which these individuals are continuously exposed to concentrations of $PM_{2.5}$ that are 10 $\mu g/m^3$ lower than in the actual scenario. The life expectancy of this cohort of 25-30 year old Dutch males was calculated to be 50.21 years in the actual scenario, based on a 1992 life table from the Netherlands. Assuming that the relative risk of mortality associated with an increase of 10 $\mu g/m^3$ $PM_{2.5}$ is 1.1 (the average of the relative risks of 1.14 from Dockery et al., 1993, and 1.07 from Pope et al., 1995), the study authors calculated death rates in the hypothetical "cleaner" scenario by dividing the age-specific death rates in the actual scenario by 1.1. Using these slightly lower death rates, and assuming that the effect of PM does not begin until 15 years of exposure, the authors constructed a life table for the cohort in the hypothetical "cleaner" scenario. Based on this new life table in a cleaner world, the life expectancy of the cohort of 25-30 year old Dutch males was calculated to be 51.32 years in the hypothetical cleaner scenario. (In calculating life expectancies in both the "dirty" scenario and the "clean" scenario, it is assumed that any individual who does not survive to the next 5-year age group lives zero more years. For example, a 30 year old individual either survives to age 35 or dies at age 30.) The change in life expectancy for this cohort of 25-30 year old Dutch males, due to a change in PM exposure of 10 $\mu g/m^3$ for the rest of their lives (until the age of 90), was therefore 51.32 years - 50.21 years = 1.11 years. That is, the average life-years lost by an exposed individual in this population, under these assumptions, is 1.11 years.

The estimate of 1.11 years of expected life lost depends on several things, as mentioned above. If the study authors had used the relative risk from Pope et al., 1995, alone, (1.07 instead of 1.1), for example, the change in life expectancy (the *ex ante* measure of life-years lost) for this cohort of 25-30 year old Dutch males would have been 0.80 years. Similarly, changing the assumption about the duration of exposure also changes the estimate of *ex ante* life-years lost. Using a relative risk of 1.1, but assuming that exposure lasts only during the first 5 years (i.e., that the death rate in the first five years, from age 25 through age 30, is lower but that after that it is the same as in the "dirty" scenario), the average life-years lost by an exposed individual in this population is reduced from 1.11 years to 0.02 years.

By their construction and definitions, the average life-years lost per exposed individual and the average life-years lost per affected individual (i.e., per individual who dies prematurely from PM) take the same total number of life-years lost by the exposed population and divide them by different denominators. The average life-years lost per exposed individual divides the total life-years lost by the total population exposed; the average life-years lost per affected individual divides the same total life-years lost by only a small subset of the total population exposed, namely, those who died from PM. The average per exposed individual is therefore much smaller than the average per affected individual. Because both types of average may be reported, and both are valid measurements, it is important to understand that, although the numbers will be very dissimilar, they are consistent with each other and are simply different measures of the estimated mortality impact of PM.

To calculate the total (estimated) life-years lost by a population, it is necessary to follow each age cohort in the population through their lives in both scenarios, the "dirty" scenario and the "clean" scenario, and compute the difference in total years lived between the two scenarios, as WHO (1996) did for the cohort of Dutch males 25-30 years old. This method will be referred to as Method 1. In practice, however, it is not always possible to do this. (Other changes to the population, such as those from recruitment and immigration, for example, would make such an exercise difficult.) An alternative method, which approximates this, is to predict the numbers of individuals in each age category who will die prematurely from exposure to PM (i.e., who will die prematurely in the "dirty" scenario), and multiply each of these numbers by the corresponding expected number of years remaining to individuals in that age category, determined from life expectancy tables. This method will be referred to as Method 2. Suppose, for example, that individuals age 25 are expected to live to age 75, or alternatively, have an expected 50 years of life remaining. Suppose that ten 25 year olds are estimated to die prematurely because of exposure to PM. Their expected loss of life-years is therefore 50 years each, or a total of 500 life-years. If the same calculation is carried out for the individuals dying prematurely in each age category, the sum is an estimate of the total life-years lost by the population.

Using Method 1 (and retaining the assumptions made by WHO, 1996), the average life-years lost per PM-related death among the cohort of Dutch males is calculated to be 14.28 years. Using Method 2 it is estimated to be 14.43 years.

Although this *ex post* measure of life-years lost is much larger than the *ex ante* measure (1.11 life-years lost per exposed individual), it only applies to those individuals who actually die from exposure to PM. The number of individuals in the age 25-30 Dutch cohort example who eventually die from exposure to PM (7,646) is much smaller than the number of individuals in the age 25-30 Dutch cohort who are exposed to PM (98,177). The total life-years lost can be calculated either as the number of exposed individuals times the expected life-years lost per exposed individual (98,177*1.11 = 109,192.1) or as the number of affected individuals times the expected life-years lost per affected individual (7,646*14.28 = 109,192.1).

To further illustrate the different measures of life-years lost and the effects of various input assumptions on these measures, death rates from the 1992 U.S. Statistical Abstract were used to follow a cohort of 100,000 U.S. males from birth to age 90 in a "dirty" scenario and a "clean" scenario, under various assumptions. Death rates were available for age less than 1, ages 1-4, and for ten-year age groups thereafter. The ten-year age groups were divided into five-year age groups, applying the death rate for the ten-year group to each of the corresponding five-year age groups. *Ex ante* and *ex post* measures of life-years lost among those individuals who survive to the 25-29 year old category were first calculated under the assumptions in the WHO (1996) study. These assumptions were that the relative risk of mortality in the "dirty" scenario versus the "clean" scenario is 1.1; that exposure does not begin until age 25; that the effect of exposure takes fifteen years; that individuals at the beginning of each age grouping either survive to the next age grouping or live zero more years; and that all individuals age 85 live exactly five more years. Under these assumptions, the expected life-years lost per exposed individual in the 25-29 year old cohort is 1.32 years. There are 96,947 exposed individuals in this age cohort. The expected life-years lost per affected individual (i.e., per PM-related death) is 16.44 years (Method 1). There are 7,804 affected individuals. The total life-years lost by individuals in this cohort is 128,329.3 (1.32*96,947 = 16.44* 7,804 = 128,329.3).

If the relative risk is changed to 1.07, the expected life-years lost per exposed individual in the cohort of 25-29 year old U.S. males is reduced from 1.32 to 0.95 years. The expected life-years lost per affected individual (i.e., per PM-related death) is 16.44 years (Method 1). Using a relative risk of 1.1 but assuming no lag (i.e., assuming that exposure starts either at birth or at age 25 and has an immediate effect), the expected life-years lost per exposed individual in the 25-29 year old cohort changes from 1.32 to 1.12. The expected life-years lost per affected individual (i.e., per PM-related death) becomes 19.7 years (Method 1).

Estimating Morbidity Effects

In addition to mortality effects, this analysis quantifies effects for a number of non-fatal health endpoints. Several issues arise in implementing the studies selected for this analysis.

Overlapping Health Effects

Several endpoints reported in the health effects literature overlap with each other. For example, the literature reports relationships for hospital admissions for single respiratory ailments (e.g. pneumonia or chronic obstructive pulmonary disease) as well as for all respiratory ailments combined. Similarly, several studies quantify the occurrence of respiratory symptoms where the definitions of symptoms are not unique (e.g., shortness of breath, upper respiratory symptoms, and any of 19 symptoms). Measures of restricted activity provide a final example of overlapping health endpoints. Estimates are available for pollution-induced restricted activity days, mild restricted activity days, activity restriction resulting in work loss. This analysis models incidence for all endpoints. Double-counting of benefits is avoided in aggregating economic benefits across overlapping endpoints (see Appendix I).

Studies Requiring Adjustments

Applying concentration-response relationships reported in the epidemiological literature to the national scale benefits analysis required by section 812 required a variety of adjustments.

Normalization of coefficients by population. To be applied nationwide, concentration-response coefficients must reflect the change in risk per person per unit change in air quality. However, some studies report the concentration-response coefficient, , as the change in risk for the entire studied population. For example, Thurston et al. (1994) reported the total number of respiratory-related hospital admissions/day in the Toronto, Canada area. To normalize the coefficient so that it might be applied universally across the country, it was divided by the population in the geographical area of study (yielding an estimate of the change in admissions/person/day due to a change in pollutant levels).

Within-study meta-analysis. In some cases, studies reported several estimates of the concentration-response coefficient, each corresponding to a particular year or particular study area. For example, Ostro and Rothschild (1989) report six separate regression coefficients that correspond to regression models run for six separate years. This analysis combined the individual estimates using a fixed coefficient meta-analysis on the six years of data.

Conversion of coefficients dependent on symptom status during the previous day. Krupnick et al. (1990) employed a Markov process to determine the probability of symptoms that were dependent on symptom status of the previous day. The current analysis adjusts the regression coefficients produced by the model in order to eliminate this dependence on previous day's symptom status.

Concentration-Response Functions: Health Effects

After selecting studies appropriate for the section 812 analysis, taking into account the considerations discussed above, the published information was used to derive a concentration-response function for estimating nationwide benefits for each health effect considered. In general, these functions combine air quality changes, the affected population and information regarding the expected per person change in incidence per unit change in pollutant level. The following tables present the functions used in this analysis, incorporating information needed to apply these functions and references for information.

Particulate Matter

The concentration-response functions used to quantify expected changes in health effects associated with reduced exposure to particulate matter are summarized in Table D-6. The data profiles selected for use in this analysis are PM_{10}. In those cases in which PM_{10} was not the measure used in a study, this analysis either converted PM_{10} air quality data to the appropriate air quality data (e.g., $PM_{2.5}$ or TSP) or, equivalently, converted the pollutant coefficient from the study to the corresponding PM_{10} coefficient, based on location-specific information whenever possible.

Table D-6. Summary of Concentration-Response Functions for Particulate Matter.

Except where noted otherwise, the functional form is

$$\Delta cases = cases * (e^{\beta * \Delta PM\text{-}10} - 1)$$

where “cases” refers to incidence at the first pollution level.

Health Endpoint (ICD-9 code)	**Baseline Incidence (per 100,000)**	**Expos Meas. from Original Study**	**Study Pop.**	**Applied Pop.**	**Functional form**[a]	**Uncert & Var.**	**Sources**
mortality (long-term exposure)	non-accidental deaths by county[b]	annual median $PM_{2.5}$	50 cities, all deaths	over age 30	$\beta_{PM2.5} = 0.006408$ PM_{10} data converted to $PM_{2.5}$ data[c]	s.e. = 0.00148	Pope et al., 1995 American Cancer Society cohort
hospital admissions--all resp. illnesses (ICD 460-519)	504[d]/year (incidence in pop. > 65 years of total U.S. pop.)	same day PM_{10}	65 and older in New Haven, CT, Tacoma, WA	65 and older	New Haven: 0.00172 Tacoma: 0.00227 average: 0.0020	c.i. = New Haven: 1.00-1.12 s.e. = 0.00093 Tacoma: 0.97-1.29 s.e. = 0.00146	Schwartz, 1995 New Haven and Tacoma
hospital admissions -- all resp. illnesses (ICD 460-519)	n/a	mean monthly PM_{10}	variety of ages in Salt Lake Valley, Utah	all	Δ cases = $\beta * \Delta PM_{10}$ * Pop. where $\beta = 0.8047$ monthly admissions / Salt Lake Valley population (780,000). $= 3.4 \times 10^{-8}$ (converted from monthly to daily admissions)	s.e. = 0.28	Pope, 1991 Salt Lake Valley
daily respiratory admissions (total) includes 466, 480, 481, 482, 485, 490, 491, 492, 493	n/a	same-day PM_{10}	Toronto metro area	all	Δ cases = $\beta * \Delta PM_{10}$ * Pop where $\beta = 0.0339$ daily admissions / Toronto population (2.4 million) $= 1.4 \times 10^{-8}$ (model also includes O_3)	s.e. = 0.034/2.4 million $= 1.4 \times 10^{-8}$	Thurston et al. 1994 Toronto
hospital admissions pneumonia (480-487)	229[d]/year (incidence in pop. > 65 years of total U.S. pop.)	same day PM_{10}	over 65, Birmingham AL	over 65	$\beta = 0.00174$	c.i. = 1.07 - 1.32 s.e. = 0.000536	Schwartz, 1994a Birmingham

Health Endpoint (ICD-9 code)	Baseline Incidence (per 100,000)	Expos Meas. from Original Study	Study Pop.	Applied Pop.	Functional form[a]	Uncert & Var.	Sources
hospital admissions COPD (490-496)	103[d]/year (incidence in pop. > 65 years of total U.S. pop.)	same day PM_{10}	over 65, Birmingham AL	over 65	$\beta = 0.00239$	c.i. = 1.08 - 1.50 s.e. = 0.00084	Schwartz, 1994a Birmingham
hospital admissions pneumonia (480-487)	229[d]/year (incidence in pop. > 65 years of total U.S. pop.)	same day PM_{10}	over 65, Detroit	over 65	$\beta = 0.00115$	s.e. = 0.00039	Schwartz, 1994b Detroit
hospital admissions COPD (490-496)	103[d]/year (incidence in pop. > 65 years of total U.S. pop.)	same day PM_{10}	over 65, Detroit	over 65	$\beta = 0.00202$	s.e. = 0.00059	Schwartz, 1994b Detroit
hospital admissions pneumonia (480-487)	229[d]/year (incidence in pop. > 65 years of total U.S. pop.)	same day PM_{10}	65 and over in Mpls	over 65	$\beta = 0.00157$	c.i. = 1.02 - 1.33 s.e. = 0.00068	Schwartz, 1994c Mpls, St. Paul
hospital admissions COPD (490-496)	103[d]/year (incidence in pop. > 65 years of total U.S. pop.)	current and previous day PM_{10}	65 and over in Mpls	over 65	$\beta = 0.00451$	c.i. = 1.20 - 2.06 s.e. = 0.00138	Schwartz, 1994c Mpls, St. Paul
hospital admissions for congestive heart failure (ICD 428)	231[d]/year (incidence in pop. > 65 years of total U.S. pop.)	avg same and previous day PM_{10}	65 and older in Detroit	65 and older	$\beta = 0.00098$	c.i. = 1.012-1.052 s.e. = 0.00031	Schwartz and Morris, 1995 Detroit
hospital admissions for ischemic heart disease (ICD 410-414)	450[d]/year (incidence in pop. > 65 years of total U.S. pop.)	24 hr avg PM_{10} same day	65 and older in Detroit	65 and older	$\beta = 0.00056$	c.i. = 1.005-1.032 s.e. = 0.00021	Schwartz and Morris, 1995 Detroit

Health Endpoint (ICD-9 code)	Baseline Incidence (per 100,000)	Expos Meas. from Original Study	Study Pop.	Applied Pop.	Functional form[a]	Uncert & Var.	Sources
hospital admissions -- all resp. illnesses (ICD 460-519)	504[d]/year (incidence in pop. > 65 years of total U.S. pop.)	24 hr avg PM_{10}	over 65, Spokane	over 65	$\beta = 0.00163$	s.e. = 0.00047	Schwartz, 1996, Spokane
hospital admissions COPD (490-496)	103[d]/year (incidence in pop. > 65 years of total U.S. pop.)	24 hr avg PM_{10}	over 65, Spokane	over 65	$\beta = 0.00316$	s.e. = 0.00084	Schwartz, 1996, Spokane
hospital admissions pneumonia (480-487)	229[d]/year (incidence in pop. > 65 years of total U.S. pop.)	24 hr avg PM_{10}	over 65, Spokane	over 65	$\beta = 0.00103$	s.e. = 0.00068	Schwartz, 1996, Spokane
LRS defined as cough, chest pain, phlegm, and wheeze	not applicable	same day PM_{10}	8-12 yr olds	0-12 yr olds	Δcases = $\frac{P_0}{(1-P_0) * e^{\beta * \Delta PM10}} - P_0$ where P_0 = the probability of a child in the study pop suffering from LRS in the base case = 1.45 % and $\beta = 0.014176$	s.e. = 0.0041	Schwartz et al., 1994d
shortness of breath, days	not applicable	24 hour avg PM_{10}	African-American asthmatics between ages 7 and 12	same as study pop.	Δcases = $\frac{P_0}{(1-P_0) * e^{\beta * \Delta PM10}} - P_0$ where P_0 = the probability of a child in the study pop. suffering from shortness of breath in the base case = 5.6 % and $\beta = 0.008412$	s.e. = 0.00363	Ostro et al., 1995

Health Endpoint (ICD-9 code)	Baseline Incidence (per 100,000)	Expos Meas. from Original Study	Study Pop.	Applied Pop.	Functional form[a]	Uncert & Var.	Sources
URI defined as runny or stuffy nose, wet cough, burning, aching, or red eyes	1,192[e] (ages 10-12) 5,307[e] (ages <= 12)	same day PM_{10}	10-12 yr old non-symptomatic	12 and under	$\beta = 0.0036$	s.e. = 0.0015	Pope et al., 1991 Utah
acute bronchitis (ICD 466)	n/a	PM_{10} annual avg (converted)	10 to 12 year olds	18 and under	$\beta = 0.0330$ $\Delta cases = \frac{P_0(e^{\beta * \Delta PM\text{-}10})}{1 - P_0 + P_0(e^{\beta * \Delta PM\text{-}10})} * Pop$ P_0 = baseline probability of having bronchitis =0.065[e]	s.e. = 0.0216	Dockery et al., 1989 6 cities
chronic bronchitis	710/year (of study pop.)	annual mean TSP	Seventh Day Adventists in California	all	$\beta = 0.00512$ convert PM_{10} to TSP: $\Delta\ TSP = \frac{\Delta PM10}{0.56}$ where 0.56 is the specific conversion based on region and initial TSP conc.	not available	Abbey et al., 1993

Health Endpoint (ICD-9 code)	Baseline Incidence (per 100,000)	Expos Meas. from Original Study	Study Pop.	Applied Pop.	Functional form[a]	Uncert & Var.	Sources
chronic bronchitis	600/year	annual mean TSP	adults 30-74 years old in 53 U.S. urban areas	all	Δ cases/year = $(p_1 - p_0)$ * Pop where $p_1 = \frac{1}{1 + e^{-(\ln\frac{p_0}{1 - p_0} + \beta * \Delta PM_{10})}}$ where $p_0 = 0.006$ = the probability of developing physician-diagnosed chronic bronchitis per individual per year and $\beta = 0.0012$, the PM_{10} coefficient, converted from the TSP coefficient, using the relationship: $\Delta TSP = \frac{\Delta PM10}{0.56}$ where 0.56 is the specific conversion based on region and initial TSP conc.	95% CI = (1.02 - 1.12) for odds ratio corresponding to a 10 μg/m3 increase in annual TSP	Schwartz, 1993b
presence of any of 19 acute respiratory symptoms	not applicable	24 hour average COH in units/ 100 ft)[g] COH = coeff. of haze	adult members of families of elementary school-aged children in Glendora-Covina-Azusa, CA	adults 18-65	Δ $Sympt_{days}$/day = $(p_1 - p_0)$ * Pop where $p_1 = \frac{1}{1 + e^{-(\ln\frac{p_0}{1 - p_0} + \beta * \Delta O_3)}}$ and p_0 = the probability of $Sympt_{days}$ per individual for a 24-hour period in the base case = 0.19 $\beta = 0.00046$[g] (Model includes O_3, COH, SO_2)	s.e. = 0.00024[h]	Krupnick et al., 1990

Health Endpoint (ICD-9 code)	Baseline Incidence (per 100,000)	Expos Meas. from Original Study	Study Pop.	Applied Pop.	Functional form[a]	Uncert & Var.	Sources
moderate or worse asthma status	n/a	average $PM_{2.5}$ during 9:00 am to 4:00 pm ($\mu g/m^3$)	Denver asthmatics between ages 18 and 70	asthmatic (4%[i] of total pop.)	Δ asthma status= $\beta[\ln(X_1/X_0)]$*Pop where $X_0 = PM_{10}$ concentrations with CAA, $X_1 = PM_{10}$ concentrations without CAA, and $\beta = 0.00038$[j] (model includes $PM_{2.5}$ and modeled $PM_{2.5}$ measures for periods where $PM_{2.5}$ measures were missing)	s.e. = 0.00019	Ostro et al., 1991 Denver
Restricted Activity Days (RADs)	400,531 days/year[k] (of the total U.S. pop)	2-wk average $PM_{2.5}$ ($\mu g/m^3$)	All adults 18-65 in metropolitan areas in the U.S.	adults aged 18-65	Δ health effects determined over a 2 wk period $\beta = 0.0030$[j, l]	s.e. = 0.00018[l]	Ostro, 1987
respiratory and nonrespiratory conditions resulting in a minor restricted activity day (MRAD)	780,000 days/year (cited as 7.68 days per person per year in study)	$PM_{2.5}$ averaged over a 2-wk period	employed adults across the U.S. between the ages of 18-65	adults aged 18-65	number of health effects determined over a 2-week period $\beta = 0.00463$[j, l] (Model includes fine particulates and O_3)	s.e. = 0.00044[l]	Ostro and Rothschild, 1989
respiratory restricted activity days (RRADs)	306,000 days/year (cited as 3.06 days per person per year in study)	$PM_{2.5}$ averaged over a 2 wk period	employed adults across the U.S. between the ages of 18-65	adults aged 18-65	number of health effects determined over a 2-wk period $\beta = 0.00936$[j, l] (Model includes fine particulates and O_3)	s.e. = 0.00103[l]	Ostro and Rothschild, 1989
Work Loss Days (WLDs)	150,750[m] (of total U.S. pop)	2-wk average $PM_{2.5}$ ($\mu g/m^3$)	All adults 18-65 in metropolitan areas in the U.S.	adults aged 18-65	Δ health effects determined over a 2 wk period $\beta = 0.0029$[j, l]	s.e. = 0.00022[l]	Ostro, 1987

NOTES:

[a] Pollutant coefficients reflect changes in health effects per change in μg/m^3 PM$_{10}$.

[b] Mortality baseline incidence data for each county taken from Vital Statistics of the United States, Vol. II - Mortality, Part B, (U.S. Dept. of Health and Human Services). Incidence rates were generated for total mortality excluding accidental deaths and adverse effects, suicide, homicide, and other external causes (ICD E800-E999). Rates calculated based on 1990 population.

[c] PM$_{10}$ data converted to PM$_{2.5}$ data by using national urban average PM$_{2.5}$/PM$_{10}$ ratio = 0.56.

[d] Centers for Disease Control, 1992. Vital and Health Statistics, Detailed Diagnoses and Procedures, National Hospital Discharge Survey, 1990. Number of 1990 discharges divided by 1990 U.S. population (248,709,873) from City and County Databook, 12th edition, 1994, U.S. Dept. of Commerce, Bureau of the Census, Washington, D.C.

[e] Pope et al., 1991 NOTE: rates were not available from standard incidence sources and so were calculated from incidence in the study of 10-12 year olds. This may not be entirely appropriate for older or younger individuals. Children of this age are less likely to have colds than much younger children and may be more representative of the adult population.

[f] Dockery et al., 1989.

[g] Coefficient and standard error are converted from a β and s.e. for coefficient of haze (COH) to a β and s.e. for PM$_{10}$. This was done by using a ratio of COH to TSP of 0.116 from the study authors (as cited in ESEERCO, 1994) and a ratio of PM$_{10}$ to TSP of 0.55 (U.S. EPA, 1986).

[h] Coefficient and standard error incorporate the stationary probabilities as described in Krupnick et al. (1990). To do this, the calculation used the transitional probabilities supplied by the authors and presented in ESEERCO, 1994.

[i] U.S. EPA, 1994a.

[j] β converted from a change in health effects per change in μg/m^3 PM$_{2.5}$ to a change per μg/m^3 PM$_{10}$ using the following relationship: 1 μg/m^3 PM$_{2.5}$ = 0.56 μg/m^3 PM$_{10}$ (ESEERCO, 1994)

[k] Number of RADs for all acute conditions from: National Center for Health Statistics. Current Estimates from the National Health Interview Survey: United States, 1990. (Hyattsville, MD). This number is divided by the U.S. population for 50 states for 1990 (248,709,873) and multiplied by 100,000 (to obtain the incidence per 100,000).

[l] Based on fixed-weight meta-analysis of single-year coefficients and standard errors reported in study.

[m] Number of WLDs of 374,933,000 from: National Center for Health Statistics. Current Estimates from the National Health Interview Survey from 1990. (Hyattsville, MD). Series 10, No. 181. This number is divided by the U.S. population for 50 states for 1990 (248,709,873) and multiplied by 100,000 (to obtain the incidence per 100,000).

Ozone

The health effects literature includes studies of the relationships between ozone and a variety of non-fatal health effects. Many of these relationships are provided by the same studies that reported the particulate matter relationships shown above. For some health endpoints, most notably hospital admissions, multiple studies report alternative estimates of the concentration-response relationship. The variability between these reported estimates is incorporated into the Monte Carlo approach used to combine estimates of avoided health effects with economic valuations (discussed in Appendix I). Table D-7 documents the concentration-response functions used in this analysis.

Table D-7. Summary of Concentration-Response Functions for Ozone.

Except where noted otherwise, the functional form is

$$\Delta cases = cases * (e^{\beta * \Delta O3} - 1)$$

where “cases” refers to incidence at the first pollution level.

Health Endpoint (ICD-9 code)	Baseline Incidence (per 100,000)	Expos Meas from original study	Study Pop.	Applied Pop.	Functional form[a]	Uncert & Var.	Sources
hospital admissions -- all resp. illnesses (ICD 460-519)	504/year[b] (incidence in pop .> 65 years of total U.S. pop.)	24 hr avg (μg/m^3)	65 and older in New Haven, CT, Tacoma, WA	over 65 only	β = New Haven: 0.0027 Tacoma: 0.007 where 1 μg/m3= 0.510 ppb (two pollutant model with PM_{10} and O_3)	New Haven: s.e. = 0.0014 Tacoma: s.e. = 0.0025 where 1μg/m^3 = 0.51 ppb	Schwartz, 1995 New Haven and Tacoma
daily respiratory admissions- includes 466, 480, 481, 482, 485, 490, 491, 492, 493	n/a	1 hour daily max ozone (ppb)	all	all	for Toronto: β = 0.0388/2.4 million =1.62x10^{-8} Δ cases/day = β * Δ O_3 * pop (ozone and PM_{10} model used)	se = 0.0241/2.4 million =1.0x10^{-8}	Thurston et al., 1994 Toronto
hospital admissions pneumonia (480-487)	229/year[b] (incidence in pop .> 65 years of total U.S. pop.)	24-hr avg ppb	over 65, Birmingham AL	over 65	β = 0.00262 for O_3 alone (single pollutant model only avail.)	s.e. = 0.00196	Schwartz, 1994a Birmingham

Health Endpoint (ICD-9 code)	Baseline Incidence (per 100,000)	Expos Meas from original study	Study Pop.	Applied Pop.	Functional form[a]	Uncert & Var.	Sources
hospital admissions COPD (490-496)	103/year[b] (incidence in pop .> 65 years of total U.S. pop.)	24-hr avg ppb	over 65, Birmingham AL	over 65	$\beta = 0.00314$ for O_3 only (only single pollutant model avail.)	s.e. = 0.00316	Schwartz, 1994a Birmingham
hospital admissions pneumonia (480-487)	229/year[b] (incidence in pop .> 65 years of total U.S. pop.)	24-hr avg ppb	over 65, Detroit	over 65	$\beta = 0.00521$ (two pollutant model with O_3 and PM_{10}) note: authors suggest a threshold of 25 ppb	s.e. = 0.0013	Schwartz, 1994b Detroit
hospital admissions COPD (490-496)	103/year[b] (incidence in pop .> 65 years of total U.S. pop.)	24-hr avg ppb	over 65, Detroit	over 65	$\beta = 0.00549$ (two pollutant model with O_3 and PM_{10}) note: authors suggest a threshold of 25 ppb	s.e. = 0.00205	Schwartz, 1994b Detroit
hospital admissions pneumonia (ICD 480-487)	229/year[b] (incidence in pop .> 65 years of total U.S. pop.)	24 hr avg ppb	65 and over in Mpls	over 65	$\beta = 0.002795$ (two pollutant model with O_3 and PM_{10})	s.e. = 0.00172	Schwartz 1994c Mpls, St. Paul
hospital admissions -- all resp. illnesses (ICD 460-519)	504/year[b] (incidence in pop .> 65 years of total U.S. pop.)	1 hour daily max ozone (ppb)	over 65, Spokane	over 65	$\beta = 0.008562$	s.e. = 0.004326	Schwartz, 1996, Spokane
hospital admissions COPD (490-496)	103/year[b] (incidence in pop .> 65 years of total U.S. pop.)	1 hour daily max ozone (ppb)	over 65, Spokane	over 65	$\beta = 0.004619$	s.e. = 0.007739	Schwartz, 1996, Spokane

Health Endpoint (ICD-9 code)	Baseline Incidence (per 100,000)	Expos Meas from original study	Study Pop.	Applied Pop.	Functional form[a]	Uncert & Var.	Sources
hospital admissions pneumonia (ICD 480-487)	229/year[b] (incidence in pop .> 65 years of total U.S. pop.)	1 hour daily max ozone (ppb)	over 65, Spokane	over 65	$\beta = 0.00965$	s.e. = 0.006011	Schwartz, 1996, Spokane
presence of any of 19 acute respiratory symptoms	n/a	daily one-hour max. O_3 (pphm)	adult members of families of elementary school-aged children in Glendora-Covina-Azusa, CA	adults 18-65	$\Delta\ Sympt_{days}/day = (p_1 - p_0) * Pop$ where $p_1 = \frac{1}{1 + e^{-(\ln\frac{p_0}{1 - p_0} + \beta * \Delta O_3)}}$ and p_0 = the probability of having $Sympt_{days}$ per individual for a 24-hour period in the base case = 0.19 $\beta = 1.4 \times 10^{-4}$ [c] (Model includes O_3, COH, SO_2)	s.e. 6.7×10^{-5} [c]	Krupnick et al., 1990

Health Endpoint (ICD-9 code)	Baseline Incidence (per 100,000)	Expos Meas from original study	Study Pop.	Applied Pop.	Functional form[a]	Uncert & Var.	Sources
self-reported asthma attacks	n/a	1 hour daily max. oxidants (ppm)	asthmatics in Los Angeles	all asthmatics ($4\%^d$ of the total population)	Δ asthma attacks/day = $(p_1 - p_0)$ * Pop where $$p_1 = \frac{1}{1 + e^{-(\ln\frac{p_0}{1-p_0} + \beta * \Delta O_3)}}$$ and p_0 = the probability of attacks per asthmatic for a 24-hour period in the base case, = 0.027^e $\beta = 1.9 \times 10^{-3\ f}$ 1.11 = factor to convert measured O_3 levels to oxidants (only model includes oxidants and TSP)	s.e. = $7.2 \times 10^{-4\ f,g}$	Whittemore and Korn, 1980 and U.S. EPA, 1993b
respiratory and nonrespiratory conditions resulting in a minor restricted activity day (MRAD)	780,000/ year[h] (of study pop.)	1 hour daily max. O_3 (ppm) averaged over 2 weeks	employed adults across the U.S. between the ages of 18-65 (urban residents)	all adults aged 18-65	equation predicts daily change in MRAD $\beta = 2.2 \times 10^{-3\ i}$ (Model includes O_3 and fine particulates)	s.e. = $6.6 \times 10^{-4\ j}$	Ostro and Rothschild, 1989
respiratory restricted activity days (RRADs)	310,000/ year[k] (of study pop.)	1 hour daily max O_3 (ppm) averaged over 2 weeks	employed adults across the U.S. between the ages of 18-65 (urban residents)	all adults aged 18-65	equation predicts daily change in RRAD $\beta = -0.0054^i$ (Model includes O_3 and fine particulates)	s.e. = 0.0017^j	Ostro and Rothschild, 1989

Health Endpoint (ICD-9 code)	Baseline Incidence (per 100,000)	Expos Meas from original study	Study Pop.	Applied Pop.	Functional form[a]	Uncert & Var.	Sources
sinusitis and hay fever	n/a	hourly O_3 averaged over six years (1974-1979) in ppm	adults in urban areas surveyed in the National Health Interview Survey	all	$\Delta cases = \frac{[\Phi(\alpha + \beta x_1) - \Phi(\alpha + \beta x_0)]}{6} * Pop$ where: Φ = standard normal distribution function x_1 = average hourly O_3 concentration over six years in the no-CAA scenario x_0 = average hourly O_3 concentration over six years in the CAA scenario a = -1.13[l] $\beta = 0.017$ maximum likelihood probit model	s.e. = 0.0070[m]	Portney and Mullahy, 1990

Health Endpoint (ICD-9 code)	Baseline Incidence (per 100,000)	Expos Meas from original study	Study Pop.	Applied Pop.	Functional form[a]	Uncert & Var.	Sources
The following two rows should be combined, e.g., cases of $DFEV_1 \geq 15\%$ for heavy exercisers (using equation based on Avol et al., 1984) should be added to cases of $DFEV_1 \geq 15\%$ for moderate exercisers (using equation based on data from Seal et al., 1993)							
Decrements in lung function as measured by forced expiratory volume in one second (FEV_1)	n/a	Exposure to ozone for 1.33 hours during which individuals were exercising continuously for one hour (controlled setting)	Heavily exercising male and female bicyclists (mean age = 26.4 yrs)	all under age 50[n]	$\Delta cases = \alpha * \beta * \Delta O_3 * Pop.$ where, $\beta = 0.00297$ for $DFEV_1 \geq 15\%$ $= 0.00268$ for $DFEV_1 \geq 20\%$ $\alpha = 0.06656$[o]	--	Avol et al., 1984
Decrements in lung function as measured by FEV_1	n/a	Exposure to ozone for 2.33 hours during which individuals were exercising intermittently (total exercise time = 1 hour) (controlled setting)	Moderately exercising male and female college students (ages 18-35)	all under age 50[n]	$= \sum_{i=1}^{3}\left\{\Phi\left[\frac{\ln\left(\frac{X_o \cdot d_i}{1-X_o \cdot d_i}\right)-a}{b}\right]_{e_i} - \Phi\left[\frac{\ln\left(\frac{X_1 \cdot d_i}{1-X_1 \cdot d_i}\right)-a}{b}\right]_{e_i}\right\} \cdot Pop$ where, $a = -0.664$ for $DFEV_1 \geq 15\%$ $= -0.326$ for $DFEV_1 \geq 20\%$ $b = 0.000840$ for $DFEV_1 \geq 15\%$ $= 0.000919$ for $DFEV_1 \geq 20\%$ $d_1 = 1.06$[p] $d_2 = 1.00$ $d_3 = 0.70$ $e_1 = 0.288$[q] $e_2 = 0.224$ $e_3 = 0.640$ X_0 and X_1 are ozone concentrations in the CAA and No-CAA scenarios	--	Seal, et al., 1993

NOTES:

[a] Pollutant coefficients expressed as a change in health effects per change in ppb O_3.

[b] Centers for Disease Control, 1992. Vital and Health Statistics, Detailed Diagnoses and Procedures, National Hospital Discharge Survey, 1990. Number of 1990 discharges divided by 1990 U.S. population (248,709,873) from City and County Databook, 12th edition, 1994, U.S. Dept. of Commerce, Bureau of the Census, Washington, D.C.

[c] Determined the incremental effect/unit O_3 by incorporating stationary probabilities from transitional probabilities. ESEERCO (1994) obtained transitional probabilities for adults from original study authors.

[d] U.S. EPA, 1994a.

[e] Calculated as baseline asthma attack rate (number of attacks per person per year) divided by 365 days per year. Number of attacks per person per year = 9.9 from National Center for Health Statistics, National Health Interview Survey, 1979 (as cited by Krupnick and Kopp, 1988).

[f] β coefficient and s.e. converted to Δ in cases/ppb O_3 based on the following relationship: 1 ppb O_3 = 1.11 ppb oxidants.

[g] Study did not report a s.e. Thus, the analysis assumed the largest s.e. possible (at $p = 0.01$, using a two-tailed test of significance)

[h] Ostro and Rothschild (1989) report average annual MRADs as 7.8 per person, using data from 6 years.

[i] β is a weighted mean using separate coefficients for six years. Each year's coefficient was weighted by the inverse of the variance for that coefficient.

[j] Standard error is the square root of the sum of the weights (sqrt[sum(1/var$_i$)], where I indicates the individual year).

[k] Ostro and Rothschild (1989) report average annual RRADs as 3.1 per person, using data from 6 years.

[l] Obtained by determining the products of beta coefficients for other independent variables and their mean values and summing these and the constant value.

[m] Calculated by dividing β by asymptotic t-ratio.

[n] From Table 12 in 1992 Statistical Abstracts, the percent of individuals in the U.S. population under age 50 = 75%.

[o] Factor to adjust for differences in concentration among microenvironments and amount of time spent in different microenvironments at heavy exercise rates.

[p] The values, d_i, adjust ozone concentrations for various microenvironments (outdoor — near road, outdoor — other, and indoor) using values reported in U.S. EPA, 1993.

[q] The values, e_i, adjust the response rates by the percent of time spent in each microenvironment at the relevant exercise rates (i.e., percent of time at a fast rate is used for Avol et al., 1984, and percent of time at a moderate rate is used for Seal et al., 1993). U.S. EPA (1993) presents information to determine e_i values.

Nitrogen Oxides

Nitrogen dioxide (NO_2) is the primary focus of health studies on the nitrogen oxides and serves as the basis for this analysis. The primary pathophysiology of NO_2 in humans involves the respiratory system and the concentration-response function identified for NO_2 describes the relationships between measures of NO_2 and respiratory illness.

A number of epidemiological studies of NO_2 are available; however, most have either confounded exposures (with other pollutants) or insufficient exposure quantification (e.g., exposure assessment indicates only absence or presence of a gas stove). Most studies consider NO_2 generated by gas stoves or other combustion sources in homes and are therefore not directly usable in concentration-response functions. However, studies by Melia et al, 1980 and Hasselblad et al, 1992 provide a reasonable basis for development of a concentration response function. Table D-8 presents the function obtained from their work. The function relates NO_2 to respiratory illness in children.

Table D-8. Summary of Concentration-Response Functions for NO_2.

Health Endpoint	Exposure Measure from Original Studies	Study Population	Applied Population	Functional Form[a]	Uncertainty/ Variab.	Sources
respiratory illness (as indicated by respiratory symptoms)	NO_2 measurements in bedrooms with Palmes tubes (one year time weighted average concentration in $\mu g/m^3$)	children ages 6 to 7	all (combining functions for men and women)	$\Delta Resp\ cases = \Delta Prob(Resp) * Pop$ where: Prob(resp) = probability of respiratory illness during a one year period: $Prob(resp) = \frac{1}{1 + e^{-\log odds}}$ and $\log odds\ Resp = -0.536 + 0.0275\ NO_2 - 0.0295\ gender$ gender = 1 for boys and 0 for girls (the term drops out for girls)	s.e. = 0.0132	Hasselblad, et al., 1992.

NOTES:

[a] This equation was obtained from two sources. The NO_2 coefficient was reported in Hasselblad et al., 1992. The background and gender intercepts were obtained via personal communication with V. Hasselblad 2/28/95 by Abt Associates. The equation was based on an evaluation by Hasselblad et al. of study results obtained by Melia et al. (1980). See text for further discussion.

Carbon Monoxide

Three concentration-response relationships are available for estimating the health effects of carbon monoxide. The first relates ambient CO levels to hospital admissions for congestive heart failure (Morris et al., 1995). The second equation (Allred et al., 1989a,b, 1991) relates the CO level in the bloodstream to the relative change in time of onset of angina pain upon exertion. The third relates the CO level in the bloodstream to the relative change in time of onset of silent ischemia. Due to the lack of quantitative information relating silent ischemia to a meaningful physical health effect, this analysis uses only the first two dose-response functions shown in Table D-9.

Table D-9. Summary of Concentration-Response Functions for Carbon Monoxide.

Health Endpoint	**Baseline Incidence**	**Exposure Measure**	**Study Population**	**Applied Population**	**Functional Form**	**Uncert./ Variability**	**Sources**
Hospital admiss. for congestive heart failure	n/a	average of hourly max CO (ppm)	Medicare population in 7 large U.S. cities (96% of which are ≥65)	65 and over	Δcases = β * ΔCO * Pop where $\beta = 1.1 \times 10^{-7}$	s.e. = 1.9×10^{-8}	Morris et al., 1995 7 large U.S. cities
percent change in time to angina	baseline time to onset of angina during treadmill test from Allred et al. studies = 515 seconds at %COHb = 0.63[a]	CO (in ppm) averaged over 1 or 8 hours	men, age 35-75 years, stable angina, nonsmokers (of at least 3 months) at time of study	Angina patients in U.S. = 3,080,000 in 1989[b] Frequency of angina attacks for the study population = 4.6 per week (range = 0 - 63)[c, d]	percent change in time to angina =β*Δ%COHb where: β = -1.89% and COHb = blood level of carboxyhemoglobin and Δ %COHb = 0.45 * ΔCO[e], where: CO = concentration of CO (ppm), for non-smoking adults undertaking light exercise (alveolar ventilation rate of 20L/min) for one hour at low altitude, with an initial COHb = 0.5%. OR Δ %COHb = 0.12 * ΔCO[e], where: conditions are the same as above except that study individuals are at rest (alveolar ventilation rate of 10L/min) for 8 hours.	s.e. = 0.81%	Allred, et al., 1989a,b, 1991

NOTES:

[a] Calculated as the mean of means from 3 pre-exposure treadmill tests and 1 post-exposure test (control exposure to air) (Allred et al., 1991).

[b] American Heart Association (1991)

[c] Allred et al. (1991)

[d] Multiple daily events are not modeled. Although it is possible that angina attacks may occur more than once per day, the average frequency of attacks was 4.6 per week (< 1 per day).

[e] Equation calculated from figure in U.S. EPA (1991a), p. 2-7.

Sulfur Dioxide

This analysis estimated one concentration-response function for SO_2 using clinical data from two sources on the responses of exercising asthmatics to SO_2, as measured by the occurrence of respiratory symptoms in mild and moderate asthmatics (see Table D-10).

Table D-10. Summary of Concentration-Response Functions for Sulfur Dioxide.

Health Endpoint	Expos Meas. from original study	Study Pop.	Applied Pop.	Functional Form	Uncert & Var.	Sources
Any Symptom (chest tightness, shortness of breath, etc.)	5-minute SO_2 concentration, ppm (using peak to mean ratio from hourly SO_2 concentration of 2:1 to 3:1)	generally young exercising asthmatics (ventilation rate 0.4 m^3/min)	exercising asthmatics - defined as 4% of general population, of whom 1.7% (range 0.2% to 3.3%) are exercising during waking hours	$\log odds\ Symp = -5.65 + 0.0059\ SO_2 + 1.10\ status$ where *status* = asthma status (0 for mild, 1 for moderate) $Prob(symp) = \frac{1}{1 + e^{-\log odds}}$ $Cases = Prob_{mild}\ (effects) \cdot Pop_{mild} + Prob_{mod}\ (effects) \cdot Pop_{mod}$ Cases = number of individuals with occurrences of at least moderate effects for all three measures. where Pop_{mild} = exposed population of exercising mild asthmatics (assumed to be 2/3 of asthmatic population); Pop_{mod} = exposed population of exercising moderate asthmatics (assumed to be 1/3 of asthmatic population)	s.e. for: const. term= 2.60 for SO_2 coeff = 0.0025 for *status* coeff = 1.44	data from Linn et al.(1987, 1988, 1990), Roger et al. (1985)

Estimating Welfare Effects of Exposure

In addition to avoided incidences of adverse human health effects, the air quality improvements estimated to result from the CAA yield additional benefits, namely welfare benefits. Table D-10 indicates a variety of benefits expected to have accrued through the avoidance of air pollution damage to resources. As indicated, data supporting quantified estimates of welfare benefits are more limited than those quantifying the relationship between air pollution exposure and human health. While evidence exists that a variety of welfare benefits result from air quality improvements, currently available data supports quantifying only a limited number of potential effects at this time. The Table lists the effects quantified in the section 812 analysis; each is discussed below.

Table D-11. Selected Welfare Effects of Criteria Pollutants.

Pollutant	Quantified Welfare Effects	Unquantified Welfare Effects
Ozone	Agriculture - Changes in crop yields (for 7 crops) Decreased worker productivity	Changes in other crop yields Materials damage Ecological - effects on forests Ecological - effects on wildlife
Particulate Matter/ TSP/ Sulfates	Materials Damage - Household soiling Visibility	Other materials damage Ecological - effects on wildlife
Nitrogen Oxides	Visibility	Crop losses due to acid deposition Materials damage due to acid deposition Effects on fisheries due to acid deposition Effects on forest
Sulfur Dioxide	Visibility	Crop losses due to acid deposition Materials damage due to acid deposition Effects on fisheries due to acid deposition Effects on forest

Agricultural Effects

This analysis was able to quantify the benefits to economic welfare attributable to the increased crop yields expected from CAA-related air quality improvements. Appendix F describes the method used to estimate such benefits using reported relationships between ozone exposure and yields of a variety of commodity crops.

It should be noted that the method used to allocate monitor-level ozone concentrations to estimate crop exposure differed from that used to estimate ozone health effects. Instead of assigning concentrations from the nearest monitor, the agricultural benefits analysis estimated ozone concentrations for each county nationwide. This was necessary because of two factors specific to the agricultural analysis. First, crop production is reported at the county level, so changes in crop yields associated with changes in ozone levels must be estimated for each county. Second, much of the nation's agricultural production of "commodity crops" (corn, wheat, soybeans, etc.) occurs at significant distances from the location of the population-oriented ozone monitors. Thus, an algorithm was used to assign ozone concentrations for the agricultural analysis for the control and no-control scenarios to county centroids based on a planar interpolation of concentrations at the nearest three monitors. Appendix F documents the details of the triangulation of ozone air quality data.

Materials Damage

Welfare benefits also accrue from avoided air pollution damage, both aesthetic and structural, to architectural materials and to culturally important articles. At this time, data limitations preclude the ability to quantify benefits for all materials whose deterioration may have been promoted and accelerated by air pollution exposure. However, this analysis does address one small effect in this category, the soiling of households by particulate matter. Table D-11 documents the function used to associate nationwide PM-10 levels with household willingness to pay to avoid the cleaning costs incurred for each additional $\mu g/m^3$ of PM-10.

Visibility

In addition to the health and welfare benefits estimated directly from reduced ambient concentrations of individual criteria air pollutants, this analysis also estimates the general visibility improvements attributed to improved air quality. Visibility effects are measured in terms of changes in DeciView, a measure useful for comparing the effects of air quality on visibility across a range of geographic locations for a range of time periods. It is directly related to two other common visibility measures, visual range (measured in km) and light extinction (measured in km^{-1}); however, it characterizes visibility in terms of perceptible changes in haziness independent of baseline conditions.

Visibility conditions under the control and no-control scenarios were modeled separately for the eastern and western U.S. In the east, the Regional Acid Deposition Model (RADM) generated extinction coefficient estimates for each of 1,330 grid cells in the RADM domain (essentially the eastern half of the country). The extinction coefficients were translated to DeciView using the relationship reported in Pitchford and Malm (1994). In the Western U.S., a conventional extinction budget approach provided DeciView estimates for 30 metropolitan areas (SAI, 1994). A linear rollback model provided the corresponding no-control estimates. Visibility estimates for both portions of the country were generated for the target years 1975, 1980, 1985, and 1990.

Table D-12 summarizes the methodology used to predict visibility benefits attributable to the CAA. Physical benefits for a given year are reported in terms of the average DeciView change per person in the modeled population.

Worker Productivity

Available data permits quantification of a final human welfare endpoint, worker productivity. Crocker and Horst (1981) and U.S. EPA (1994c) present evidence regarding the inverse relationship between ozone exposure and productivity in exposed citrus workers. This analysis applies the worker productivity relationship (reported as income elasticity with respect to ozone) to outdoor workers in the U.S. (approximately one percent of the population). Table D-12 details the form of the concentration response function.

Ecological Effects

It is likely that the air pollution reductions achieved under the CAA resulted in improvements in the health of aquatic and terrestrial ecosystems. To the extent that these ecosystems provide a variety of services (e.g., fishing, timber production, and recreational opportunities), human welfare benefits also accrued. However, due to a lack of quantified concentration-response relationships (or a lack of information concerning affected population), ecological effects were not quantified in this analysis. Appendix E provides discussion of many of the important ecological benefits which may have accrued due to historical implementation of the CAA.

Table D-12. Summary of Functions Quantifying Welfare Benefits.

Endpoint	Expos Meas.	Applied Pop.	Functional Form	Uncert & Var.	Sources
Household Soiling Damage (change in dollar valuation)	annual mean PM_{10}	all households (study based on households in 20 metropolitan areas)	Soiling Damage = β * Pop/PPH * ΔPM_{10} where β = \$2.52 PPH = people per household (2.68)[a]	Beta distribution with mean = \$2.52 s.e. = \$1.00 interval = [\$1.26 - \$10.08] slope parameters: $\alpha = 1.2$, $\beta = 7.3$	Manuel et al. (1982); McClelland, et al. (1991); Watson and Jaksch (1982); ESEERCO (1994)
Visibility (average change in DeciView per person)[b, c]	Eastern U.S.: Extinction coefficient (Ext) in units of m^{-1} Western U.S.: DeciView, dv (unitless)	all	$\Delta Vis = \frac{\sum_i (dv_{No\text{-}CAA,\,i} - dv_{CAA,\,i}) \times Pop_i}{\sum_i Pop_i}$ where, ΔVis = avg. change in DeciView per person in modeled population i = modeled area $dv_{No\text{-}CAA}$ = DeciView under no control scenario dv_{CAA} = DeciView under control scenario Pop_i = modeled population in modeled area, i In the East, Ext (in units of km^{-1}) is converted to dv as follows: $deciview = 10 \ln\left(\frac{Ext}{0.01\ km^{-1}}\right)$	not available	Pitchford and Malm (1994)

Endpoint	Expos Meas.	Applied Pop.	Functional Form	Uncert & Var.	Sources
worker productivity (resulting in changes in daily wages)	hourly O_3 concentration averaged over a workday or 24-hours (ppm)	individuals in occupations that require heavy outdoor physical labor (study based on citrus workers in S. California)	$\Delta I = I*\eta*(X_1 - X_0)/X_0 * Pop * W$ ΔI = change in total daily income, η = income elasticity with respect to O_3 conc., $\eta = -0.14$ for 24-hour period, I = total daily income per worker engaged in strenuous outdoor labor = \$73[d] W = proportion of outdoor workers in the U.S. population = 0.012[e] X_0 = average hourly O_3 concentrations with CAA, X_1 = average hourly O_3 concentrations without CAA (NOTE: Average number of days worked per year for workers engaged in strenuous outdoor labor = 213)[f] (model includes O_3 only)	not available	Estimated using data from Crocker and Horst (1981) and U.S. EPA, 1994c

NOTES:

[a] 1990 Census

[b] Visibility is measured in two ways: (1) in terms of extinction coefficient in the eastern U.S. (based on modeling of RADM domain); and (2) as DeciView (dv) in the west (modeling of 30 western cities) (SAI, 1994).

[c] DeciView is a haziness index used to characterize visibility through uniform hazes.

[d] Average daily wage, assuming an 8-hour day, by workers in the job categories listed below, taken from U.S. Bureau of the Census, Earnings by Occupation and Education, 1990.

[e] Full- and part-time workers (total of 3,100,000) taken from U.S. Bureau of the Census, Earnings by Occupation and Education, 1990. Includes the following job categories: farm workers; groundskeepers and gardeners, except farm; forestry workers, except logging; timber cutting and logging occupations; brickmasons and stonemasons; brickmason and stonemason apprentices; roofers; structural metal workers; construction trades, n.e.c.; construction laborers; garbage collectors; and stevedores. Value is divided by total U.S. population.

[f] Average number of days worked per year, assuming an 8-hour day, by workers in the job categories listed above, taken from U.S. Bureau of the Census, Earnings by Occupation and Education, 1990.

Modeling Results

This section summarizes results of the health and welfare effects modeling. As indicated previously, the Project Team adopted a Monte Carlo approach in an effort to capture uncertainty in the benefits analysis. With respect to estimating avoided incidence of adverse health and welfare effects, two sources of variability are considered. The first is the statistical uncertainty associated with each concentration-response relationship reported in the literature. In addition to an estimate of a concentration-response function coefficient, studies typically report a standard error of the reported estimate. The second source of uncertainty lies in the choice of studies, where multiple studies offer estimates for the same endpoint. Different published results reported in the scientific literature typically do not report identical findings; in some instances the differences are substantial. This between-study variability is captured by considering the range of estimates for a given endpoint.

Table D-13 summarizes health and welfare effects for each study included in the analysis. The values presented are mean estimates of the number of cases of each endpoint *avoided* due to implementation of the CAA. A distribution is associated with each mean estimate, capturing the uncertainty inherent in the estimate of the concentration-response coefficient. The distribution of estimated effects corresponding to a given study was generated by randomly sampling from the distribution of coefficients (given by the estimated coefficient and its standard error reported in the study) and evaluating the concentration-response function, yielding an estimate of avoided incidence for the given effect. This procedure was repeated many times. While only the central estimates of the resulting distributions are presented here, the distributions were retained for use in monetizing and aggregating economic benefits (see Appendix I).[7]

As shown, for some health endpoints more than one concentration-response function was used, each representing a different study. The alternative concentration-response functions provide differing measures of the effect. These can be used to derive a range of possible results. In the case of lead (Pb), alternative functions were not used; rather, two analytical procedures were implemented (labeled the “backward-looking” and “forward looking” analyses), giving a range of results for most Pb endpoints (see Appendix G for discussion of Pb health effects).

The table presents the results of modeling “all U.S. population” (although, with the exception of Pb, not all of the 48 state population is modeled, with up to five percent being excluded in a given year). The results depict the *pattern* of health effects incidence across years. The accuracy of the *scale* of incidence is less certain (due to the extrapolation of air quality data). These results are almost certainly more accurate than the corresponding “50 km” results, but rely on the assumption that (for a portion of the population) distant air quality monitors provide a reasonable estimate of local air quality conditions. Thus, the results presented here are somewhat speculative. It is likely that the estimated health effects are overstated for that population group (20 to 30 percent of total population in the case of PM) for which distant monitors are used. (Note, however, that the scaling of unmonitored county PM concentrations based on regional-scale grid model projections significantly mitigates this potential overestimation in the case of PM; see Appendix C for details). Conversely, there is an implied zero health impact for that portion of the population (three to four percent in the case of PM) excluded from the analysis altogether, an understatement of health impacts for that group.

The results indicate the growth of benefits over the study period, consistent with increasing improvements in air quality between the control and no-control scenarios from 1970 to 1990.

The mortality effects documented above can be disaggregated by age. Table D-14 indicates the estimated proportions of premature mortalities for various age groups (Pb-induced mortality estimates for children, men, and women are grouped). Also presented is the average life expectancy for each group, indicating the degree of prematurity of PM and Pb-related mortality.

Table D-15 presents estimated incidence reductions for several health effects which could be quantified but not monetized for this analysis.

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[7] With the exception of visibility, welfare endpoints estimated economic benefits directly and are therefore included in the monetary benefits results presented in Appendix I.

Table D-13. Criteria Pollutants Health Effects -- Extrapolated to 48 State U.S. Population (Cases per year - mean estimates).

Endpoint	Study	Pollutant(s)	1975	1980	1985	1990
MORTALITY						
Mortality (long-term exposure)	Pope et al., 1995	PM_{10}	58,764	145,884	169,642	183,539
Mortality (Pb exposure) -Male	Average of Backward & Forward	Pb	822	5,281	10,340	12,819
Mortality (Pb exposure) -Female	Average of Backward & Forward	Pb	231	1,474	2,866	3,537
Mortality (Pb exposure) -Infant	Average of Backward & Forward	Pb	456	2,342	3,933	4,944
CHRONIC BRONCHITIS						
Chronic Bronchitis	Schwartz, 1993b	PM_{10}	198,973	554,632	720,166	741,775
	Abbey et al., 1993	PM_{10}	173,571	454,309	564,753	602,990
OTHER Pb-INDUCED AILMENTS						
Lost IQ Points	Average of Backward & Forward	Pb	1,028,492	5,031,157	8,559,426	10,378,268
IQ < 70	Average of Backward & Forward	Pb	3,780	20,074	36,520	45,393
Hypertension-Men	Average of Backward & Forward	Pb	830,299	5,276,999	10,087,115	12,646,876
Cor. Heart Disease	Average of Backward & Forward	Pb	1,313	8,444	16,671	21,069
Atherothrombotic brain infarction - Men	Average of Backward & Forward	Pb	181	1,128	2,165	2,690
Atherothrombotic brain infarction - Women	Average of Backward & Forward	Pb	84	529	1,020	1,255
Initial cerebrovascular accident - Men	Average of Backward & Forward	Pb	260	1,635	3,154	3,926
Initial cerebrovascular accident - Women	Average of Backward & Forward	Pb	120	758	1,466	1,804
HOSPITAL ADMISSIONS						
All Respiratory	Schwartz, 1995, Tacoma	PM_{10} & O3	32,004	77,827	95,435	106,777
	Schwartz, 1996, Spokane	PM_{10} & O3	29,393	69,449	93,137	119,290
	Pope, 1991, Salt Lake Valley	PM_{10}	30,982	73,093	86,407	95,486
	Schwartz, 1995, New Haven	PM_{10} & O3	23,137	55,096	66,385	73,842
	Thurston et al., 1994, Toronto	PM_{10} & O3	13,746	32,383	39,691	46,013
COPD + Pneumonia	Schwartz, 1994c	PM_{10} & O3	21,898	53,928	64,217	70,528
	Schwartz, 1996, Spokane	PM_{10} & O3	19,769	47,294	63,116	80,113
	Schwartz, 1994a	PM_{10} & O3	16,942	40,882	49,290	55,227
	Schwartz, 1994b	PM_{10} & O3	13,006	30,679	37,434	43,410
Ischemic Heart Disease	Schwartz and Morris, 1995	PM_{10}	6,348	14,709	17,289	19,098
Congestive Heart Failure	Schwartz and Morris, 1995	PM_{10}	5,733	13,365	15,742	17,362
	Morris et al., 1995	CO	3,022	8,543	17,028	21,835
OTHER RESPIRATORY-RELATED AILMENTS						
- Adults						
Any of 19 Acute Symptoms	Krupnick et al., 1990	PM_{10} & O3	41,631,456	98,876,110	117,275,400	129,529,717
- Children						
Shortness of breath, days	Ostro et al., 1995	PM_{10}	20,752,402	50,758,872	58,575,484	68,375,216
Acute Bronchitis	Dockery et al., 1989	PM_{10}	1,936,260	6,255,801	7,644,924	8,541,833
Lower Respiratory Symptoms	Schwartz et al., 1994d	PM_{10}	2,994,048	6,100,276	6,977,680	7,804,860
Upper Respiratory Symptoms	Pope et al., 1991	PM_{10}	500,395	1,292,922	1,557,177	1,683,854
- All Ages						
Asthma Attacks	Ostro et al., 1991	PM_{10}	264,430	548,306	686,953	841,916
	Whittemore and Korn, 1980; EPA ,1983	O3	193	482	816	1,080
Increase in Respiratory Illness	Hasselblad, 1992	NO2	729,306	2,686,813	6,113,639	9,776,267
Any Symptom	Linn et al. (1987, 1988, 1990)	SO2	104,896	319,192	282,846	265,650
RESTRICTED ACTIVITY AND WORK LOSS DAYS						
RAD	Ostro, 1987	PM_{10}	19,170,337	47,445,314	56,939,271	62,187,720
MRAD	Ostro and Rothschild, 1989	PM_{10} & O3	60,871,610	155,799,151	190,333,140	209,924,785
RRAD	Ostro and Rothschild, 1989	PM_{10} & O3	47,669,732	237,799,482	176,850,171	174,329,691
Work Loss Days	Ostro, 1987	PM_{10}	6,966,775	17,213,581	20,648,906	22,562,752
HUMAN WELFARE						
Household Soiling Damage	ESEERCO, 1994	PM_{10}	direct economic valuation			
Visibility - East (DeciView chg. per person)	Pitchford and Malm, 1994	DeciView	0.4	1.4	1.9	2.0
Visibility - West (DeciView chg. per person)	Pitchford and Malm, 1994	DeciView	2.4	4.9	5.0	6.0
Decreased Worker Productivity	Crocker & Horst, 1981 and EPA, 1994c	O3	direct economic valuation			
Agriculture (Net Surplus)	Minimum Estimate	O3	direct economic valuation			
	Maximum Estimate	O3	direct economic valuation			

Table D-14. Mortality Distribution by Age: Proportion of PM- and Pb-related Premature Mortalities and Associated Life Expectancies.

Age Group	**Proportion of Premature Mortalities by Age**[a]		**Life Expectancy (years)**
	PM[b]	**Pb**[c] **Forward (Backward)**[d]	
Infants		33% (20%)	75
5-30			
30-34	2%		48
35-39	4%		38
40-44		11% (13%)	
45-54	6%	21% (25%)	29
55-64	13%	22% (27%)	21
65-74	24%	12% (15%)	14
75-84	29%		9
85+	22%		6
	100%	100%	

Notes:

[a] Distribution of premature mortalities across ages is fairly consistent across years.

[b] PM-related mortality incidence estimated only for individuals 30 years and older, consistent with the population studied by Pope et al., 1995.

[c] Pb-related mortality incidence was estimated for infants, women aged 45-74, and men in three age groups (40-54, 55-64, 65-74), each with a distinct concentration-response relationship.

[d] Forward (backward) analysis holds other lead sources at constant 1970 (1990) levels - see Appendix G. Values may not sum to 100% due to rounding.

Table D-15. Quantified Benefits Which Could Not Be Monetized -- Extrapolated to the Entire 48 State Population.

Endpoint	Study	Pollutant	1975	1980	1985	1990	Units
Pulmonary Function Decrements							
Decreased FEV by 15 % or more	Avol et al. 1984 & Seal et al. 1993	O3	53	121	196	312	*million person-days with decreased FEV (per year)*
Decreased FEV by 20 % or more	Avol et al. 1984 & Seal et al. 1993	O3	39	87	141	224	*million person-days with decreased FEV (per year)*
Chronic Sinusitis and Hay Fever	Portney and Mullahy, 1990	O3	6	8	8	9	*million cases/year*
Time to Onset of Angina Pain	Allred, et al., 1989a,b, 1991	CO	0.1%	0.4%	0.7%	0.8%	*fractional increase in time to onset of angina attack*

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Appendix E: Ecological Effects of Criteria Pollutants

Introduction

Benefits to human welfare from air pollution reductions achieved under the CAA can be expected to arise from likely improvements in the health of aquatic and terrestrial ecosystems and the myriad of ecological services they provide. For example, improvements in water quality stemming from a reduction in acid deposition-related air pollutants (e.g., SO_x and NO_x) could benefit human welfare through enhancements in certain consumptive services such as commercial and recreational fishing, as well as non-consumptive services such as wildlife viewing, maintenance of biodiversity, and nutrient cycling. Increased growth and productivity of U.S. forests could result from reduced emissions of ozone-forming precursors, particularly VOCs and NO_x, and thus may yield benefits from increased timber production; greater opportunities for recreational services such as hunting, camping, wildlife observation; and nonuse benefits such as nutrient cycling, temporary CO_2 sequestration, and existence value.

In this Appendix, the potential ecological benefits from CAA pollutant controls are discussed in the context of three types of ecosystems: aquatic, wetland, and forest. In describing the potential ecological benefits of the CAA, it is clearly recognized that this discussion is far from being comprehensive in terms of the types and magnitude of ecological benefits that may actually have occurred from the implementation of the CAA. Rather, this discussion reflects current limitations in understanding and quantifying the linkages which exist between air quality and ecological services, in addition to limitations in the subsequent valuation of these services in monetary terms. This discussion also does not cover potential benefits from improvements in other ecological services, namely agriculture and visibility, which are discussed and quantified in other sections of this report. This appendix is dedicated to a *qualitative* evaluation of ecological benefits. However, where possible, the existing body of scientific literature is drawn upon in an attempt to provide insights to the possible magnitude of benefits that may have resulted from CAA-related improvements of selected ecological services. It is important to note that the inability to fully value ecological services results in a significant undervaluation of the ecological benefits of air pollution reductions. This undervaluation should not be interpreted as a devaluation.

Benefits From Avoidance of Damages to Aquatic Ecosystems

Aquatic ecosystems (lakes, streams, rivers, estuaries, coastal areas) provide a diverse range of services that benefit the welfare of the human population. Commercially, aquatic ecosystems provide a valuable food source to humans (e.g., commercial fish and shellfish harvesting), are used for the transportation of goods and services, serve as important drinking water sources, and are used extensively for irrigation and industrial processes (e.g., cooling water, electrical generation). Recreationally, water bodies provide important services that include recreational fishing, boating, swimming, and wildlife viewing. They also provide numerous indirect services such as nutrient cycling, and the maintenance of biological diversity.

Clearly, these and other services of aquatic ecosystems would not be expected to be equally responsive to changes in air pollution resulting from the implementation of the CAA. The available scientific information suggests that the CAA-regulated pollutants that can be most clearly linked to effects on aquatic resources include SO_x and NO_x (through acid deposition and increases in trace element bioavailability), NO_x (through eutrophication of nitrogen-limited water bodies), and mercury (through changes in atmospheric deposition). Potential benefits from each of these processes (acid deposition, eutrophication, mercury accumulation in fish) are described separately in the following sections.

Acid Deposition

Background

Acid deposition refers to the depositing of strong acids (e.g., H_2SO_4, HNO_3) and weak acids ($(NH_4)_2SO_4$, NH_4NO_3) from the atmosphere to the earth's surface. Acid deposition can occur in the wet or dry form and can adversely affect aquatic resources through the acidification of water bodies and watersheds. Acidification of aquatic ecosystems is of primary concern because of the adverse effects of low pH and associated high aluminum concentrations on fish and other aquatic organisms. Low pH can produce direct effects on organisms, through physiological stress and toxicity processes, and indirect effects, mediated by population and community changes within aquatic ecosystems. Acidification can affect many different aquatic organisms and communities. As pH decreases to 5.5, species richness in the phytoplankton, zooplankton, and benthic invertebrate communities decreases.[1] Additional decreases in pH affect species richness more significantly, and may sometimes affect overall biomass.[2] Table E-1 presents descriptions of the biological effects of acidification at different pH levels. In evaluating the severity of biological changes due to acidification, the reversibility of any changes is an important consideration; biological populations and communities may not readily recover from improved water quality under certain circumstances. Researchers have addressed acidification effects through many different experimental protocols, including laboratory bioassays, particularly concerning pH, aluminum, and calcium; manipulative whole-system acidification studies in the field; and comparative, nonmanipulative field studies.

Although acidification affects phytoplankton, zooplankton, benthic invertebrates, fish, amphibians, and waterfowl, most acidification research has concentrated on fish populations.[3] Aluminum, which can be toxic to organisms, is soluble at low pH and is leached from watershed soils by acidic deposition.[4] Acidification may affect fish in several ways. The direct physiological effects of low pH and high aluminum include increased fish mortality, decreased growth, and decreased reproductive potential. The mechanism of toxicity involves impaired ion regulation at the gill.[5] Population losses occur frequently because of recruitment failure,[6] specifically due to increased mortality of early life stages.[7] Changes at other trophic levels may affect fish populations by altering food availability.[8] Fish in poorly buffered, low pH water bodies may accumulate higher levels of mercury, a toxic metal, than in less acidic water bodies, due to increased mercury bioavailability. The primary consequence of mercury accumulation appears to be hazardous levels to humans and wildlife who consume fish, rather than direct harm to aquatic organisms (discussed further below).

The CAA-regulated pollutants that are likely to have the greatest effect on aquatic ecosystems through acid deposition and acidification are SO_2 and NO_x. In the atmosphere, SO_2 and NO_x react to form sulfate and nitrate particulates, which may be dry-deposited; also the pollutants may react with water and be wet-deposited as dilute sulfuric and nitric acids. SO_2 is considered the primary cause of acidic deposition, contributing 75 to 95 percent of the acidity in rainfall in the eastern United States.[9]

Current Impacts of Acid Deposition

Effects on Water Chemistry

The effects of acid deposition and resulting acidification of water bodies was intensively studied as part of a 10-year, congressionally-mandated study of acid rain problems in the United States.[10] Based on the NAPAP study, it is estimated that 4 percent of the lakes and 8 percent of the streams in acid-sensitive

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[1] J. Baker et al., NAPAP SOS/T 13, 1990; Locke, 1993.

[2] J. Baker et al., NAPAP SOS/T 13, 1990.

[3] NAPAP, 1991.

[4] J. Baker et al., NAPAP SOS/T 13, 1990.

[5] J. Baker et al., NAPAP SOS/T 13, 1990.

[6] Rosseland, 1986.

[7] J. Baker et al., NAPAP SOS/T 13, 1990.

[8] Mills et al., 1987.

[9] NAPAP, 1991.

[10] NAPAP, 1991.

Table E-1. Summary of Biological Changes with Surface Water Acidification.

pH Decrease	**Biological Effects**
6.5 to 6.0	Small decrease in species richness of phytoplankton, zooplankton, and benthic invertebrate communities resulting from the loss of some acid-sensitive species, but no measurable change in total community abundance or production. Some adverse effects (decreased reproductive success) may occur for acid-sensitive fish species (e.g., fathead minnow, striped bass).
6.0 to 5.5	Loss of sensitive species of minnows and dace, such as blacknose dace and fathead minnow; in some waters decreased reproductive success of lake trout and walleye. Distinct decrease in the species richness and change in species composition of the phytoplankton, zooplankton, and benthic invertebrate communities. Loss of a number of common invertebrate species from the zooplankton and benthic invertebrate communities, including zooplankton species such as *Diaptomus silicis*, *Mysis relicta*, *Epischura lacustris*; many species of snails, clams, mayflies, and amphipods, and some crayfish. Visual accumulations of filamentous green algae in the littoral zone of many lakes and in some streams.
5.5 to 5.0	Loss of several important sport fish species, including lake trout, walleye, rainbow trout, and smallmouth bass; as well as additional non-game species such as creek chub. Continued shift in the species composition and decline in species richness of the phytoplankton, periphyton, zooplankton, and benthic invertebrate communities; decreases in the total abundance and biomass of benthic invertebrates and zooplankton may occur in some waters. Loss of several additional invertebrate species common in oligotrophic waters, including *Daphnia galeata mendotae*, *Diaphanosoma leuchtenbergianum*, *Asplancha priodonta*; all snails, most species of clams, and many species of mayflies, stoneflies, and other benthic invertebrates. Inhibition of nitrification. Further increase in the extent and abundance of filamentous green algae in lake littoral areas and streams.
5.0 to 4.5	Loss of most fish species, including most important sport fish species such as brook trout and Atlantic salmon. Measurable decline in the whole-system rates of decomposition of some forms of organic matter, potentially resulting in decreased rates of nutrient cycling. Substantial decrease in the number of species of zooplankton and benthic invertebrates, including loss of all clams and many insects and crustaceans; measurable decrease in the total community biomass of zooplankton and benthic invertebrates in most waters. Further decline in the species richness of the phytoplankton and periphyton communities. Reproductive failure of some acid-sensitive species of amphibians such as spotted salamanders, Jefferson salamanders, and the leopard frog.

Source: Baker, J. et al. (NAPAP SOS/T 13, 1990), p. 13-173.

regions of the U.S. are chronically acidic due to natural and anthropogenic causes. NAPAP defines acidic conditions as occurring when the acid neutralizing capacity[11] (ANC) is below 0 µeq/L. Furthermore, approximately 20 percent of the streams and lakes in these regions are considered to be extremely susceptible to acidity (defined as ANC <50 µeq/L) and slightly more than half show some susceptibility to acidification (defined as ANC <200 µeq/L).

In terms of the role of acid deposition as a causal mechanism for the acidification of water bodies, it is estimated that 75 percent of the 1,181 acidic lakes and 47 percent of the 4,668 streams studied under

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[11] ANC is expressed in units of microequivalents per liter (µeq/L), where an equivalent ANC is the capacity to neutralize one mole of H^+ ions. Generally, waters with an ANC < 0 have corresponding pH values of less than 5.5 (L. Baker et al., NAPAP SOS/T 9, 1990).

NAPAP receive their dominant source of acid anions from atmospheric deposition (see Table E-2). On a regional basis, the importance of acid deposition varies considerably, which is believed to result from regional differences in SO_x and NO_x emissions and differences in the biogeochemistry of individual watersheds. For acidic lakes (ANC <0), the regions that appear most likely to be influenced by acid deposition include the Adirondacks and Mid-Atlantic Highland region, with acid deposition cited as the dominant source of acidity in 100 percent of the acidic lakes studied (Table E-2). This is in stark contrast to the West region, where none of the acidic lakes studied were dominated by acid deposition (notably, the sample size of lakes for this region was small to begin with). For acidic streams, the Mid-Atlantic Highland region contains the greatest proportion of streams whose acidic inputs are dominated by acid deposition (56 percent). This contrasts with acidic streams of Florida, where the vast majority (79 percent) are acidic primarily due to organic acids, rather than acid deposition.

Table E-2. Comparison of Population of Acidic National Surface Water Survey (NSWS) by Chemical Category[1]

Region	Number of Acidic Waters	Deposition Dominated (%)	Organic Dominated (%)	Acid Mine Drainage Dominated (%)	Watershed Sulfate Dominated (%)
LAKES					
New England	173	79	21	--	--
Adirondacks	181	100	--	--	--
Mid-Atlantic Highlands	88	100	--	--	--
Southeastern Highlands	--	--	--	--	--
Florida	477	59	37	--	4
Upper Midwest	247	73	24	--	3
West	15	--	--	--	100
All Lakes	1,181	75	22	--	3
STREAMS					
Mid-Atlantic Highlands	2,414	56	--	44	--
Mid-Atlantic Coastal Plain	1,334	44	54	--	2
Southeastern Highlands	243	50	--	50	--
Florida	677	21	79	--	--
All Streams	4,668	47	27	26	<1

[1] Source: NAPAP 1991 (Table 2.2-3, p. 28).

Effects on Fish Habitat Quality

By combining information on relevant water chemistry parameters (pH, aluminum, calcium), fish toxicity models, and historical and current distributions of fish populations in the lakes and streams included in the National Surface Water Survey (NSWS), NAPAP investigators estimated the proportion of water bodies with water chemistry conditions that are unsuitable for survival of various fish species.[12] In the Adirondack region, where the acidic lakes are dominated by acid deposition, it is estimated that ten percent of the lakes are unsuitable for the survival of acid-tolerant fish species such as brook trout; twenty percent of the lakes are estimated to be unsuitable for the survival of acid-sensitive species such as minnows. About two percent and six percent of the lakes in the New England region are estimated to be unsuitable for acid-tolerant and acid-sensitive fish species, respectively. A greater proportion of streams in the Mid-Atlantic Highland region are estimated to be unsuitable for acid-tolerant and acid-resistant fish species (18 percent and 30 percent, respectively); however, about 44 percent of streams surveyed in this region are thought to be heavily influenced by acid mine drainage (Table E-2).

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[12] NAPAP, 1991.

Economic Damages to Recreational Fishing

In an effort to assess some of the impacts from *existing* levels of acid deposition to public welfare, NAPAP investigated the current economic damages associated with acid deposition to trout anglers of New York, Maine, Vermont, and New Hampshire. The general approach used consisted of linking the catch per unit effort (CPUE) for four species of trout at individual lakes (estimated using participation survey data) to the relevant water quality conditions at these lakes (namely, the acid stress index or ASI). Using historical water quality data, critical water quality conditions (i.e., the ASI values) were estimated for lakes in the absence of acid deposition and compared to current conditions reflecting the presence of acid deposition. Using two types of travel cost models, the Random Utility Model (RUM) and Hedonic travel-cost model (HTCM), estimates of the willingness to pay (WTP) per trip of sampled trout anglers were obtained. Aggregate estimates of the WTP were obtained across the populations of trout anglers using statistical weighting factors. Finally, the difference in total WTP between the current (acid deposition) scenario and the historical (acid deposition-free) scenarios was determined.

The resulting estimates of economic damages to trout anglers in the four state region are relatively small. Specifically, damage estimates range from $0.3 million to $1.8 million (in 1989 dollars) for the hedonic travel-cost and random utility models, respectively. By many accounts, these estimates can be considered to underestimate actual damages to anglers in these states. First, data limitations precluded the development of meaningful WTP estimates for brook trout anglers, which may be a significant component of trout fishing in these areas. Second, resource constraints necessitated exclusion of a large population of trout anglers (i.e., those residing in New York City). Third, the economic damage estimates were limited to trout anglers, thus excluding potentially similar if not greater economic damages to anglers fishing for other coldwater or warmwater fish species. In addition, the NAPAP analysis was performed in the context of recreational fishing in lakes, thereby excluding potentially important welfare impacts from recreational fishing in streams. Finally, these estimates do not address non-use values of lakes in this region.

Benefits From Acid Deposition Avoidance Under the CAA

It is currently estimated that in the absence of pollution reductions achieved under the Clean Air Act, total sulfur emissions to the atmosphere would have increased by nearly sixteen million tons by 1990, a 40 percent increase above 1990 levels estimated with CAA controls remaining in place.[13] Based on atmospheric transport and deposition modeling, this increase in sulfur emissions corresponds to an approximate 25 to 35 percent increase in total sulfur deposition (wet & dry) in large portions of the northeastern portion of the United States.[14] Given sulfur emission and deposition changes of this magnitude, and the importance of sulfur emissions in contributing to acid deposition, one would expect some benefits to human welfare to be achieved as a result of improved quality of aquatic ecosystems. To date, however, no formal benefits assessment of CAA-avoided acid deposition impacts has been conducted for aquatic ecosystems. Nevertheless, past benefit assessments involving acid deposition impacts on aquatic ecosystems provide some opportunity to gain insights into the relative magnitude of certain aquatic-based benefits that may be achieved through pollution reductions under the CAA.[15]

Recreational Fishing

NAPAP evaluated the impact of changes in acid deposition on use values of aquatic ecosystems (i.e., recreational fishing).[16] In their integrated assessment, NAPAP valued the impacts of three different sulfur-induced acid deposition scenarios to trout anglers from NY, VT, NH and ME.[17] The three scenarios evaluated were:

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[13] U.S. EPA, 1995; Table B-2.

[14] U.S. EPA 1995, p. 3-10.

[15] See, for example, NAPAP, 1991.

[16] NAPAP, 1991.

1. No change in acid deposition.
2. A 50 percent reduction in acid deposition.
3. A 30 percent increase in acid deposition.

As described above, equations were developed by NAPAP to estimate the catch per hour for species at each lake as a function of the ASI value for each lake and of the technique of the fishers. Baseline and predicted changes in CPUE were evaluated for all lakes modeled in the region. Willingness-to-pay estimates for CPUE per trip were derived for the baseline and sulfur emission scenarios using two travel-cost models, a random utility model and a hedonic travel cost model. These willingness-to-pay estimates were then combined with the results of a participation model that predicted the total number of trips taken by trout anglers. Total welfare changes were determined over a 50 year period (from 1990 to 2040).

At current levels of acid deposition, NAPAP estimates that trout anglers in these four states will experience annual losses by the year 2030 of $5.3 or $27.5 million (in 1989 dollars) for the random utility model and hedonic travel cost model, respectively (see Table E-3). If acid deposition *increases* by 30 percent, which roughly corresponds to the 25 to 35 percent increase predicted for the northeast U.S. in the absence of CAA sulfur controls,[18] the resulting economic losses to trout anglers in 2030 would range from $10 million to nearly $100 million annually (in 1989 dollars) for the RUM and HTCM, respectively. If deposition decreases by 50 percent, annual benefits to recreational anglers are estimated to be $14.7 million (RUM) or $4.2 million (HTCM).

While an estimation of CAA-related benefits to trout anglers based on the 30 percent increase in acid deposition scenario has some appeal, a strict transfer of these benefits to the section 812 retrospective analysis is hindered by several factors. First, the NAPAP benefits estimates are projected for future conditions (the year 2030). Therefore, the extent to which the NAPAP benefits reflect conditions and benefits in 1990 (the focus of the section 812 retrospective assessment) is unclear. Second, the NAPAP and CAA section 812 analyses operate from different baselines (1990 for the NAPAP study versus 1970-1990 for the section 812 study). However, the NAPAP estimates of annual benefits of $10 to $100 million provide a rough benchmark for assessing the likely magnitude of the avoided damages to an important and sensitive recreational fishery in a four-state area most impacted by surface water acidification from atmospheric deposition.

Table E-3. Results from Benefits Assessments of Aquatic Ecosystem Use Values from Acid Deposition Avoidance.

Study	Use Value	Scenario Modeled	Method	Annual Benefits
NAPAP (1991)	Trout Fishing	No change in acid deposition	RUM HTCM	-$5.3 million - $27.5 million
	(NY, ME, VT, NH)	50% decrease in acid deposition	RUM HTCM	$14.4 million $4.2 million
		30% increase in acid deposition	RUM HTCM	-$10.3 million -$97.7 million
		No new emission reductions after 1985	RUM HTCM	-$5.5 million -$3.5 million
		10 million ton reduction of SO$_2$ from 1980 levels by 2000	RUM HTCM	$9.7 million $4.4 million

Eutrophication

Eutrophication is the process by which aquatic systems respond to nutrient enrichment. The most common nutrients involved in eutrophication are nitrogen and phosphorous (and related chemical species). When water bodies receive excessive amounts of nutrients, adverse impacts on their resident species and on ecosystem functions can occur from excessive algal growth and the reduction in dissolved oxygen caused by decaying algal biomass. Under highly eutrophic conditions, excessive nutrients can cause depleted oxygen levels that result in subsequent loss of economically important benthic organisms (shellfish), fish kills, and changes in phytoplankton, zooplankton, and fish community composition.[19] Nuisance algal blooms can have numerous economic and biological costs, including water quality deterioration affecting biological resources, toxicity to vertebrates and higher invertebrates, and decreased recreational and aesthetic value of waters.[20] Although severe eutrophication is likely to adversely affect organisms, especially fish, a moderate increase in nutrient levels may also increase fish stocks, by increasing productivity in the food chain.[21]

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[17] NAPAP, 1991; p. 383-384.

[18] U.S. EPA, 1995.

Atmospheric Deposition and Eutrophication

The deposition of NO_x in aquatic systems and their watersheds is one source of nitrogen that may contribute to eutrophication. The relative importance of NO_x deposition as a contributor to aquatic eutrophication depends on the extent to which the productivity of an aquatic ecosystem is limited by nitrogen availability and the relative importance of nitrogen deposition compared to other internal and external sources of nitrogen to the aquatic ecosystem. Furthermore, the vulnerability of aquatic ecosystems to eutrophication is known to vary seasonally and spatially, although these systems are affected by nutrient deposition throughout the year. In general, freshwater ecosystems appear to be more often limited by phosphorus, rather than nitrogen, and are not as likely to be heavily impacted by nitrogen deposition compared to some estuarine and coastal ecosystems.[22] In contrast to acidification of streams and lakes, eutrophication from atmospheric deposition of nitrogen is more commonly found in coastal and estuarine ecosystems, which are more frequently nitrogen-limited.[23]

Unfortunately, there is limited information with regard to the relative importance of atmospheric deposition as a nitrogen source in many estuarine and marine ecosystems. Estimates of the importance of atmospheric nitrogen deposition are difficult to make because of uncertainties in estimating deposition, especially dry deposition, as well as watershed nitrogen retention.[24] Paerl (1993) reviews the importance of atmospheric nitrogen deposition as a contributor to eutrophication of coastal ecosystems; he concludes that 10 to 50 percent of the total nitrogen loading to coastal waters is from direct and indirect atmospheric deposition. Estimates for the economically important Chesapeake Bay indicate that about 25 to 40 percent of the nitrogen loadings to the bay occur via atmospheric deposition.[25] Hinga et al. (1991) estimate that anthropogenic deposition provides 11 percent of total anthropogenic inputs of nitrogen in Narragansett Bay, 33 percent for the New York Bight, and 10 percent for New York Bay. Fisher and Oppenheimer (1991) estimate that atmospheric nitrogen provides 23 percent of total nitrogen loading to Long Island Sound and 23 percent to the lower Neuse River in North Carolina. Information on the importance of atmospheric nitrogen deposition for most other U.S. coastal ecosystems is not available in the literature. Episodic atmospheric inputs of nitrogen may be an important source of nitrogen to nutrient-poor marine ecosystems, such as the North Atlantic near Bermuda and the North Sea.[26]

Valuing Potential Benefits from Eutrophication Avoidance Under the CAA

It is currently estimated that in the absence of pollution reductions achieved under the Clean Air Act, total nitrogen emissions to the atmosphere would have increased by nearly 90 million tons by 1990, a two-fold increase above 1990 levels estimated with CAA controls remaining in place.[27] However, the ability to determine the potential economic benefit from such a reduction in nitrogen emissions is heavily constrained by gaps in our current biological and economic knowledge base of aquatic ecosystems.

One water body that has received much study in the area of nitrogen-induced eutrophication is Chesapeake Bay. As previously discussed, it is estimated that atmospheric deposition of nitrogen contributes approximately 25 percent to the total nitrogen loadings to the bay.[28] In deposition terms, an estimated 15 to more than 25 percent increase in total nitrogen deposition has been forecast in the Chesapeake Bay watershed by 1990 in the absence of CAA pollution controls.[29] These results are based on an estimated 40,000 tons of atmospherically deposited nitrogen (as nitrate and ammonia) to Chesapeake Bay in 1985,[30] which means a 20 percent increase in atmospheric deposition would amount to approximately 8,000 additional tons.

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[19] Paerl, 1993.

[20] Paerl, 1988.

[21] Hansson and Rudstam, 1990; Rosenberg et al., 1990; Paerl, 1993.

[22] Hecky and Kilham, 1988; Vitousek and Howarth, 1991.

[23] U.S. EPA, 1993; Paerl, 1993.

[24] U.S. EPA, 1993.

[25] U.S. EPA, 1994.

[26] Owens et al., 1992.

[27] U.S. EPA, 1995; Table B-3.

One indirect method available to gauge the potential economic relevance of avoidance of such atmospheric nitrogen loadings to Chesapeake Bay is through the avoidance cost of nitrogen controls. However, such an assessment is difficult due to the site, facility, and treatment-specific variation in treatment costs. For example, Camacho (1993) reviewed nitrogen treatment costs for chemical treatment of water from important point sources (mostly public owned treatment works) and found that costs ranged from $9,600 to $20,600 per ton (annual costs, 1990 dollars), depending on the facility evaluated. Biological treatment of nitrogen from point sources was far more expensive, varying from $4,000 to $36,000 per ton. For control of non-point source loading, values of nitrogen removal practices ranged from $1,000 to $285,000 per ton.[31] Taking chemical addition as one possible example, the avoided costs of treatment of 8,000 tons of nitrogen would range from about $75 million to about $170 million annually (in 1990 dollars).

Mercury

Mercury, in the form of methyl mercury, is a neurotoxin of concern and can accumulate in tissue of fish to levels that are hazardous to humans and aquatic-feeding wildlife in the U.S. In relation to the section 812 CAA retrospective analysis, mercury is of interest for two reasons. First, potential benefits to human welfare may have occurred as a result of mercury emission controls implemented under EPA's National Emission Standards for Hazardous Air Pollutants (NESHAP). Second, experimental and observational evidence suggests that acidification of water bodies enhances mercury accumulation in fish tissues.[32] Therefore, CAA-mandated reductions in sulfur and nitrogen oxide emissions and subsequent acid deposition may have resulted in indirect benefits from a reduction in mercury accumulation in fish and subsequent improvements to human health and welfare.

The accumulation of mercury to hazardous levels in fish has become a pervasive problem in the U.S. and Canada. A rapid increase in advisories occurred during the 1980s, including a blanket advisory affecting 11,000 lakes in Michigan.[33] The Ontario Ministries of Environment and Natural Resources (1990) recommend fish consumption restrictions for 90 percent of the walleye populations, 80 percent of smallmouth bass populations, and 60 percent of lake trout populations in 1,218 Ontario lakes because of mercury accumulation. In many instances, mercury has accumulated to hazardous levels in fish in highly remote water bodies that are free from direct aqueous discharges of mercury.[34] Mass balance studies have shown that atmospheric deposition of mercury can account for the accumulation of mercury in fish to high levels in lakes of these remote regions.[35] The potential impacts of mercury on the health of humans and fish-eating (piscivorous) wildlife has lead EPA to recently establish water quality criteria to protect piscivorous species in the Great Lakes.[36]

Although mercury accumulation in fish via atmospheric deposition is now widely recognized as a potential hazard to human health and certain wildlife species, studies establishing quantitative linkages between sources of mercury emissions, atmospheric deposition of mercury, and subsequent accumulation in fish are lacking. Thus at the present time, we are unable to quantify potential benefits from CAA-avoided mercury accumulation in fish of U.S. water bodies. Given the pervasiveness of the mercury problem with CAA-pollution controls, potential benefits to human health and welfare from avoidance of further mercury related damages to aquatic ecosystems could be substantial.

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[28] U.S. EPA, 1993.

[29] U.S. EPA 1995, Figure C-6.

[30] NERA, 1994.

[31] Shuyler, 1992.

[32] Bloom et al., 1991; Watras and Bloom, 1992; Miskimmin et al., 1992; Spry and Wiener, 1991; Wiener et al., 1990.

[33] Watras et al., 1994.

[34] Glass et al., 1990; Sorenson et al., 1990; Grieb et al. 1990; Schofield et al. 1994.

[35] Fitzgerald et al. ,1991.

[36] U.S. EPA, 1995.

It should also be noted that atmospheric deposition is a major contributor to surface water loads of other toxic pollutants as well. For example, scientists believe that about 35 to 50 percent of the annual loadings of a variety of toxic chemicals to the Great Lakes may be from the air; for lead, atmospheric deposition currently accounts for an estimated 95 percent of the total load in the Great Lakes.[37] CAA-related reductions in air emissions of toxic pollutants (such as lead) undoubtedly reduced the loading of these chemicals to the Great Lakes and other water bodies; the magnitude of the benefits of reducing these exposures to humans and wildlife is not known.

Benefits from Avoided Damages to Wetland Ecosystems

Introduction

This review addresses the effects of air pollutants on wetland ecosystems; the focus is on acidification and nutrient loading. Valuable service flows of wetland ecosystems include flood control, water quality protection and improvement, wildlife and fish habitat, and biodiversity. The limited scientific evidence suggests that air pollutants may most affect biodiversity, in particular because of nutrient loading through nitrogen deposition.

Wetlands are broadly characterized as transitional areas between terrestrial and aquatic systems in which the water table is at or near the surface or the land is periodically covered by shallow water.[38] Types of wetlands include swamps (forested wetlands), marshes (herbaceous vegetation), and peatlands, which are wetlands that accumulate partially decayed vegetative matter due to limited decomposition.[39] Peatlands include bogs and fens. Bogs receive water solely from precipitation, are generally dominated by *Sphagnum* moss, and are low in nutrients. Fens receive water from groundwater and precipitation, contain more marsh-like vegetation, and have higher pH and nutrient levels than bogs.[40] Most of the limited work on the effects of atmospheric deposition on wetlands has been done in peatlands, specifically in Europe, where levels of atmospheric deposition are generally much higher than in the U.S.

The air pollutants of greatest concern with respect to effects on wetland ecosystems are oxides of nitrogen (NO_x) and oxides of sulfur (SO_x), primarily sulfur dioxide (SO_2). Air pollutants may affect wetland ecosystems by acidification of vulnerable wetlands and by increasing nutrient levels. Acidification in vulnerable wetlands may affect vegetation adversely, as appears to have occurred in Europe. In wetlands where nitrogen levels are low, increased nitrogen deposition may alter the dynamics of competition between plant species. Species adapted to low-nitrogen levels, including many endangered species, may decrease in abundance.[41]

Effects of Acidification

Limited evidence suggests that acidic deposition and decreased pH may harm certain wetland plants, alter competitive relations between wetland plants and cause changes in wetland drainage and water retention.

Work concerning the possible acidification of peatlands is inconclusive. Acidic deposition is unlikely to result in displacement of base cations from cation exchange sites in bogs, and therefore it will not cause a drop in pH.[42] Peatland sediments are low in Al^{3+}, so mobilization of toxic aluminum is not a concern as it is in forest soils and aquatic ecosystems.[43] Acidification might affect certain fen ecosystems. Gorham et al. (1984) have hypothesized that acidic deposition could leach base cations from mineral-poor fens and decrease pH levels. This could result in a transition to bog vegetation such as *Sphagnum* and away from sedge meadow vegetation. At this time, this remains a hypothesis; however, pH did not decrease in a mineral-poor Ontario fen during a four-year period in which researchers experimentally increased acidic deposition.[44]

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[37] U.S. EPA, 1994.

[38] Cowardin et al., 1979.

[39] Mitsch and Gosselink, 1986.

[40] Mitsch and Gosselink, 1986.

[41] U.S. EPA, 1993.

[42] Gorham et al., 1984.

[43] Turner et al., NAPAP SOS/T 10, 1990.

In European wetlands affected by high levels of deposition for many years, acidic deposition has seriously affected wetland vegetation. Roelofs (1986) reports that acidification of heath pools in the Netherlands has caused a change in species composition with *Sphagnum* and rushes replacing the original vegetation. Likewise, significant declines in *Sphagnum* in British bogs have occurred in areas affected by 200 years of atmospheric pollution, including nitrogen deposition.[45] It is unclear how such changes have affected wetland service flows apart from the effects on biodiversity; however, water retention has decreased and significant erosion has occurred in seriously perturbed British bogs near Manchester and Liverpool.[46]

Effects of Nutrient Loading

Atmospheric deposition may affect wetlands by increasing the level of nutrients, particularly nitrogen, in wetlands. Sulfur is not a limiting nutrient in peatlands,[47] but nitrogen commonly limits plant growth.[48] The effects of increased nitrogen levels in wetlands include an increased threat to endangered plant species and possible large-scale changes in plant populations and community structure. Endangered and threatened plant species are common in wetlands, with wetland species representing 17 percent of the endangered plant species in the U.S. (U.S. EPA, 1993). These plants are often specifically adapted to low nitrogen levels; examples include isoetids[49] and insectivorous plants.[50] In eastern Canadian wetlands, nationally rare species are most common in infertile sites.[51] When nitrogen levels increase, other species adapted to higher levels of nitrogen may competitively displace these species. Thus, NO_x emissions that increase nitrogen levels in nitrogen-poor wetlands may increase the danger of extinction for threatened and endangered species.

By changing competitive relations between plant species, increased nitrogen deposition may broadly affect community structure in certain wetlands. Common species that thrive in nitrogen-poor wetlands may become less abundant. Many nitrogen-poor bogs in the northern U.S. are dominated by *Sphagnum* species. These species capture low levels of nitrogen from precipitation. Increased nitrogen levels may directly harm *Sphagnum* and cause increased nitrogen to be available to vascular plants that may out compete *Sphagnum.*[52] Studies in Great Britain have documented large declines in *Sphagnum* moss because of atmospheric pollution;[53] nitrogen loading may play an important role in these declines. However, Rochefort et al. (1990) document limited effects of fertilization from experimentally-increased NO_3^- and SO_4^{2-} deposition on an Ontario mineral-poor fen over a four-year period, apart from initially increased *Sphagnum* growth. Thus, increased nitrogen loading might adversely or beneficially affect wetland plants depending on baseline nitrogen concentrations in the wetland, atmospheric nitrogen loading, and species requirements for and sensitivity to nitrogen.

Increases in nitrogen levels due to NO_x emissions will have the greatest effect on wetlands that are extremely nitrogen-limited and that receive small amounts of nitrogen naturally. Since bogs, including *Sphagnum* bogs, receive little surface water runoff, they get most of their nutrient and water loadings through precipitation. These bogs may receive a total of approximately 10 kg nitrogen per hectare per year (kg N/ha/yr), which is one to two orders of magnitude less nitrogen than other freshwater wetlands and saltmarshes receive.[54] As atmospheric deposition of nitrogen has been estimated to be at least 5.5 to 11.7 kg N/ha/yr,[55] changes in NO_x emissions would most likely affect these bogs. The results of a model by Logofet and Alexandrov (1984) suggest that a treeless, nutrient-poor bog may undergo succession to a forested bog because of the input of greater than 7 kg N/ha/yr.

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[44] Rochefort et al., 1990.

[45] Lee et al., 1986.

[46] Lee et al., 1986.

[47] Turner et al., NAPAP SOS/T 10, 1990.

[48] U.S. EPA, 1993.

[49] Boston, 1986.

[50] Moore et al., 1989.

[51] Moore et al., 1989; Wisheu and Keddy, 1989.

[52] Lee & Woodin 1988, Aerts et al., 1992.

[53] Ferguson et al., 1984; Lee et al., 1986.

As in freshwater wetlands, significantly increased nitrogen deposition to coastal wetlands will increase productivity and alter the competitive relationships between species.[56] However, studies showing this increased productivity have used 100 to 3000 kg N/ha/yr.[57] Therefore, limited changes in NO_x emissions may not affect coastal wetland productivity.

Summary of Wetland Ecosystem Effects

The effects of air pollutants on wetlands have received little attention, in contrast to the large body of work on the effects of acid rain on aquatic and forest ecosystems. Little evidence exists suggesting that acidification due to atmospheric deposition is a major threat to wetlands. In particular, peatlands are naturally acidic, although mineral-poor fens may be at risk from acidification. Nitrogen loading may alter community composition in wetlands naturally low in nutrients, such as bogs. Nitrogen loading may threaten rare species adapted to low nitrogen levels. In Britain and The Netherlands, heavy atmospheric deposition over a long period appears to have caused serious declines in *Sphagnum* in peatlands.

Air pollutants appear to most seriously threaten rare and endangered species, biodiversity, and community composition in wetlands, particularly bogs. These changes are difficult to associate with changes in economic value; even the qualitative nature of the effects is uncertain. Air pollutants may not significantly affect such important wetland service flows as flood control, water quality protection, and wildlife habitat in most wetlands, so the impacts on the more readily monetized aspects of the economic value of wetlands may be limited.

Benefits from Avoided Damages to Forests

Introduction

Forests occupy 33 percent of the land mass in the U.S. (some 738 million acres) and provide a wealth of services to the U.S. population.[58] Notable services provided by forests include timber production, recreational opportunities such as hunting, camping, hiking, and wildlife observation, water quality protection, nutrient removal and cycling, flood control, erosion control, temporary carbon sequestration, preservation of diversity, and existence values. In 1991, hunting participation alone accounted for 236 million recreation days that included 214 million person trips with estimated expenditures valued at $12.3 billion.[59]

The Clean Air Act-regulated pollutants of greatest concern with respect to effects on forest ecosystems are oxides of sulfur (SO_x), primarily sulfur dioxide (SO_2), oxides of nitrogen (NO_x), and volatile organic compounds (VOCs). While extremely high ambient concentrations of SO_2 and NO_x may directly affect vegetation, such effects are uncommon in the U.S.;[60] the indirect effects of these pollutants are of greater concern. Specifically, emissions of SO_2 and NO_x are known to contribute to acid deposition in portions of the United States, with SO_2 contributing 75 to 95 percent of the acidity in rainfall in the eastern U.S.[61] Acid deposition is of concern to forests primarily from the acidification of soils (i.e., by reducing seed germination, altering nutrient and heavy metal availability). Direct foliar damage can occur from precipitation with extremely low pH levels (i.e., 3.0-3.6 and below), although these levels are lower than ambient levels in the U.S.[62] VOCs and NO_x are important precursors to ozone formation, which can affect leaf photosynthesis and senescence and decrease cold hardiness, thereby causing deleterious impacts on tree growth, survival and reproduction. Deposition of NO_x may also alter the nutrient balance of forest soils, which in turn might alter the competitive relationships between tree species and affect species composition and diversity.[63]

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[54] U.S. EPA, 1993.

[55] U.S. EPA, 1993.

[56] U.S. EPA, 1993.

[57] U.S. EPA, 1993.

[58] Powell et al. 1993.

[59] U.S. DOI, 1993.

[60] Shriner et al., NAPAP SOS/T 18, 1990.

[61] NAPAP, 1991.

[62] Shriner et al., NAPAP SOS/T 18, 1990.

Current Air Pollutant Effects on Forests

Acid Deposition Impacts

In 1985, NAPAP organized the Forest Response Program (FRP) to evaluate the significance of forest damage caused by acidic deposition, the causal relationships between air pollutants and forest damage, and the dynamics of these relationships regionally. Research was focussed on four forest regions: Eastern Spruce-Fir, Southern Commercial Forests, Eastern Hardwoods, and Western Conifers. With the exception of high-elevation spruce-fir forests, the available evidence suggests that acidic deposition does not currently affect these forests and that observed declines in sugar maple and southern pines are not due to acidic deposition.[64]

Circumstantial evidence suggests that acidic deposition may affect high-elevation spruce-fir forests in the northeastern U.S. These forests have extensive contact with acidic cloud water.[65] Experimental evidence suggests that acidic deposition may affect cold hardiness in red spruce, an important component of the spruce-fir forest. Significant declines in red spruce growth and in its importance in the forest have occurred in New York and northern New England. The proximate cause of death of red spruce in the region is pathogens and insects; acidic deposition may interact with these biological stresses and with weather-induced stress to produce adverse effects in red spruce. Ozone may also play a role in red spruce decline in this region.[66] Available evidence suggests that soil aluminum and soil pH levels have not affected red spruce adversely.[67]

Ozone Impacts

Experimental Evidence

For practical reasons, the majority of experimental evidence linking ozone exposure to damage to tree species has been derived from studies of individual plants, especially seedling and branch studies.[68] Results from these studies suggest that ozone exposure can reduce photosynthesis and increase senescence in leaves. Subsequently, such effects from ozone may alter the carbohydrate allocation to plant tissues such as roots, which may affect plant growth and cold hardiness. Decreases in cold tolerance may be particularly important for trees in northern latitudes and high elevations. Recent work on quantifying the relationship between ozone exposure and plant responses suggest that seedlings of aspen, ponderosa pine, black cherry, tulip poplar, sugar maple, and eastern white pine seedlings may experience biomass reductions of approximately 10 percent at or near ambient ozone exposures.[69] Because trees are perennials, the effect of even a 1-2 percent per year loss in seedling biomass (versus 10 to 20 percent yield loss in crops), if compounded over multiple years under natural field conditions of competition for resources, could be severe.

Although indicative of short-term relative response to ozone exposure, results from these experiments are unable to provide reliable information on the long-term effects of ozone on forests. This limitation arises because the effects of ozone on forests will depend on both the response of individual plants to ozone exposure and the response of populations of plants, which interact with their environment. Population response will be altered by the varying intraspecific genetic susceptibility to ozone. Individual plant response will also be affected by many environmental factors, including insect pests, pathogens, plant symbionts, competing plants, moisture, temperature, light, and other pollutants. Consistent evidence on the interaction of ozone with other environmental factors is lacking. Furthermore, most experimental studies have only studied exposure for one growing season; effects on forest species may occur over decades.[70] Therefore, considerable uncertainties occur in scaling across individuals of different ages, from individuals to populations and communities, and across time.

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[63] U.S. EPA, 1993.

[64] Barnard et al., NAPAP SOS/T 16, 1990; NAPAP, 1991.

[65] Barnard et al., NAPAP SOS/T 16, 1990.

[66] Shriner et al., NAPAP SOS/T 18, 1990.

[67] Barnard et al., NAPAP SOS/T 16, 1990.

[68] U.S. EPA, 1996a.

[69] Hogsett et al. , 1995.

Observational Evidence

Studies of the forests of the San Bernardino Mountains provide the strongest case for linking ozone exposure to damages to an entire forest ecosystem. These forests have been exposed to extremely high ambient ozone levels over the past 50 years due to their proximity to the Los Angeles area. The area has been extensively studied regarding the effects of ozone, as described in U.S. EPA (1996a). The ecosystem has been seriously affected by ozone pollution, with the climax-dominant, but ozone-sensitive ponderosa pine and Jeffrey pine declining in abundance, replaced by more ozone-tolerant species. These sensitive species have experienced decreased growth, survival, and reproduction, and susceptibility to insects. The effects of ozone on these species have resulted in other ecosystem effects, including the buildup of a large litter layer, due to increased needle senescence. The decline of the fire-tolerant ponderosa and Jeffrey pines may seriously affect the fire ecology of the ecosystem, with fire-sensitive species becoming more common. Ozone concentrations have been declining in recent decades, and crown injury of ponderosa and Jeffrey pine has decreased. However, the two species have continued to decline in abundance, as measured by total basal area, compared with other species over the period 1974 to 1988.[71] The nature of community dynamics, particularly in mixed species, uneven aged stands, indicates that subtle long-term forest responses (e.g., shifts in species composition) to elevated levels of a chronic stress like exposure to ozone are more likely than wide-spread community degradation.[72]

Limited field studies have been completed in other forest ecosystems. Foliar injury has been observed in the Jefferson and George Washington National Forests and throughout the Blue Ridge Mountains, including areas of the Shenandoah National Park.[73] In the Great Smoky Mountains National Park, surveys made in the summers from 1987 through 1990 found 95 plant species exhibited foliar injury symptoms consistent with those thought to be caused by ozone.[74] Foliar ozone injury has also been documented in National Parks and Forests in the Sierra Nevada mountains.[75]

Growth and productivity of seedlings have been reported to be affected by ozone for numerous species in the Blue Ridge Mountains of Virginia. In the Shenandoah National Park, Duchelle et al. (1982, 1983) found that tulip poplar, green ash, sweet gum, black locust, as well as several evergreen species (e.g., Eastern hemlock, Table Mountain pine, pitch pine, and Virginia pine), common milkweed, and common blackberry all demonstrated growth suppression of seedlings. Except for the last two species mentioned, almost no visible injury symptoms accompanied the growth reductions. Studies of mature trees in the Appalachian Mountains also indicate that injury associated with exposure to ozone and other oxidants has been occurring for many years.[76] Researchers have also found that major decreases in growth occurred for both symptomatic and asymptomatic trees during the 1950s and 1960s in the Western U.S.[77] The adverse response of a number of fruit and nut trees to ozone exposure has been reported.[78]

Monitoring by the USDA Forest Service shows that growth rates of yellow pine in the Southeast have been decreasing over the past two decades in natural stands but not in pine plantations.[79] Solid evidence linking this growth reduction to air pollutants is lacking, although ozone, in particular, may be a factor.[80] Ambient ozone levels in the region are high enough to damage sensitive tree species, including pine seedlings during experimental exposure.[81] Due to the commercial importance of yellow pine, the economic impacts of ozone on forest ecosystems in this area could be significant if ozone is affecting growth.

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[70] U.S. EPA, 1996a.

[71] Miller et al., 1989 and Miller et al., 1991.

[72] Shaver et al., 1994

[73] Hayes and Skelly, 1977; Skelly et al., 1984

[74] Neufeld, et al., 1992

[75] Peterson and Arbaugh, 1992

[76] Benoit et al., 1982

[77] Peterson et al., 1987; Peterson and Arbaugh, 1988, 1992; Peterson et al., 1991

[78] McCool and Musselman, 1990; Retzlaff et al., 1991, 1992a, b

[79] NAPAP, 1991.

Although the ecosystem effects occurring in the San Bernardino forest ecosystem have occurred at very high ozone exposures, lower ozone exposure elsewhere in the U.S. may still affect forests. The EPA Ozone Staff Paper[82] assessed the risk to vegetation, including forests, under current ambient air quality. Using a GIS approach, it was found that under the base year (1990) air quality, a large portion of California and a few localized areas in North Carolina and Georgia have seasonal ozone levels above those which have been reported to produce greater than 17 percent biomass loss in 50 percent of studied tree seedling species. A broader multistate region in the east is estimated to have air quality sufficient to cause 17 percent biomass loss in seedlings, while at least a third of the country, again mostly in the eastern U.S., most likely has seasonal exposure levels which could allow up to 10 percent yield loss in 50 percent of studied seedlings. The Staff Paper did not present monetized benefits because of lack of exposure-response functions.[83]

Even small changes in the health of ozone-sensitive species may affect competition between sensitive and tolerant species, changing forest stand dynamics.[84] Depending on the sensitivities of individual competing species, this could affect timber production either positively or negatively, and affect community composition and, possibly, ecosystem processes.

Endangered species

Ozone effects may also reduce the ability of affected areas to provide habitats to endangered species. For example, two listed endangered plant species, the spreading aven and Roan Mountain bluet, are currently found at a small number of sites in eastern Tennessee and western North Carolina — forested areas where ozone-related injury is of concern.[85] In addition, ozone-related effects on individual ozone-sensitive species that provide unique support to other species can have broader impacts. For example, one such species is the common milkweed, long known for its sensitivity to ozone and usefulness as an indicator species of elevated ozone levels, as well as being the sole food of the monarch butterfly larvae. Thus, a major risk associated with of the loss of milkweed foliage for a season is that it might have significant indirect effects on the monarch butterfly population. A large number of studies have shown that ozone-sensitive vegetation exists over much of the U.S., with many native species located in forests and Class I areas, which are federally mandated to preserve certain air quality related values.

Valuation of Benefits From CAA-Avoided Damages to Forests

Background

To quantitatively assess the economic benefits of avoided damages of relevant CAA pollutants to forests, it is necessary to link estimated changes in air pollution to measures of forest health and conditions that can be readily quantified in economic terms. For commercial timber production, this would require quantifying the relationship between atmospheric deposition and measures of forest productivity such as timber yield. For assessing recreational benefits, linkages would have to be drawn between air pollution and vulnerable factors that influence forest-based recreation (e.g., site-characteristics such as canopy density, type of tree species, degree of visible tree damage, etc.). While important strides have been made in establishing these linkages (e.g., NAPAP modeling of air pollution effects on forest soil chemistry and tree branch physiology), critical gaps in our ability to predict whole tree and forest responses to air pollution changes have precluded the establishment of such quantitative linkages.[86] Critical knowledge gaps exist in our ability to extrapolate experimental results from seedling and branch studies to whole tree and forest responses, to account for key growth processes of mature trees, to integrate various mechanisms by which air pollution can affect trees (e.g., soil acidification, nitrification, and direct foliar damage, winter stress, etc.), and to account for the interaction of other stressors on forest health and dynamics (susceptibility to insect damage, drought, disease, fire, nutrient and light competition, etc.).

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[80] NAPAP, 1991.

[81] NAPAP, 1991.

[82] U.S. EPA, 1996b

[83] U.S. EPA, 1996b.

[84] U.S. EPA, 1996a.

[85] U.S. EPA, 1996b

[86] NAPAP, 1991.

Despite these constraints to quantifying economic benefits from air pollution reductions on forest ecosystems, relevant studies that have attempted to value air pollution damages on forests are reviewed and summarized below. In some cases, the relationship between air pollution and forest response is estimated using expert judgement (e.g., for NAPAP assessment from various growth scenarios). In other cases, damage estimates reflect current impacts of air pollution on forests, and the dose-response relationship is absent. In the aggregate, this summary provides some insight into possible CAA-related benefits from avoided damages to a select and narrowly focussed group of forest services, but, because of severe data constraints, does not provide an estimate of the overall range of forest-based benefits possible under the CAA.

Commercial Timber Harvesting

The economic impact of hypothetical growth reductions in northeastern and southeastern trees (both hardwood and softwood species) was intensively studied under NAPAP.[87] Growth reductions ranging from 5 to 10 percent over a 5 to 10 year period, depending on the species and location, were assumed to occur as a result of all forms of air pollution based on expert opinion derived from a survey by deSteigner and Pye (1988). Timber market responses to these hypothesized growth declines were modeled until the year 2040 using a revised version of the Timber Assessment Market Model (TAMM90) and the Aggregate Timberland Assessment System (ATLAS), which was used to simulate timber inventories on private timberland in the United States. Economic welfare outputs included changes in consumer and producer surplus and changes in revenue to southeast stumpage owners. Results indicate that annualized reductions in consumer and producer surplus would total $0.5 billion by the year 2000 and $3 billion by the year 2040 (in 1967 dollars). Simulated effects on stumpage owners' revenues were minimal ($10 to $20 million).

In an attempt to estimate the net economic damages from ozone effects on selected U.S. forests, NAPAP studied the effect of various *assumed* reductions in growth rates of commercial southeastern pine forests (both natural and planted).[88] For both planted and natural plus planted pines, the following changes in growth rates were assumed to occur: a two percent increase, no change, a two percent decrease, a five percent decrease, and a ten percent decrease. The two to five percent growth reductions were considered as possible outcomes from current ozone induced damage to southeastern forests, although no quantitative linkage between ozone exposure and damages was established. The ten percent growth reduction scenario was primarily included for evaluating model sensitivity to severe changes in growth and was considered out of the range of likely ozone damage estimates. The TAMM and ATLAS models were again used to simulate timber market responses under baseline and hypothesized growth change scenarios from 1985 to 2040. Results indicate that *annual* changes in total economic surplus (i.e., the sum of consumer and producer surplus and timber owner revenues in 1989 dollars) would range from an increase of $40 million (for the two percent increase in growth scenario) to a decrease of $110 million (for the ten percent decrease in growth scenario) for planted and natural pine model simulations.

In the context of estimated benefits from avoidance of other damages in the absence of the Clean Air Act from 1970 to 1990,[89] the magnitude of economic damages estimated to the commercial timber industry are comparatively small. For example, economic damage estimates range up to $3 billion annually for five to ten percent growth rate reductions in northeast and southeast forests, and just $110 million for southeastern pines. However, in the context of damages to forest-based services as a whole, the NAPAP-derived commercial timber damage estimates should be viewed as representing a lower bound estimate for a variety of reasons. First, these damage estimates exclude other categories of possible forest-based benefits, including recreational and non-use values. Second, even within the context of timber-related damages, the NAPAP forest-damage studies focused on a portion of U.S. forests (northeastern and southeastern U.S.); a much greater geographic range of forests could become susceptible to timber-related damages in the absence of CAA controls. Finally, the NAPAP damage estimates consider only two types of tree species: planted and naturally grown pines, although these species are economically important. Damages to other commercially harvested tree species, such as mixed pine and hardwood forests, are therefore excluded.

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[87] Haynes and Kaiser, NAPAP SOS/T 27 Section B, 1990.

[88] NAPAP, 1991.

[89] Most notably avoided human health effects, which are estimated on the order of $300 to $800 billion annually.

Non-marketed Forest Services

In an effort to address the potential benefits resulting from avoidance of acid deposition-induced damages to non-marketed forest-based services (e.g., recreation use, existence value), an extensive review of the economic literature was conducted under the auspices of NAPAP.[90] From their review, NAPAP could not identify any single study or model that could be reliably used to quantify economic benefits from avoided acid deposition-caused damages to non-marketed forest services (such as recreational use) on a regional or national basis. The primary limitation in many of the studies reviewed was the absence of a quantitative linkage between the value of a recreational user day and important site characteristics which could be tied to air pollution effects. In addition, most studies were narrowly focused geographically to specific sites and did not attempt to value system-wide (larger scale) damages that could result from acid deposition over an entire region. Since the availability of nearby substitution sites will affect the recreational value for a given site, the benefits from such site-specific studies may not reflect actual economic damages incurred from wide-scale air pollution impacts on forests. The inability of studies to consider additional crowding at unaffected sites in addition to changes in recreational participation rates as a function of air pollution damages was also recognized as an important limitation.

Despite not being able to quantitatively assess the benefits from avoided acid deposition-induced damages to nonmarket forest services, several important concepts emerge from NAPAP's review of recreational benefits, that bear relevance to the section 812 retrospective analysis. First, several studies were identified that established a relationship between key forest site characteristics and the value of recreational participation. For example, Brown et al. (1989) used contingent valuation to evaluate the relationship between scenic beauty ratings and willingness of recreationalists to pay at pictured sites. Based on their interviews with over 1400 recreationalists at ten different sites in Arizona, positive correlations were established between scenic beauty rankings determined from one group of recreationalists and willingness to pay to recreate determined by a separate group of recreationalists (r^2 ranged from 0.27 to 0.98 depending on ranking). In another study, Walsh et al. (1989) developed a functional relationship between reduction of recreational benefits and tree density changes that reflected varying levels of insect damage at six campgrounds in the Front Range of the Colorado Rockies. By using both contingent valuation and travel cost models, Walsh et al. (1989) were able to show that 10 percent, 20 percent, and 30 percent decreases in tree densities reduces the total recreational benefits at their sites by 7 percent, 15 percent and 24 percent, respectively. Although results from these studies are limited to the sites from which they were derived, they do support the intuition that the degree of visible damage to forests is to some extent correlated with the magnitude of damages to forest-based recreation expected. This finding supports the notion that the avoidance of damages to forest ecosystems from CAA-induced pollution controls (albeit currently unquantified) have likely benefited forest-based recreation in the U.S.

In addition to establishing relationships between recreational value and visible damage to forest sites, there is evidence linking air pollution (ozone) effects on forests to economic damages to non-use values of forests. For example, D.C. Peterson et al. (1987) valued ozone-induced damages to forests surrounding the Los Angeles area. Using contingent valuation methods, D.C. Peterson et al. (1987) surveyed recreationalists (a random survey of households in the San Bernardino, Los Angles and Orange counties) and residents (a sample of property owners within the San Bernardino and Angeles national forests) for their willingness to pay to prevent forest scenes from degrading one step on a "forest quality ladder" depicting various levels of ozone-induced damages. The mean willingness to pay to protect further degradation was \$37.61 and \$119.48 per household for recreationalists and residents, respectively. Annual damages to Los Angeles area residences from a one-step drop on the forest quality ladder were estimated between \$27 million and \$147 million.

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[90] Rosenthal, NAPAP SOS/T 27 Section B, 1990.

These estimates cannot be directly translated into a rough estimate of the potential non-use values of avoided forest damages. Considering the limited size of the population generating the estimated benefits of forest degradation, however, they do provide evidence that the recreational and non-use benefits may substantially exceed the commercial timber values.

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Appendix F: Effects of Criteria Pollutants on Agriculture

Introduction

One potential impact of air pollutants on economic welfare is their effect on agricultural crops, including annual and perennial species. Pollutants may affect processes within individual plants that affect growth and reproduction, thereby affecting yields of agricultural crops. Possible physiological effects of pollutants include the following: decreased photosynthesis; changes in carbohydrate allocation; increased foliar leaching; decreased nutrient uptake; increased sensitivity to climatic stress, pests, and pathogens; decreased competitive ability; and decreased reproductive efficiency. These physiological effects, in conjunction with environmental factors and intraspecies differences in susceptibility, may affect crop yields.

Primary air pollutants that might damage plants include SO_2, NO_x, and volatile organic compounds (VOCs). These pollutants may have direct effects on crops, or they may damage crops indirectly by contributing to tropospheric (ground-level) ozone, peroxyacetyl nitrate (PAN), and/or acid deposition, all of which damage plants. Tropospheric ozone is formed by photochemical reactions involving VOCs and NO_x, while SO_2 and NO_x cause acidic deposition.

While all of these air pollutants may inflict incremental stresses on crop plants, in most cases air pollutants other than ozone are not a significant danger to crops. Based primarily on EPA's National Acid Precipitation Assessment Program (NAPAP) conclusions,[1] this analysis considers ozone to be the primary pollutant affecting agricultural production.

This analysis estimates the economic value of the difference in agricultural production that has resulted due to the existence of the CAA since 1970. The analysis is restricted to a subset of agricultural commodities, and excludes those commodity crops for which ozone response data are not available. Fruits, vegetables, ornamentals, and specialty crops are also excluded from this analysis. To estimate the economic value of ozone reductions under the CAA, agricultural production levels expected from control scenario ozone conditions are first compared with those expected to be associated with ozone levels predicted under the no-control scenario. Estimated changes in economic welfare are then calculated based on a comparison of estimated economic benefits associated with each level of production.

Ozone Concentration Data

To estimate the nationwide crop damages as a result of ozone exposure, the first step is to estimate the nationwide ozone concentrations under the control and no-control scenarios. This section describes the methodology used to estimate ozone concentrations for each county in each of these two scenarios.

First, historical ozone concentration data at the monitor level were compiled from EPA's AIRS system. Differences between the modeled control and no-control scenario ozone concentrations were then used to scale historical data to derive no-control scenario ozone air quality profiles.[2] Next, the ozone index used in the exposure response evaluation was calculated and applied at the monitor level. For this analysis, the W126 index, a peak-weighted average of cumulative ozone concentrations, was selected to conform with the index currently being used by EPA in ozone NAAQS benefits analysis. The W126 index is one of several cumulative statistics, and may correlate more closely to crop damage than do unweighted indices.[3] EPA has not yet made a final determination of the appropriate index to use in agricultural benefits analysis; thus this analysis should be viewed only as an indicator of the magnitude of potential benefits.

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[1] Shriner et al., 1990; NAPAP, 1991.

[2] Derivation of these ozone air quality profiles for the control and no-control scenario is summarized in the following section and described in detail in Appendix C.

[3] Lefohn et al., 1988.

The third step in ozone concentration estimation involved the use of triangulation and planar interpolation to arrive at a W126 statistic at the county, rather than at the monitor, level. For each county centroid, the closest surrounding triangle of monitors is located and the W126 is calculated for that county using a distance-weighted average of the ozone concentration at each of these monitors.

Control and No-control Scenario Ozone Concentration Data

The initial estimation of ozone concentrations in the control and no-control scenarios was performed by Systems Applications International (SAI). To create the control scenario, SAI compiled ozone data from the EPA's Aerometric Information and Retrieval System (AIRS).[4] SAI summarized these data by fitting gamma distributions to them and providing the alpha and the beta parameters to these distributions. Each of these distributions describes a set of ozone concentration levels, and the distributions are categorized by year, season, and averaging time. SAI defines six distinct "seasons," each composed of a two month period in the year. This analysis uses those distributions which describe 1-hour average ozone concentrations taken from 7 AM to 7 PM and separated into seasons. The analysis utilizes only those monitor records that were modeled in both the control and no-control scenarios.

To determine the ozone concentrations for the no-control scenario, SAI utilized the Ozone Isopleth Plotting with Optional Mechanisms-IV (OZIPM4) model. The input data required for OZIPM4 includes air quality data, surface and upper-air meteorological data, and estimates of anthropogenic and biogenic emissions of volatile organic compounds, NO_x and CO.[5] To create these inputs, SAI used (among other sources) outputs from the Regional Acid Deposition Model (RADM) and the SJVAQS/AUSPEX Regional Modeling Adaptation project (SARMAP). Additional detail concerning the development of ozone concentration data is available in Appendix C and in the SAI report to EPA.[6]

Calculation of the W126 Statistic

Using the SAI ozone concentration distributions, we calculated a sigmoidally weighted ozone index for each monitor. The generalized sigmoidal weighting function used in calculating such indices is presented in Lefohn and Runeckles (1987) as:

where:

$$w_i = 1 / [1 + M \bullet \exp(-A \bullet i)] \qquad (1)$$

w_i = weighting factor for concentration$_i$ (unitless)
c_i = concentration$_i$ (ppm)
M= an arbitrary constant
A = an arbitrary constant

The constants M and A are chosen to give different weights to higher or lower concentrations. The index used in this analysis is the W126 statistic, which is calculated as follows:[7]

$$w_i = 1 / [1 + 4403 \bullet \exp(-126 \bullet c_i)] \qquad (2)$$

and

$$W126 = \sum w_i \qquad (3)$$

Missing values are accounted for by multiplying the resulting W126 statistic by the ratio of the number of potential observations to the number of actual observations (i.e., total hours in period/hours of data in period).

To calculate W126 indices from the monitor level gamma distributions, we used an inverse cumulative density function to calculate a separate representative air concentration for each hour in the two month season. These values are then used in the above equation to obtain a monitor-level W126 statistic.

To ensure that the interpretation of the gamma distributions in this manner does not generate errors, we tested our gamma-derived control-scenario W126s against W126s calculated directly from the AIRS database. We found that insignificant error resulted from the utilization of the gamma distributions to create W126 statistics.

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[4] SAI, ICF Kaiser, 1995.

[5] SAI, ICF Kaiser, 1995.

[6] SAI, ICF Kaiser, 1995.

[7] Lefohn et al., 1988.

Aggregating Ozone Data to the County Level

Because crop production data are available at the county level, the lowest level of aggregation that could be used for ozone indices is also the county level. Therefore, monitor level data needed to be aggregated to a county level. For each county, we first located the monitors from which we would be interpolating data. To identify these monitors, we searched for the three monitors which formed the closest triangle around the centroid of the county.[8] The closest triangle was defined as that triangle in which the sum of the distances from the three monitors to the county centroid was the least. The algorithm stopped searching for closest triangles of monitors when it had searched all monitors within 500 km of a given county centroid (an arbitrary distance, selected to reduce computational requirements).

For coastal counties and some rural counties in some years, monitor triangles around the county centroid do not exist. We assigned the W126 value from the monitor closest to the centroid to these counties. Approximately 15 percent of all county-years (36,973 of 248,880 records) were assigned W126s in this manner.

For the remaining 85 percent, after the closest triangle of monitors was found, a “planar interpolation” was used to calculate the W126 at that county for that year. One way to picture this process is to plot each of the three monitors as a point in space. For each monitor, the x axis represents longitude, the y axis represents latitude and the z axis represents the W126 statistic. A plane can then be drawn through these three points in space. Furthermore, using the equation for the plane, and given the x and y values (latitude and longitude) for the county centroid, the county centroid’s z value (W126 statistic) can be calculated. In essence, this procedure calculates a distance-weighted average of three monitors’ W126 index values and assigns this value to the county centroid.

The result of this data manipulation is a monthly W126 statistic for each county in the continental United States for the years 1971-1990. From these data, yield change estimates were generated, and economic impacts were estimated.

Yield Change Estimates

There are several steps involved in generating yield change estimates. The first is the selection of relevant ozone exposure-response functions (minimum and maximum) for each crop in the analysis. Ozone data, triangulated to the county level, are transformed into an index suitable for use in the selected function(s) to estimate county level predicted yield losses for both the control and no-control scenarios. In the next step, the proportion of each county to the national production of each crop is calculated to permit national aggregation of estimated yield losses. Finally, the control scenario percentage relative yield loss (PRYL) is compared to the minimum and maximum PRYL for the no-control scenario. Each step is discussed in more detail below.

Exposure-Response Functions

To estimate yield impacts from ozone, exposure-response functions are required for each crop to be analyzed. This analysis was restricted to estimating changes in yields for those commodity crops for which consistent exposure-response functions are available and that are included in national agricultural sector models. To maintain consistency with the current ozone NAAQS benefits analysis being conducted by OAQPS, NCLAN-based exposure-response functions using a Weibull functional form and a 12-hour W126 ozone index were used.

Several crops included in the NCLAN research program were not evaluated in this analysis. Non-commodity crops that are not modeled in national agricultural sector models were not included in this analysis: lettuce, tomatoes, potatoes, alfalfa, tobacco, turnips, and kidney beans. In addition, one commodity crop, spring wheat, was excluded because the NCLAN exposure-response function was only developed for winter wheat.

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[8] The vast majority of monitors had latitude and longitude data available through AIRS. 1,528 of 1,536 monitors were located in this manner. For the remaining 8 monitors, if in a given year of monitor data another monitor exists in the county of the unfound monitor, we discarded the unlocated monitor’s data. Otherwise, we located that monitor at the county’s centroid. We located 5 of the remaining 8 monitors in this fashion.

Minimum/Maximum Exposure-Response Functions

Estimated responsiveness of a given crop to ozone varies within the NCLAN data. This range of response is partially explained by the program's evaluation of several cultivars for some crops; ozone sensitivity varies across cultivars. In addition, the conditions for different experiments varied due to variations in location, year, and additional treatments included in some experiments. No one exposure-response function can be assumed to be representative of all cultivars in use, or of all environmental conditions for crop production. To develop a range of benefits estimates that reflects this variation in responsiveness, a minimum responsiveness and a maximum responsiveness function were selected for each crop. In actuality, a number of different cultivars are planted by producers, and so ozone response will be a weighted average of the responsiveness of each cultivar to its ozone condition and its proportion of total acreage. It is important to note that these values do not necessarily bound the analysis, since the number of cultivars evaluated by NCLAN is small relative to the number grown for many crops.

For the crops used in this study, CERL conducted an analysis to identify the ozone concentration required to reduce yields by 10 percent for each crop cultivar using its 12-hour W126 exposure-response function. For each crop, the function demonstrating the lowest ozone concentration at a 10 percent yield loss represents the maximum response, and the function with the highest concentration at 10 percent yield loss represents the minimum response. Table F-1 reports the minimum and maximum exposure-response functions for each crop. Two crops, peanuts and sorghum, did not have multiple NCLAN experiments on which to base a comparison of the responsiveness of different cultivars or the variation in response with different experimental conditions.

Table F-1. Agriculture Exposure-Response Functions.

Crop	Cultivar	Equation Type	Yield Function (PRYL, ppm)	Duration (days)
Barley	CM-72	Both	$1-\exp(-(W126/6998.5)^{1.388}$	95
Corn-Field	PAG 397	Min	$1-\exp(-(W126/94.2)^{4.176}$	83
Corn-Field	Pioneer 3780	Max	$1-\exp(-(W126/92.7)^{2.585}$	83
Cotton	McNair 235	Min	$1-\exp(-(W126/113.3)^{1.397}$	125
Cotton	Acala SJ2	Max	$1-\exp(-(W126/74.6)^{1.066}$	98
Grain Sorghum	DeKalb A28+	Both	$1-\exp(-(W126/205.3)^{1.957}$	85
Peanuts	NC-6	Both	$1-\exp(-(W126/96.8)^{1.890}$	112
Soybeans	Corsoy-79	Min	$1-\exp(-(W126/476.7)^{1.113}$	93
Soybeans	Davis	Max	$1-\exp(-(W126/130.1)^{1.000}$	93
Wheat	ART	Min	$1-\exp(-(W126/76.8)^{2.031}$	54
Wheat	VONA	Max	$1-\exp(-(W126/24.7)^{1.00)}$	61

Source: EPA/CERL (unpublished) for all functions.

Calculation of Ozone Indices

Each NCLAN ozone exposure-response experiment exposed each studied crop over a portion of the crop's growing season. The duration of the NCLAN experiments was provided by CERL and was rounded to the nearest month. The W126 index is cumulative, and so is sensitive both to the duration over which it is calculated and to the specific month(s) within a growing season that are included in it. Because cropping seasons vary across the U.S., the ozone index used to calculate county-level changes in yield due to ozone must reflect the local season for each crop. To determine which portion of the growing season a particular exposure period pertains to (in order to calculate an exposure index), we developed state-specific growing seasons based on planting and harvesting data developed by USDA.[9] The W126 index was calculated using the county level ozone data developed in the prior section, summed for the number of months of NCLAN experimental duration, with the exposure period anchored on the usual harvest month for each crop.[10]

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[9] USDA, 1984. Some states did not have explicit growing seasons reported for certain crops due to the low production in these states. In these cases a proxy state growing season was used. In most of these cases the proxy growing season was taken from a state with an adjoining boundary within the same geographic region.

Calculations of County Weights

Because the benefits analysis did not require a regional level of disaggregation and to minimize computational burdens the economic analysis was conducted at a national level. Ozone data and estimated yield responses, however, were developed at a county level. To conduct a national analysis, the county level yield change estimates were weighted to develop a single national percent relative yield loss for each crop relative to the control scenario, for both the minimum and the maximum yield responses. As a part of calculating a percent change in yield at the national level, weights for each county and crop were created for 1975, 1980, 1985, and 1990. The weights for these four years were used to represent the year itself and the four years immediately following it (e.g., 1975 weights were also used for 1976, 1977, 1978, and 1979). Although weather and other conditions may change the proportion of counties' production to the total national production in each year, five year weights should reflect stable periods of agricultural policy between each Farm Bill, and are sufficient for the level of precision needed for this analysis. The weights were calculated by dividing the production level of a crop in a county[11] by the sum of all states' reported production for that crop.[12] These county weights were applied to the percent relative yield loss results for each county, as discussed below.

Calculation of Percent Change in Yield

Ozone exposure-response functions are expressed in terms of percent relative yield loss (PRYL); the ozone level being analyzed is compared with "clean" (charcoal filtered/zero ozone) air. To calculate a percent change in yield between the control and no-control scenarios, we first calculate a PRYL based on the county-level control scenario W126 ozone index, and a PRYL based on the no-control scenario W126 index. Next, the county weights are applied to the PRYLs. The change in yield, measured relative to the hypothetical zero-ozone crop production, is then:

$$(PRYL_C - PRYL_{NC}) \qquad (4)$$

To obtain the change in terms of our (non-zero) baseline yield, we divide by that yield, and get:

$$(PRYL_C - PRYL_{NC}) \,/\, (100 - PRYL_C) \qquad (5)$$

To create the national percent change in yield for each crop, the results of this equation are summed for each scenario (maximum and minimum) and for each year. Tables F-2 and F-3 present the resulting percent yield changes that were used as inputs to the economic model.

Economic Impact Estimates

To estimate the economic benefits of controls on ozone precursor pollutants under the Clean Air Act, changes in yields due to those controls need to be evaluated in terms of their effect on agricultural markets. To do this, yield changes can be incorporated into an economic model capable of estimating the associated changes in economic surpluses within the agricultural economy, preferably one that reflects changes in producers' production decisions and demand substitution between crops. This type of dynamic analysis is needed because even small changes in yield or price expectations can cause large shifts in the acreage allocated to specific crops, and the degree to which alternative crops will be substituted (particularly for feed uses).

Agricultural Simulation Model (AGSIM)

The modeling approach used in this analysis is an econometric model of the agricultural sector, which estimates demand and supply under different production technologies and policy conditions. The AGricultural SImulation Model (AGSIM) has been

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[10] This analysis required "rounding" some months: if a harvest date was specified to be from the 15th to the end of a month, the W126 index was calculated using that month's data; if the harvest date was specified to be from the first to the 14th of a month, the W126 index was calculated using the prior month's data as the final month in the exposure period.

[11] USDA, 1995.

[12] The national total does not include USDA areas designated "other counties". These areas are groups of counties that for one reason or another (disclosure rules, low amount of production, etc.) are not individually listed. Because we did not have ozone values for these groups, we did not use their production levels in the calculation of the total national production.

Table F-2. Relative No-control to Control Percent Yield Change (harvested acres) for the Minimum Scenario.

	Crop						
Year	**Barley**	**Corn**	**Cotton**	**Peanuts**	**Soybeans**	**Sorghum**	**Winter Wheat**
1975	-0.000020	-0.000171	-0.011936	-0.006635	-0.001166	-0.000717	-0.005631
1976	-0.000013	-0.000329	-0.017505	-0.024048	-0.002171	-0.001841	-0.004841
1977	-0.000013	-0.000169	-0.013114	-0.015150	-0.001562	-0.001118	-0.005464
1978	-0.000019	-0.000291	-0.018692	-0.017606	-0.002480	-0.001844	-0.005894
1979	-0.000027	-0.000100	-0.017217	-0.013067	-0.001898	-0.001389	-0.004998
1980	-0.000019	-0.000200	-0.021315	-0.022761	-0.002397	-0.002222	-0.005385
1981	-0.000016	-0.000071	-0.018552	-0.014269	-0.001951	-0.000802	-0.003964
1982	-0.000020	-0.000070	-0.017295	-0.014200	-0.002107	-0.001050	-0.004773
1983	-0.000023	-0.000617	-0.020842	-0.028601	-0.003901	-0.002366	-0.005904
1984	-0.000027	-0.000111	-0.023552	-0.019225	-0.002919	-0.002881	-0.006121
1985	-0.000025	-0.000132	-0.020947	-0.017965	-0.002645	-0.001726	-0.007316
1986	-0.000029	-0.000158	-0.027968	-0.031605	-0.002899	-0.001564	-0.007597
1987	-0.000033	-0.000358	-0.034584	-0.043854	-0.003776	-0.001812	-0.009669
1988	-0.000027	-0.000662	-0.035069	-0.038085	-0.004563	-0.002922	-0.019873
1989	-0.000024	-0.000150	-0.031245	-0.022094	-0.003769	-0.001359	-0.007605
1990	-0.000024	-0.000210	-0.037988	-0.047395	-0.003819	-0.001567	-0.006449

Note: There is only one scenario for barley, peanuts, and sorghum, because there was only one exposure-response function..

Table F-3. Relative No-control to Control Percent Yield Change (harvested acres) for the Maximum Scenario.

	Crop						
Year	**Barley**	**Corn**	**Cotton**	**Peanuts**	**Soybeans**	**Sorghum**	**Winter Wheat**
1975	-0.000020	-0.001139	-0.021059	-0.006635	-0.005808	-0.000717	-0.034803
1976	-0.000013	-0.002281	-0.032063	-0.024048	-0.010298	-0.001841	-0.040303
1977	-0.000013	-0.001232	-0.025773	-0.015150	-0.007764	-0.001118	-0.049636
1978	-0.000019	-0.002015	-0.033075	-0.017606	-0.011803	-0.001844	-0.050308
1979	-0.000027	-0.001052	-0.031433	-0.013067	-0.009592	-0.001389	-0.052211
1980	-0.000019	-0.001537	-0.037278	-0.022761	-0.011845	-0.002222	-0.054128
1981	-0.000016	-0.000923	-0.035058	-0.014269	-0.009902	-0.000802	-0.053470
1982	-0.000020	-0.000974	-0.034101	-0.014200	-0.010815	-0.001050	-0.058409
1983	-0.000023	-0.003888	-0.040405	-0.028601	-0.018597	-0.002366	-0.063556
1984	-0.000027	-0.001443	-0.043890	-0.019225	-0.014502	-0.002881	-0.067612
1985	-0.000025	-0.001377	-0.040845	-0.017965	-0.013384	-0.001726	-0.072177
1986	-0.000029	-0.001451	-0.052426	-0.031605	-0.014754	-0.001564	-0.081225
1987	-0.000033	-0.002565	-0.061295	-0.043854	-0.018578	-0.001812	-0.089042
1988	-0.000027	-0.004318	-0.061660	-0.038085	-0.021767	-0.002922	-0.120703
1989	-0.000024	-0.001987	-0.059573	-0.022094	-0.018739	-0.001359	-0.086958
1990	-0.000024	-0.002056	-0.071659	-0.047395	-0.018670	-0.001567	-0.082309

Note: There is only one scenario for barley, peanuts, and sorghum, because there was only one exposure-response function.

used extensively to evaluate air pollution impacts, as well as a number of other environmental policy analyses. AGSIM is an econometric-simulation model that is based on a large set of statistically estimated demand and supply equations for agricultural commodities produced in the United States. The model is capable of estimating how farmers will adjust their crop acreages between commodities when relative profitability changes as a result of crop yield and production cost changes. Acreage and yield changes from various scenarios will affect total production of crops, which then affects commodity prices and consumption. The commodity price changes, in turn, affect profitability and cropping patterns in subsequent years. Federal farm program and conservation reserve effects are also incorporated into the model.

The initial version of AGSIM (which went through various acronym revisions) was developed in 1977.[13] The model was developed to permit estimation of aggregate impacts associated with relatively small changes in crop yields or production costs, which might result from various policy conditions such as changes in pesticide input availability, or in this case, changes in crop exposure to ozone. Subsequent revisions to the model as well as the current specification are described in Taylor (1993a).[14] Several policy applications of AGSIM were tested and reported in Taylor (1993b)[15] to provide a comparison to the results of several alternative agricultural sector models. These tests included an expansion of Conservation Reserve acreage, reduced target prices, elimination of agricultural programs in the U.S. other than the Conservation Reserve Program (CRP), and a tax on nitrogenous fertilizer use in the U.S. The model has been used to evaluate the effects of changes to the CRP,[16] changes in agricultural price support programs,[17] and evaluations of policies concerning pesticide availability.[18]

AGSIM is designed to estimate changes in the agricultural sector resulting from policies that affect either the yields or the costs of crop production. Changes in economic variables are computed by comparing a policy simulation of the model with a baseline simulation of the model. For this retrospective evaluation, the baseline reflects actual farm programs, prices, and other parameters since 1970. The model's author, Dr. C. Robert Taylor, modified AGSIM for this analysis to reflect production conditions and policies as they changed through the 20-year span of the Clean Air Act, from 1970 to 1990. During this period, U.S. farm policy parameters changed every five years with the enactment of each Farm Bill, and producer participation varied significantly over the period. Over this time, due to policy, weather, technological development, and other variations, production levels and prices have varied, as have production technologies, costs of production, and relevant cultivars. To reflect these changes, Dr. Taylor re-estimated demand relationships for three periods (1975-79; 1980-84; and 1985-89) based on the farm policies in effect in each period, and structured the model to run on a national level rather than a regional level. The period from 1970-1975 was not modeled because of data limitations and because there was limited impact from the CAA on ozone levels during that period.

The AGSIM baseline production and price data serve as the control scenario baseline. Percent relative yield losses (PRYLs) between the control and no-control scenarios are the relevant input parameter for this analysis, from which AGSIM calculates new yield per planted acre values. Based on these values (as well as on lagged price data, ending stocks from the previous year, and other variables), AGSIM predicts acreage, production, supply, and price parameters for each crop for each year, as well as calculating yield per harvested acre. From these results and the demand relationships embedded in the model, AGSIM calculates the utilization of each crop (i.e., exports, feed use, other domestic use, etc.), as well as the change in consumer surplus, net crop income, deficiency payments and other government support payments. Net surplus is calculated as net crop income plus consumer surplus, less government payments. The first year of results is 1976 because AGSIM must have one year (1975) of lagged data.

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[13] Taylor, C.R., R.D. Lacewell, and H. Talpaz. 1979. Use of Extraneous Information with the Econometric Model to Evaluate Impacts of Pesticide Withdrawals. Western J. of Ag. Econ. 4:1-8.

[14] Taylor, C.R. 1993a. AGSIM: An Econometric-Simulation Model of Regional Crop and National Livestock Production in the United States. In: C.R. Taylor, K.H. Reichelderfer, and S.R. Johnson (Eds) Agricultural Sector Models for the United States: Descriptions and Selected Policy Applications. Ames Iowa: Iowa State University Press.

[15] Taylor, C.R. 1993b. Policy Evaluation Exercises with AGSIM. In: C.R. Taylor, K.H. Reichelderfer, and S.R. Johnson (Eds) Agricultural Sector Models for the United States: Descriptions and Selected Policy Applications. Ames Iowa: Iowa State University Press.

[16] Taylor, C.R. 1990. Supply Control Aspects of the Conservation Reserve. In: T.L. Napier (Ed) Implementing the Conservation Title of the Food Security Act of 1985. Ankeny, Iowa: Soil and Water Conservation Society; Taylor, C.R., H.A. Smith, J.B. Johnson, and T.R. Clark. 1994. Aggregate Economic Effects of CRP Land Returning to Production. J. of Soil and Water Conservation 49:325-328.

[17] Talyor, C.R. 1994. Deterministic vs. Stochastic Evaluation of the Aggregate Effects of Price Support Programs. Agricultural Systems 44:461-474.

[18] Taylor, C.R., G.A. Carlson, F.T. Cooke, K.H. Reichelderfer, and I.R. Starbird. Aggregate Economic Effects of Alternative Boll Weevil Management Strategies. Agricultural Econ. Res. 35:19-19;Taylor, C.R., J.B. Penson Jr., E.G. Smith, and R.D. Knutson. 1991. Impacts of Chemical Use Reduction in the South. S. J. Of Ag. Econ. 23:15-23.

Table F-4 presents the net *changes* in economic surpluses (in 1990 dollars) annually and as a cumulative present value (discounted at 5 percent) over the period 1976-1990 due to the Clean Air Act. The positive surpluses exhibited in almost all years are a result of the increase in yields associated with lower ozone levels than those predicted to occur under the no-control scenario. The present value of the estimated agricultural benefits of the CAA ranges between $7.8 billion in the minimum response case to approximately $37 billion in the maximum response case. This range represents the impacts that would occur if all of the acreage planted to a given crop had an ozone response function similar to either the minimum *available* response function or the maximum *available* response function. The available response functions do not necessarily bracket the true range of potential crop responses, and it is unrealistic to anticipate that all acreage will be planted in cultivars with a uniform response to ozone exposure. These considerations notwithstanding, these values do indicate the likely magnitude of agricultural benefits associated with control of ozone precursors under the CAA, but not the precise value of those benefits. In addition to estimating the present value of net surplus at a discount rate of five percent, two alternative discount rates were used. At a three percent discount rate, the range of net surplus is between $6.7 billion and $32 billion; at seven percent discount rate, the range is between $9 billion and $43 billion. For more detail on AGSIM intermediate model outputs, see Abt Associates (1996).

Table F-4. Change in Farm Program Payments, Net Crop Income, Consumer Surplus, and Net Surplus Due to the CAA (millions 1990 $).

	Change in Farm Program Payments		**Change in Net Crop Income**		**Change in Consumer Surplus**		**Change in Net Surplus**	
Year	**Minimum**	**Maximum**	**Minimum**	**Maximum**	**Minimum**	**Maximum**	**Minimum**	**Maximum**
1976/77	0	0	243	486	236	993	477	1,479
1977/78	0	0	-97	-259	349	1,557	253	1,297
1978/79	43	345	30	298	392	1,646	379	1,597
1979/80	0	0	-140	-406	449	2,000	309	1,594
1980/81	0	0	8	-178	392	2,049	400	1,870
1981/82	112	518	-99	-406	440	2,594	231	1,670
1982/83	168	981	64	107	377	2,730	273	1,856
1983/84	153	1,009	231	958	316	1,969	395	1,917
1984/85	-182	808	82	560	-279	1,686	-14	1,437
1985/86	289	1,291	181	879	616	2,054	509	1,644
1986/87	270	1,356	230	966	462	2,265	422	1,875
1987/88	469	2,033	320	1,405	708	2,990	558	2,361
1988/89	557	2,023	316	1,508	796	2,943	556	2,428
1989/90	329	1,401	161	614	527	2,572	358	1,785
1990/91	414	1,927	180	473	618	3,047	384	1,593
Cumulative Present Value of Net Surplus at 5 percent ($ 1990)							7,763	37,225

Conclusions

Agricultural benefits associated with control of ozone precursors under the Clean Air Act are likely to be fairly large. Because it is possible that over time producers have adopted more ozone-resistant cultivars, it may be appropriate to consider the lower end of the range of predicted benefits to be more indicative of the likely total benefits. The estimates developed in this analysis, however, do not represent all of the likely benefits accruing to agriculture, in that many high-value and/or ozone sensitive crops could not be included in the analysis due to either exposure-response data limitations or agricultural sector modeling limitations. The second consideration implies that benefits will likely be larger than estimated. The minimum case may be the most appropriate starting point, however, due to the first consideration: the current crop mix is probably biased toward lower ozone responsiveness. Therefore, we anticipate that cumulative total agricultural benefits from the Clean Air Act are on the order of ten billion dollars (real terms).

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Appendix G: Lead Benefits Analysis

Introduction

The scientific understanding of the relationship between lead and human health is rapidly expanding. This expansion is documented in numerous EPA studies on the health effects associated with lead exposure. In a pioneering study, Schwartz et al. (U.S. EPA, 1985) quantified a number of health benefits that would result from reductions in the lead content of gasoline. The work was extended by EPA's analysis of lead in drinking water (U.S. EPA, 1986a) and by an EPA-funded study of alternative lead National Ambient Air Quality Standards (U.S. EPA, 1987).

Despite this substantial research, much uncertainty remains. While the health effects of very high levels of blood lead (PbB) are quite severe (including convulsions, coma and death from lead toxicity) and have been known for many years, the effects of lower lead doses continue to be the subject of intensive scientific investigation. Dose-response functions are available for only a handful of health endpoints associated with elevated blood lead levels. Other known or strongly suspected health endpoints cannot be quantified due to a lack of information on the relationship between dose and effect. Table G-1 presents the health effects that are quantified in this analysis, as well as important known health effects that are not quantified.

Some of the health effects that are quantified in this analysis have not been estimated in previous EPA analyses. This is largely due to more recent information about the dose-response functions that makes it possible to expand the health effect coverage beyond what was done previously. Recent information is available for previously unquantified health effects, and new information on previously estimated dose-response functions is also available.

Table G-1. Quantified and Unquantified Health Effects of Lead.

Population Group	Quantified Health Effect	Unquantified Health Effect
Adult Male	*For men in specified age ranges:* Hypertension Non-fatal coronary heart disease Non-fatal Strokes Mortality	Quantified health effects for men in other age ranges Other cardiovascular diseases Neurobehavioral function
Adult Female	*For women in specified age ranges:* Non-fatal coronary heart disease Non-fatal stroke Mortality	Quantified health effects for women in other age ranges Other cardiovascular diseases Reproductive effects Neurobehavioral function
Children	IQ loss effect on lifetime earnings IQ loss effects on special educational needs Neonatal mortality due to low birth weight caused by maternal exposure to lead	Fetal effects from maternal exposure (including diminished IQ) Other neurobehavioral and physiological effects Delinquent and anti-social behavior

Methods Used to Measure and Value Health Effects

The following sections present relevant dose-response relationships for three population groups: children, men, and women. These sections also discuss data sources used for the dose-response relationships, although an extensive review of the literature is not given.[1] In addition, each section includes the methods used to value the changes in health effects determined using these dose-response relationships.

Health Benefits to Children

Changes in IQ

Elevated Pb levels may induce a number of effects on the human nervous system. Generally, these neurobehavioral effects are more serious for children than for adults because of children's rapid rate of development. It is believed that neurobehavioral deficits in children may result from both pre-natal and post-natal exposure. These nervous system effects may include hyperactivity, behavioral and attentional difficulties, delayed mental development, and motor and perceptual skill deficits. Quantification of certain manifestations of these effects is possible because sufficient data exist to estimate a dose-response relationship and IQ loss. The relationship used in the analysis is discussed below.

Quantifying the Relationship Between Blood Lead Levels and IQ

A dose-response relationship for IQ decrements has been estimated by a meta-analysis of seven research studies.[2] Regression coefficients for each study were used to determine a weighted average linear regression coefficient for the relationship between lead and IQ. Each regression coefficient was weighted by the inverse of the variance of each estimate. To determine an overall coefficient, the regression coefficients for studies that used natural logarithms of lead as the exposure index were linearized. In general, the coefficient was linearized in the blood lead range of 10 to 20 µg/dL. However, in one study (Bellinger et al., 1991), 70 percent of the data were below 10 µg/dL; thus, the Bellinger data were linearized in the 5 to 15 µg/dL range. For the studies that did not transform lead concentrations, the regression coefficients were used directly. Given the typical uncertainty within individual studies, the variation in the regression coefficients among studies was not more than would be expected. The relationship determined by Schwartz (1993) suggests that for a 1 µg/dL increase in lead, a decrease of 0.25 IQ points can be expected. The p-value (< 0.0001) indicates that this relationship is highly significant.

To obtain the total change in number of IQ points for a population of children, the 0.25 points lost per µg/dL change in blood lead is multiplied by the average blood lead level for that population. The average blood lead level modeled in this analysis is a geometric mean, not an arithmetic mean. To adjust for this, we use a relationship between the expected value and the geometric mean of a lognormally distributed random variable:

$$E[x] = \exp 1\text{n}\left[(GM) + \frac{(1\text{n}\,(GSD))^2}{2}\right] \qquad (1)$$

where E(X) is the expected value (mean) of the distribution, GM is the geometric mean, and GSD is the geometric standard deviation. Taking the natural logarithm of Equation 1 and rearranging gives the ratio between the expected value and the GM:

$$1\text{n}\,(E(X)) - 1\text{n}\,(GM) = \frac{(1\text{n}\,(GSD))^2}{2} \qquad (2)$$

$$1\text{n}\left[\frac{E(X)}{GM}\right] = \frac{(1\text{n}\,(GSD))^2}{2} \qquad (3)$$

$$\frac{E(X)}{GM} = \exp\left[\frac{(1\text{n}\,(GSD))^2}{2}\right] \qquad (4)$$

For a GSD of 1.6 (the assumed GSD of children's blood lead levels[3]), the resulting ratio between E(X) and GM is 1.117. This ratio is used in equation 5.

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[1] For a detailed review of this literature see U.S. Environmental Protection Agency, (1986b) *Air Quality Criteria Document for Lead*, and 1989 Addendum. Environmental Criteria and Assessment Office, Office of Research and Development, March.

[2] Schwartz, 1993.

[3] Suggested value for sub-populations provided by IEUBK guidance manual (U.S. EPA, 1994).

The total lost IQ points for each group was estimated as:

$$(TOTAL\ LOST\ IQ)_k = \Delta GM_k \times 1.117 \times 0.25 \times (Pop)_k / 7 \quad (5)$$

where $(Pop)_k$ represents the number of children (up to age six) around a given industrial source (in the case of estimating benefits from reduced industrial emissions) or the total U.S. population of children (in the case of estimating benefits from reductions in gasoline lead emissions).

As shown in equation 5, the population of children up to age six is divided by seven to avoid double counting. If we assume that children are evenly distributed by age, this division applies this equation to only children age 0-1. If we did not divide, this equation would count a child who is age zero in the first year of the analysis and count that same child 6 more times in successive years. Dividing by seven does create some undercounting because in the first year of the analysis children from age 1 to 6 are not accounted for, while presumably they are affected by the lead exposure.

The analysis assumes a permanent loss of IQ based on blood lead levels estimated for children six years and younger. Recent studies[4] provide concrete evidence of long-term effects from childhood lead exposure.

Valuing Changes in Children's Intelligence

Available economic research provides little empirical data for society's willingness to pay (WTP) to avoid a decrease in an infant's IQ. Some research, however, has addressed monetization of *a subset* of the effects of decreased IQ. These effects would represent components of society's WTP to avoid IQ decreases. Employed alone, these monetized effects should underestimate society's WTP. Nevertheless, for the purpose of this analysis, these effects are used to approximate the WTP to avoid IQ decrements.

IQ deficits incurred through lead exposure are assumed to persist throughout the exposed infant's lifetime. Two consequences of this IQ decrement are then considered: the decreased present value of expected lifetime earnings for the infant, and the increased educational resources expended for a infant who becomes mentally handicapped or is in need of compensatory education as a consequence of lead exposure. The value of foregone earnings is addressed in this section.

The reduction in IQ has a direct and indirect effect on earnings. The direct effect is straightforward: lower IQs decrease job attainment and performance. Reduced IQ also results in reduced educational attainment, which, in turn, affects earnings and labor force participation. These effects on earnings are additive since the studies used for this analysis have controlled for these effects separately.[5] If personal decisions about the total amount of education and labor force participation were based entirely on each individual maximizing the expected present value of lifetime income, the magnitude of the indirect effect on income of a small change in educational attainment would be close to zero,[6] and certainly less than the magnitude of the direct effect. However, individuals make educational decisions based on a number of considerations in addition to the effect on the present value of lifetime earnings, such as satisfaction (utility) derived from formal education, non-compensation aspects of alternative career opportunities, the ability to pay educational costs, etc. Such considerations could lead to either a positive or negative marginal return to education. Studies[7] of educational attainment and lifetime earnings have generally identified a positive marginal return to education, suggesting that the educational attainment decision may not be based simply on expected earnings.

This analysis uses two sets of estimates of the effects of IQ on earnings. The first estimate, used by Abt Associates in a previous analysis, is based on several older studies. The second estimate is based on Salkever (1995).

Older Estimate of the Effect of IQ on Earnings: The Direct Effect of IQ on Wage Rate

Henry Aaron, Zvi Griliches, and Paul Taubman have reviewed the literature examining the relationship between IQ and lifetime earnings.[8] They found that the direct effect, (schooling held constant) of IQ on wage rates ranged from 0.2 percent to 0.75 percent per IQ point. Perhaps the best of these studies is Griliches (1977).[9] He reported the direct effect of IQ on wage rates to be slightly more than 0.5 percent per IQ point. Because this is roughly the median estimate of the U.S. EPA review of the literature, this estimate is used.

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[4] For example, Bellinger (1992).

[5] IQ is also correlated with other socio-economic factors which have not been quantified in this analysis.

[6] This is a straightforward result of the "envelope theorem" in economics. In this context, the envelope theorem shows that if individuals select the level of education that maximizes expected income, then the marginal benefit of additional education (i.e., the partial derivative of income with respect to education) will be zero at that optimal education level.

[7] Including Chamberlain and Griliches (1977), Ashenfelter and Ham (1979), and Salkever (1995)

Older Estimate of the Effect of IQ on Earnings: The Indirect Effect of IQ on Earnings

From Needleman et al. (1990) it is possible to estimate the change in years of schooling attained per one IQ point change. The study's regression coefficients for the effect of tooth lead on achieved grade provide an estimate of current grade achieved. However, many of these children were in college at the time and are expected to achieve a higher grade level. Following Schwartz (1990), after adjusting the published results for the fact that a higher percentage of children with low tooth lead were attending college, a 0.59 year difference in expected maximum grade achieved between the high and low exposure groups was estimated. It is assumed that educational attainment relates with blood lead levels in proportion to IQ. The difference in IQ score between the high and low exposure group was 4.5 points (from Needleman et al. (1990)). Dividing 0.59/4.5 = 0.131 suggests that the increase in lead exposure which reduces IQ by one point may also reduce years of schooling by 0.131 years.

Studies that estimate the relationship between educational attainment and wage rates (while controlling for IQ and other factors) are less common. Chamberlain and Griliches (1977) estimate that a one year increase in schooling would increase wages by 6.4 percent. In a longitudinal study of 799 subjects over 8 years, Ashenfelter and Ham (1979) reported that an extra year of education increased the average wage rate over the period by 8.8 percent. We use the average of these two estimates (7.6 percent) to calculate the indirect effect of increased schooling on the present value of lifetime income. Increased wages per IQ point are calculated using: (7.6 percent wage increase/school year) x (0.131 school years/IQ) = 1.0 percent increase in earnings per IQ point.

There is one final indirect effect on earnings. Changes in IQ affect labor force participation. Failure to graduate high school, for example, correlates with participation in the labor force, principally through higher unemployment rates and earlier retirement ages. Lead is also a strong correlate with attention span deficits, which likely reduce labor force participation. The results of Needleman et al. (1990) relating lead to failure to graduate high school can be used to estimate changes in earnings due to labor force participation. Using the odds ratio from Needleman et al., it was estimated that a one IQ point deficit would also result in a 4.5 percent increase in the risk of failing to graduate. Krupnick and Cropper (1989) provide estimates of labor force participation between high school graduates and non-graduates, controlling for age, marital status, children, race, region, and other socioeconomic status factors. Based on their data, average participation in the labor force is reduced by 10.6 percent for persons failing to graduate from high school. Because labor force participation is only one component of lifetime earnings (i.e., earnings = wage rate X years of work), this indirect effect of schooling is additive to the effect on wage rates. Combining this estimate with the Needleman result of 4.5 percent increase in the risk of failing to graduate high school per IQ point, indicates that the mean impact of one IQ point loss is a (10.6 percent x 4.5 percent) = 0.477 percent decrease in expected earnings from reduced labor force participation.

Combining the direct effect of 0.5 percent with the two indirect effects (1.0 percent for less schooling and 0.477 percent for reduced labor force participation) yields a total of 1.97 percent decrease in earnings for every loss of one IQ point.

Newer Estimate of the Effect of IQ on Earnings: Salkever (1995)

One of the most recent studies of the effects of IQ on earnings is Salkever (1995). Such an analysis with more recent data is valuable because the labor market has undergone many changes over the quarter century in which earlier studies have appeared. Like the analysis of the effect of IQ on earnings presented above, Salkever (1995) estimates this as the sum of direct and indirect effects. The *direct* effect is the sum of effects of IQ test scores on employment and earnings for employed persons, holding years of schooling constant. The *indirect* effect works through the effect of IQ test scores on years of schooling attained, and the subsequent effect of years of schooling on the probability of employment, and on earnings for employed persons.

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[8] U.S. EPA, 1984.

[9] Griliches used a structural equations model to estimate the impact of multiple variables on an outcome of interest. This method has conceptual advantages over other empirical estimates used in the literature because it successfully controls for the many confounding variables that can affect earnings.

Salkever (1995) provides updated estimates all of the necessary parameters using the most recent available data set, the National Longitudinal Survey of Youth (NLSY). Three regression equations provide these parameters. The years of schooling regression shows the association between IQ scores and highest grade achieved, holding background variables constant. The employment regression shows the association between IQ test scores, highest grade, and background variables on the probability of receiving earned income. This regression thus provides an estimate of the effect of IQ score on employment, holding schooling constant, and the effect of years of schooling on employment, holding IQ constant. The earnings regression shows the association between IQ test scores, highest grade, and background variables on earnings, for those with earned income.

These regressions provide parameters needed to estimate the total effect of a loss of an IQ point on earnings. The direct effects of IQ on employment and earnings for employed persons, holding schooling constant, come from the employment and earnings regressions. The indirect effect of IQ on employment through schooling is the product of the effect of IQ on years of schooling, from the years of schooling regression, and the effect of highest grade on employment, from the employment regression. The indirect effects of IQ on earnings for employed persons through schooling is the product of the effect of IQ on years of schooling, from the years of schooling regression, and the effect of highest grade on earnings for employed persons, from the earnings regression.

The total estimated effect of a loss of an IQ point on earnings is larger than the previous estimate of 1.97 percent. Based on the Salkever study, the most recent estimate of the effect of an IQ point loss is now a reduction in earnings of 1.93 percent for men and 3.22 percent for women, which is a participation-weighted average of 2.39 percent.

Value of Foregone Earnings

In the next step to monetize intelligence effects, the percent earnings loss estimate must be combined with an estimate of the present value of expected lifetime earnings. Data on earnings for employed persons and employment rates as a function of educational attainment, age, and gender were reported for the U.S. population in 1992 by the Bureau of the Census.[10] Assuming this distribution of earnings for employed persons and labor force participation rates remains constant over time, and further assuming a trend rate of real wage growth (productivity effect), an annual discount factor, and year-to-year survival probabilities, the current Census data on earnings can be used to calculate the mean present value of lifetime earnings of a person born today. This analysis assumed a person received earned income from age 18 to age 64, and assumed a real wage growth rate of one percent and an annual discount rate of five percent. Men tend to earn more than women because of higher wage rates and higher labor force participation. However, for both men and women, expected lifetime earnings increase greatly with education.

While the Census data are most likely the best available basis for projecting lifetime earnings, a number of uncertainties deserve mention. Labor force participation rates of women, the elderly, and other groups will most likely continue to change over the next decades. Real earnings of women will probably continue to rise relative to real earnings of men. Unpredictable fluctuations in the economy's growth rate will probably affect labor force participation rates and real wage growth of all groups. Medical advances will probably raise survival probabilities.

One problem that was addressed was the fact that the current educational distribution for older persons today is an especially poor predictor of educational attainment for those born today, since educational attainment has risen over time. In fact, if one simply projected educational attainment for a person born today using this method, this person would lose years of schooling with age (starting between ages 40 and 50), since average years of schooling declines with age in a one-time snapshot of the current population. To address this issue, the analysis assumes education levels cannot fall as a person ages.

Note that use of earnings is an incomplete measure of an individual's value to society. Those individuals who choose not to participate in the labor force for all of their working years must be accounted for, since the lost value of their productive services may not be accurately measured by wage rates. The largest group are those who remain at home doing housework and child rearing. Also, volunteer work contributes significantly to social welfare and rates of volunteerism tend to increase with educational attainment and income.[11] If the opportunity cost of non-wage compensated work is assumed to be the average wage earned by persons of the same sex, age, and education, the average lifetime earnings estimates would be significantly higher and could be approximated by recalculating the tables using full employment rates for all age/sex/education groups. To be conservative, only the value of lost wages is considered in this analysis.

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[10] U.S. Department of Commerce, 1993

The adjusted value of expected lifetime earnings obtained above is a present value for an individual entering the labor force at age 18 and working until age 64. Given a five percent discount rate, the other assumptions mentioned, and current survival probabilities,[12] the present value of lifetime earnings of a person born today would be $170,169.

Costs of Additional Education

The increase in lifetime earnings from additional education is the gross return to education. The gross return to education, however, does not reflect the cost of the additional education. The cost of the marginal education must be subtracted from the gross return in order to obtain the net increase per IQ point from additional education. There are two components of the cost of marginal education; the direct cost of the education, and the opportunity cost of lost income during the education. An estimate of the educational cost component is obtained from the U.S. Department of Education.[13] The marginal cost of education used in this analysis is assumed to be $5,500 per year. This figure is derived from the Department of Education's reported ($5,532) average per-student annual expenditure (current plus capital expenditures) in public primary and secondary schools in 1989-'90. For comparison, the reported annual cost of college education (tuition, room and board) in 4 year public institutions is $4,975, and $12,284 for private institutions.

The estimated cost of an additional 0.131 years of education per IQ point (from the older estimate of IQ effects) is $721 (i.e., 0.131 x $5,500). Because this marginal cost occurs at the end of formal education, it must be discounted to the time the exposure and damage is modeled to occur (age zero). The average level of educational attainment in the population over age 25 is 12.9 years.[14] Therefore, the marginal educational cost is assumed to occur at age 19, resulting in a discounted present value cost of $285.

The other component of the marginal cost of education is the opportunity cost of lost income while in school. Income loss is frequently cited as a major factor in the decision to terminate education, and must be subtracted from the gross returns to education. An estimate of the loss of income is derived assuming that people in school are employed part time, but people out of school are employed full time. The opportunity cost of lost income is the difference between full-time and part-time earnings. The median annual income of people ages 18-24 employed full-time is $16,501, and $5,576 for part-time employment.[15] The lost income associated with being in school an additional 0.131 years is $1,431, which has a present discounted value at age zero of $566.

Salkever found a smaller effect of IQ on educational attainment (0.1007 years per IQ point, versus 0.131 years), which results in smaller estimated costs. Using the same method and data described above, the estimated present value of educational cost per IQ point is $219, and the income opportunity cost is $435.

Final Estimate of the Effect of IQ on Earnings.

Combining the value of lifetime earnings with the two estimates of percent wage loss per IQ point yields a low estimate of $170,169 x 1.97 percent = $3,000 per lost IQ point, and a higher estimate of $4,064 based on Salkever (1995). Subtracting the education and opportunity costs reduces these values to $2,505 and $3,410 per IQ point, respectively. This analysis uses the midpoint of these two estimates, which is $2,957. Of course, changing the discount rate would change this estimate. With an assumed discount rate of seven percent, the final estimate is only $1,311. With an assumed discount rate of three percent, the final estimate rises to $6,879.

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[11] U.S. Department of Commerce, 1986. Table No. 651, p. 383.

[12] Special education costs for children who do not survive to age 18 are not counted, which results in some underestimation of benefits. However, most child mortality occurs before the age of 7, when the special education begins, so this under-counting is not substantive.

[13] "Digest of Education Statistics". U.S. Dept. of Education, 1993.

[14] "Digest of Education Statistics". U.S. Dept. of Education, 1993.

[15] "Money Income of Households, Families, and Persons in the United States: 1992". U.S. Department of Commerce, 1993.

Children with IQs Less Than 70

Quantifying the Number of Children with IQs Less than 70

In addition to the total IQ point decrements that can be predicted to occur in a population of children having a specified blood lead distribution, increases are also expected to occur in the incidence of children having very low IQ scores as the mean blood lead level for that population increases. IQ scores are normalized to have a mean of 100 and a standard deviation of fifteen. An IQ score of 70, which is two standard deviations below the mean, is generally regarded as the point below which children require special compensatory education tailored to the mentally handicapped.

The relationship presented here for estimating changes in the incidence of IQ < 70 was developed to make use of the most current IQ point decrement function provided by Schwartz (1993). It is assumed that for a baseline set of conditions where a population of children has a blood lead distribution defined by some geometric mean and geometric standard deviation, that population also has a normalized IQ point distribution with a mean of 100 and a standard deviation of 15. For this baseline condition, the proportion of the population expected to have IQ < 70 is determined from the standard normal distribution function:

$$P(IQ<70) = \Phi(z) \qquad (6)$$

where:

P(IQ <70) = Probability of IQ scores less than 70

z = standard normal variate; computed for an IQ score of 70, with mean IQ score of 100 and standard deviation of 15 as:

$$z = \frac{70 - 100}{15} = -2 \qquad (7)$$

Φ(z) = Standard normal distribution function:

$$\frac{1}{\sqrt{2\pi}} \int_{-\infty}^{z} e^{-\frac{u^2}{2}}\, du \qquad (8)$$

The integral in the standard normal distribution function does not have a closed form solution. Therefore, values for Φ(z) are usually obtained readily from software with basic statistical functions or from tables typically provided in statistics texts. The solution for Φ(z) where z = -2 is 0.02275. That is, for the normalized IQ score distribution with mean of 100 and standard deviation of 15, it is expected that approximately 2.3 percent of children will have IQ scores below 70.

To estimate changes in the proportion of children with IQ scores below 70 associated with changes in mean blood lead levels for a population of children, the following two key assumptions are made:

1. The mean IQ score will change as a result of changes in the mean blood lead level as:
   $$\Delta\overline{IQ} = -0.25 \times \Delta\overline{PbB}$$
   where
   $$\Delta\overline{IQ} \text{ and } \Delta\overline{PbB}$$
   are the changes in the mean IQ score and in the mean blood lead levels, respectively, between the no-control and control scenarios. This relationship relies on Schwartz' estimate (1993) of a decrease of 0.25 IQ points for each µg/dL increase in blood lead. Note that the mean blood lead level referred to here is the arithmetic mean (or expected value) for the distribution obtained as described previously from the GM and GSD.

2. The standard deviation for the IQ distribution remains at 15.

Using these assumptions, the change in the proportion of children having IQ <70 can then be determined for a given change in mean blood lead from:

$$\Delta P(IQ<70) = \Phi(z_{No\text{-}control}) - \Phi(z_{control}) = \Phi(z_{No\text{-}control}) - 0.02275 \qquad (9)$$

where,

$$z_{No\text{-}control} = \frac{70 - (100 + 0.25 \times \Delta\overline{PbB})}{15} \qquad (10)$$

For a given change in PbB between the control and no-control scenarios a response in terms of IQ is calculated. The procedure above yields an estimate of the percent of the population with IQs less than 70. This percentile is multiplied by the exposed population of children to estimate the increased incidence of children with low IQs. As in the IQ point loss equation, the results of this function are applied to children age 0-6 and divided by seven to avoid double counting. (See discussion under equation 5).

This procedure quantifies only the change in the number of children who pass below the IQ=70 threshold. Any other changes in children's IQ are quantified using the IQ point loss function described previously. Treating these two endpoints additively does not result in double counting, because the value associated with the IQ point loss function is the change in worker productivity while the value associated with IQs less than 70 is the increased educational costs for the individual, as discussed below.

Valuing the Reduction in Number of Children with IQs less than 70

To value the reduction in the number of children with IQs less than 70, the reduction in education costs were measured - a clear underestimate of the total benefits.[16] Kakalik et al. (1981), using data from a study prepared for the Department of Education's Office of Special Education Programs, estimated that part-time special education costs for children who remained in regular classrooms cost $3,064 extra per child per year in 1978. Adjusting for inflation and real income growth using the GNP price deflator yields an estimate of $6,318 per child in 1990 dollars. For the calculations, this incremental estimate of the cost of part-time special education was used to estimate the cost per year per child needing special education as a result of impacts of lead on mental development. Costs would be incurred from grades one through twelve. Discounting future expenses at a rate of three percent yields an expected present value cost of approximately $52,700 per infant (assuming compensatory education begins at age 7 and continues through age 18). Note that this underestimates the cost, since Kakalik et al. measured the increased cost to educate children attending regular school — not a special education program.

Changes in Neonatal Mortality

Quantifying the relationship between PbB levels and neonatal mortality

U.S. EPA (1990c) cites a number of studies linking fetal exposure to lead (via *in utero* exposure from maternal intake of lead) to several adverse health effects. These effects include decreased gestational age, reduced birth weight, late fetal death, and increases in infant mortality.[17] The Centers for Disease Control (CDC, 1991a) presents a method to estimate changes in infant mortality due to changes in maternal blood lead levels during pregnancy.[18] The analysis links two relationships. The first relationship, between maternal blood lead level and gestational age of the newborn, was estimated by Dietrich et al. (1987). CDC then estimated infant mortality as a function of gestational age, using data from the Linked Birth and Infant Death Record Project from the National Center for Health Statistics. The resulting association is a decreased risk of infant mortality of 10^{-4} (or 0.0001) for each 1 µg/dL decrease in maternal blood lead level during pregnancy. This is the relationship used in the current analysis.

Valuing changes in neonatal mortality

The central estimate of the monetary benefit associated with reducing risks of neonatal mortality is $4.8 million per avoided mortality. This analysis attempts to capture the credible range of uncertainty associated with this estimate by describing the monetary benefit as a distribution of values: a Weibull distribution with a mean value of $4.8 million and a standard deviation of $3.24 million. Appendix I documents the derivation of this distribution and the sources of uncertainty in valuing reduced mortality risks.

Health Benefits to Men

In addition to adversely affecting children's health, lead exposure has also been shown to adversely affect adults. The health effects in adults that are quantified and included in the benefits analysis are all related to the effects of lead on blood pressure.[19] The estimated relationships between these health effects and lead exposure differ between men and women. The quantified health effects include increased incidence of hypertension (estimated for males only), initial coronary heart disease (CHD), strokes (initial cerebrovascular accidents and atherothrombotic brain infarctions), and premature mortality. Other health effects associated with elevated blood pressure, and other adult health effects of lead including neurobehavioral effects, are not included in this analysis. This section describes the quantified health effects for men; the next section describes the health effects for women.

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[16] The largest part of this benefit is the parents' willingness to pay to avoid having their child become mentally handicapped, above and beyond the increased educational costs.

[17] Due to unavailability of suitable data, non-fatal health impacts due to decreased gestational age or reduced birth weight have not been included in this analysis. For example, the benefits from avoided developmental disabilities such as sensory and motor dysfunction associated with decreased gestational age have not been included.

[18] The estimated change in infant mortality due to change in birth weight was not modeled because the data relating prenatal lead exposure to birth weight are not as strong as data relating lead exposure and gestational age.

Hypertension

Quantifying the relationship between PbB levels and hypertension

Elevated blood lead has been linked to elevated blood pressure (BP) in adult males, especially men aged 40-59 years.[20] Further studies have demonstrated a dose-response relationship for hypertension (defined as diastolic blood pressure above 90 mm Hg for this model) in males aged 20-74 years.[21] This relationship is:

$$\Delta Pr(HYP)= \frac{1}{1+e^{2.744\,-\,.793*(1\mathrm{n}\,PbB_1)}} - \frac{1}{1+e^{2.744\,-\,.793*(1\mathrm{n}\,PbB_2)}} \quad (11)$$

where:

ΔPr(HYP) = the change in the probability of hypertension;

PbB_1 = blood lead level in the control scenario; and

PbB_2 = blood lead level in the no-control scenario.

Valuing reductions in hypertension

The best measure of the social costs of hypertension, society's willingness to pay to avoid the condition, cannot be quantified without basic research well beyond the scope of this project. Ideally, the measure would include all the medical costs associated with treating hypertension, the individual's willingness to pay to avoid the worry that hypertension could lead to a stroke or CHD, and the individual's willingness to pay to avoid changes in behavior that may be required to reduce the probability that hypertension leads to a stroke or CHD. Medical costs of hypertension can be divided into four categories: physician charges, medication costs, hospitalization costs and lost work time.

This analysis uses recent research results to quantify two components of this benefit category. Krupnick and Cropper (1989), using data from the National Medical Care Expenditure Survey, have estimated the medical costs of hypertension. These costs include physician care, drugs and hospitalization costs. In addition, hypertensives have more bed disability days and work loss days than others of their age and sex. Krupnick and Cropper estimated the increase in work loss days at 0.8 per year, and these were valued at the mean daily wage rate. Adjusting the above costs to 1990 dollars gives an estimate of the annual cost of each case of hypertension of $681. The estimate is likely to be an underestimate of the true social benefit of avoiding a case of hypertension for several reasons. First, a measure of the value of pain, suffering and stress associated with hypertension is not included. Second, the direct costs (out-of-pocket expenses) of diet and behavior modification (e.g., salt-free diets, etc.) are not valued. These costs are likely to be significant, since modifications are typically severe. Third, the loss of satisfaction associated with the diet and behavior modifications are ignored. Finally, the medication for hypertension may produce side effects including drowsiness, nausea, vomiting, anemia, impotence, cancer, and depression. The benefits of avoiding these side effects are not included in this estimate.

Quantifying the relationship between blood lead and blood pressure

Because blood lead has been identified as a risk factor in a number of cardiovascular illnesses,[22] it is useful to quantify the effect of changes in blood lead levels on changes in blood pressure for reasons other than predicting the probability of hypertension. Based on results of a meta-analysis of several studies, Schwartz (1992a) estimated a relationship between a change in blood pressure associated with a decrease in blood lead from 10 µg/dL to 5 µg/dL.[23] The coefficient reported by Schwartz leads to the following function relating blood pressure to blood lead for men:

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[19] Citing laboratory studies with rodents, U.S. EPA (1990c) also presents evidence of the genotoxicity and/or carcinogenicity of lead compounds. While such animal toxicological evidence suggests that human cancer effects are possible, dose-response relationships are not currently available.

[20] Pirkle et al., 1985.

[21] Schwartz, 1988.

[22] Shurtleff, 1974; McGee and Gordon, 1976; Pooling Project Research Group, 1978.

$$\Delta DBP_{men} = 1.4 \times 1\mathrm{n}\left(\frac{PbB_1}{PbB_2}\right) \qquad (12)$$

where:

ΔDBP_{men} = the change in men's diastolic blood pressure expected from a change in PbB;

PbB_1 = blood lead level in the control scenario (in µg/dL); and

PbB_2 = blood lead level in the no-control scenario (in µg/dL).

This blood lead to blood pressure relationship is used to estimate the incidence of initial coronary heart disease, strokes (atherothrombotic brain infarctions and initial cerebrovascular accidents) and premature mortality in men.

Changes In Coronary Heart Disease

Quantifying the relationship between blood pressure and coronary heart disease

Estimated blood pressure changes can be used to predict the increased probability of the initial occurrence of CHD and stroke.[24] Increased blood pressure would also increase the probability of reoccurrence of CHD and stroke, but these quantified relationships are not available. First-time coronary heart disease events in men can be predicted using an equation with different coefficients for each of three age groups. For men between 40 and 59 years old, information from a 1978 study by the Pooling Project Research Group (PPRG) is used. PPRG (1978) presents a multivariate model (controlling for smoking and serum cholesterol) that relates the probability of coronary heart disease (CHD) to blood pressure. The model used data from five different epidemiological studies. From this study, the equation for the change in 10-year probability of occurrence of CHD is:

$$\Delta Pr(CHD_{40\text{-}59}) = \frac{1}{1 + e^{4.996 - 0.030365 * DBP_1)}} - \frac{1}{1 + e^{4.996 - 0.030365 * DBP_2)}} \qquad (13)$$

where:

$\Delta Pr(CHD_{40\text{-}59})$ = change in 10-year probability of occurrence of CHD event for men between 40-59 years old,

DBP_1 = mean diastolic blood pressure in the control scenario; and

DBP_2 = mean diastolic blood pressure in the no-control scenario.

The relationship between BP and first-time CHD in older men was determined from information presented in Shurtleff (1974). This study also uses data from the Framingham Study (McGee and Gordon, 1976) to estimate univariate relationships between BP and a variety of health effects by sex and for each of the following age ranges: 45-54, 55-64, and 65-74 years. Single composite analyses for ages 45-74 were also performed for each sex. For every equation, t-statistics on the variable blood pressure are significant at the 99th percent confidence interval. For men aged 60 to 64 years old, first-time CHD can be predicted from the following equation:

$$\Delta Pr(CHD_{60\text{-}64}) = \frac{1}{1 + e^{5.19676 - 0.02351 * DBP_1)}} - \frac{1}{1 + e^{5.19676 - 0.02351 * DBP_2)}} \qquad (14)$$

where:

$\Delta Pr(CHD_{60\text{-}64})$ = change in 2 year probability of occurrence of CHD event for men from 60 to 64 years old;

DBP_1 = mean diastolic blood pressure in the control scenario; and

DBP_2 = mean diastolic blood pressure in the no-control scenario.

For men aged 65 to 74 years old, the following equation uses data from Shurtleff (1974) to predict the probability of first-time CHD:

$$\Delta Pr(CHD_{65\text{-}74}) = \frac{1}{1 + e^{4.90723 - 0.02031 * DBP_1)}} - \frac{1}{1 + e^{4.90723 - 0.02031 * DBP_2)}} \qquad (15)$$

where:

$\Delta Pr(CHD_{65\text{-}74})$ = change in 2 year probability of occurrence of CHD event for men from 65 to 74 years old;

DBP_1 = mean diastolic blood pressure in the control scenario; and

DBP_2 = mean diastolic blood pressure in the no-control scenario.

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[23] Schwartz, 1992a.

[24] U.S. EPA, 1987.

The probability changes calculated using the functions above are used to estimate the number of CHD events avoided in a given year due to air quality improvements attributable to the Clean Air Act. The resulting CHD incidence estimates include both fatal and non-fatal events. However, because mortality benefits are independently estimated in this analysis, it is important to capture only the non-fatal CHD events. Shurtleff (1974) reported that two-thirds of all CHD events were non-fatal. This factor was therefore applied to the estimate of avoided CHD events for each age category.

Valuing reductions in CHD events

General methodology

Because of the lack of information on WTP to avoid an initial CHD event, WTP was estimated by estimating the associated cost of illness (COI). This will underestimate WTP, as explained in Appendix I. Full COI consists of the present discounted value of all costs associated with the event, including both direct and indirect costs incurred during the hospital stay, as well as the present discounted values of the streams of medical expenditures (direct costs) and lost earnings (indirect costs) incurred once the individual leaves the hospital.

Wittels et al. (1990) estimate the total medical costs within 5 years of diagnosis of each of several types of CHD events (including acute myocardial infarction, angina pectoris, unstable angina pectoris, sudden death and nonsudden death) examined in the Framingham Study. Costs were estimated by multiplying the probability of a medical test or treatment within five years of the initial CHD event (and associated with that event) by the estimated price of the test or treatment. All prices were in 1986 dollars. (It does not appear that any discounting was used.) The probabilities of tests or treatments were based on events examined in the Framingham Study. The authors estimate a total expected cost over a five year period (in 1986 dollars) of $51,211 for acute myocardial infarction, $24,980 for angina pectoris, and $40,581 for unstable angina pectoris. Converted to 1990 dollars (using the consumer price index for medical care, U.S. Bureau of the Census, 1992), this is $68,337 for acute myocardial infarction, $33,334 for angina pectoris, and $54,152 for unstable angina pectoris. (The figures for sudden death and nonsudden death are not included because the CHD events in this analysis exclude those that resulted in death, to avoid double counting.)

Cropper and Krupnick (1990) suggest, in an unpublished study, that CHD-related lost earnings could be a significant component of total COI, although the value of earnings lost may vary substantially with the age of onset of CHD. They estimate, for example, that an individual whose first heart attack occurs between ages 55 and 65 will have an expected annual earnings loss of $12,388 (in 1990 dollars), and a present discounted value of lost earnings over a five-year period of about $53,600, using a five percent discount rate. This is almost as much as the total medical costs over 5 years estimated by Wittels et al. (1990) for unstable angina pectoris, and substantially more than the corresponding estimate of medical costs for angina pectoris. For an individual whose first heart attack occurs between ages 45 and 54, on the other hand, Cropper and Krupnick estimate annual average lost earnings of $2,143 (in 1990 dollars), and a present discounted value of lost earnings over a five-year period of about $9,300, again using a five percent discount rate. Cropper and Krupnick do not estimate medical costs for exactly the same disease categories as Wittels et al., but their research suggests that whether the five-year COI of a CHD event, including both medical costs and lost earnings, is lower or higher than the average of the three estimates reported by Wittels et al. depends on an individual's age at the onset of CHD. Combining Cropper and Krupnick's five-year lost earnings estimates with their estimates for average annual medical expenditures for ischemic heart disease summed over five years, for example, yields a total COI of about $47,000 for a 50 year old and $72,000 for a 60 year old, compared to the $52,000 average of the three estimates reported by Wittels et al.

In addition to the variability in estimates of medical costs and lost earnings arising from CHD, there is uncertainty regarding the proportion of pollution-related CHD events associated with the various classes of CHD. To characterize this uncertainty it was assumed, in the absence of further information, that all pollution-related CHD events are either acute myocardial infarctions, angina pectoris, or unstable angina pectoris. A distribution of estimates of COI for pollution-related CHD was generated by Monte Carlo methods. On each iteration, a value was randomly selected from each of three continuous uniform distributions. Each value selected was normalized by dividing by the sum of the three values, so that the three normalized values summed to 1.0. The resulting triplet of proportions represents a possible set of proportions of pollution-related CHD events that are acute myocardial infarction, angina pectoris, and unstable angina pectoris. The corresponding dollar value for the iteration is a weighted average of the estimated dollar values for the three types of CHD event (from Wittels et al.), where the weights are the three randomly selected proportions. The central tendency estimate of the COI associated with a case of pollution-related CHD is the mean of this distribution, about $52,000.

This estimate is likely to understate full COI because it does not include lost earnings. It is likely to underestimate total WTP to an even greater extent because it does not include WTP to avoid the pain and suffering associated with the CHD event. It is, however, substantially greater than an estimate based only on the direct and indirect costs incurred during the hospital stay.

The valuation for CHD is additive with the valuation for hypertension despite the fact that the conditions often occur together, because the two values represent different costs associated with the conditions. The valuation for hypertension is based on loss of work days as a result of hypertension and some of the medical costs associated with treating hypertension. The valuation for CHD is based on the willingness to pay to avoid the pain and suffering of the CHD itself. Therefore, these two valuations can be separated and added together.

Changes in Initial Cerebrovascular Accidents and Initial Atherothrombotic Brain Infarctions

Quantifying the relationship between blood pressure and first-time stroke

Two types of health events are categorized as strokes: initial cerebrovascular accidents (CA) and initial atherothrombotic brain infarctions (BI). The risk has been quantified for the male population between 45 and 74 years old.[25] For initial cerebrovascular accidents, the logistic equation is:

$$\Delta Pr(CA_{men}) = \frac{1}{1 + e^{8.58889 - 0.04066 * DBP_1)}} - \frac{1}{1 + e^{8.58889 - 0.04066 * DBP_2)}} \quad (16)$$

where:

$\Delta Pr(CA_{men})$ = change in 2 year probability of cerebrovascular accident in men;

DBP_1 = mean diastolic blood pressure in the control scenario; and

DBP_2 = mean diastolic blood pressure in the no-control scenario.

For initial atherothrombotic brain infarctions, the logistic equation is:

$$\Delta Pr(BI_{men}) = \frac{1}{1 + e^{9.9516 - 0.04840 * DBP_1)}} - \frac{1}{1 + e^{9.9516 - 0.04840 * DBP_2)}} \quad (17)$$

where:

$\Delta Pr(BI_{men})$ = change in 2 year probability of brain infarction in men;

DBP_1 = mean diastolic blood pressure in the control scenario; and

DBP_2 = mean diastolic blood pressure in the no-control scenario.

Similar to CHD events, this analysis estimates only non-fatal strokes (to avoid double-counting with premature mortality). Shurtleff (1974) reported that 70 percent of strokes were non-fatal. This factor was applied to the estimates of both CA and BI.

Valuing reductions in strokes

Taylor et al. (1996) estimate the lifetime cost of stroke, including the present discounted value (in 1990 dollars) of the stream of medical expenditures and the present discounted value of the stream of lost earnings, using a five percent discount rate. Estimates are given for each of three separate categories of stroke, separately for males and females at ages 25, 45, 65, and 85. For all three types of stroke, the indirect costs (lost earnings) substantially exceed the direct costs at the two younger ages, and are about the same as or smaller than direct costs at the older ages.

Both types of stroke considered in this analysis fall within the third category, ischemic stroke, considered by Taylor et al. To derive a dollar value of avoiding an initial ischemic stroke for males, a dollar value for avoiding ischemic stroke among males age 55 was interpolated from the values for males ages 45 and 65; similarly, a dollar value for avoiding ischemic stroke among males age 75 was interpolated from the values for males ages 65 and 85. Of males in the United

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[25] Shurtleff, 1974.

States between the ages of 45 and 74 (the age group for which lead-related stroke is predicted), 41.2 percent are ages 45-54 and the remaining 58.8 percent are ages 55-74. The value of an avoided stroke among males was calculated as the weighted average of the values for males in age group 45-54 and males in age group 55-74 , where the weights are the above percents. The value for age group 45-54 is the average of the values for ages 45 and 55; the value for age group 55-74 is the average of the values for ages 55, 65 and 75. The resulting average value of an avoided stroke among males aged 45-74 is about $200,000.

Changes in Premature Mortality

Quantifying the relationship between blood pressure and premature mortality

Information also exists to predict the increased probability of premature death from all causes as a function of elevated blood pressure. U.S. EPA (1987) used population mean values for serum cholesterol and smoking to reduce results from a 12 year follow-up of men aged 40-54 in the Framingham Study (McGee and Gordon, 1976) to an equation in one explanatory variable:

$$\Delta Pr(MORT_{40\text{-}54}) = \frac{1}{1 + e^{5.3158 - 0.03516 * DBP_1}} - \frac{1}{1 + e^{5.3158 - 0.03516 * DBP_2}} \quad (18)$$

where:

ΔPr(MORT$_{40\text{-}54}$) = the change in 12 year probability of death for men aged 40-54;

DBP$_1$ = mean diastolic blood pressure in the control scenario; and

DBP$_2$ = mean diastolic blood pressure in the no-control scenario.

Information from Shurtleff (1974) can be used to estimate the probability of premature death in men older than 54 years old. This study has a 2 year follow up period, so a 2 year probability is estimated. For men aged 55 to 64 years old, mortality can be predicted by the following equation:

$$\Delta Pr(MORT_{55\text{-}64}) = \frac{1}{1 + e^{4.89528 - 0.01866 * DBP_1}} - \frac{1}{1 + e^{4.89528 - 0.01866 * DBP_2}} \quad (19)$$

where:

ΔPr(MORT$_{55\text{-}64}$)= the change in 2 year probability of death in men aged 55-64;

DBP$_1$ = mean diastolic blood pressure in the control scenario; and

DBP$_2$ = mean diastolic blood pressure in the no-control scenario.

For men aged 65 to 74 years old, premature mortality can be predicted by the following equation:

$$\Delta Pr(MORT_{65\text{-}74}) = \frac{1}{1 + e^{3.05723 - 0.00547 * DBP_1}} - \frac{1}{1 + e^{3.05723 - 0.00547 * DBP_2}} \quad (20)$$

where:

ΔPr(MORT$_{65\text{-}74}$) = the change in 2 year probability of death in men aged 55-64;

DBP$_1$ = mean diastolic blood pressure in the control scenario; and

DBP$_2$ = mean diastolic blood pressure in the no-control scenario.

Valuing reductions in premature mortality

As discussed above, premature mortality is valued at $4.8 million per case (discussed further in Appendix I). Because this valuation is based on the willingness to pay to the risk of death, and the CHD valuation is based on the willingness to pay to avoid the pain and suffering of a CHD event (defined as a CHD event that does not end in death, to avoid double counting), these two endpoints are additive as well.

Health Benefits to Women

Available evidence suggests the possibility of health benefits from reducing women's exposure to lead. Recent expanded analysis of data from the second National Health and Nutrition Examination Survey[26] (NHANES II) by Schwartz (1990) indicates a significant association between blood pressure and blood lead in women. Another study, by Rabinowitz et al. (1987), found a small but demonstrable association between maternal blood lead and pregnancy hypertension and blood pressure at time of delivery.

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[26] The Second National Health and Nutrition Examination Survey (NHANES II) was conducted by the U.S. Department of Health and Human Services from 1976 to 1980 and provides researchers with a comprehensive set of nutritional, demographic and health data for the U.S. population.

The effect of lead exposure on the blood pressure of women, relative to the effect on men, is examined in a review of ten published studies.[27] All of the reviewed studies included data for men, and some included data for women. A concordance procedure was used to combine data from each study to predict the decrease in diastolic BP associated with a decrease from 10 µg/dL to 5 µg/dL PbB. The results suggest that the effect on blood pressure for women of this decrease in blood lead is 60 percent of the effect of the same change observed in men. Thus, for women, Equation can be rewritten as:

$$\Delta DBP_{women} = (0.6 \times 1.4) \times 1\mathrm{n}\left(\frac{PbB_1}{PbB_2}\right) \qquad (21)$$

where:

ΔDBP_{women} = the change in women's diastolic blood pressure expected from a change in PbB;
PbB_1 = blood lead level in the control scenario; and
PbB_2 = blood lead level in the no-control scenario.

Although women are at risk of having lead-induced hypertension, there is not a dose-response function for hypertension in women available at this time. Omitting the hypertension benefits for women creates an underestimate of the total benefits, but the impact on the total benefits estimation will likely be small. Lead raises blood pressure in women less than in men, so the probability of causing hypertension is likely to be less than in men, and the total value of hypertension in men is a small portion of the overall estimated benefits.

Changes in Coronary Heart Disease

Quantifying the relationship between blood pressure and coronary heart disease

Elevated blood pressure in women results in the same effects as for men (the occurrence of CHD, two types of stroke, and premature death). However, the general relationships between BP and these health effects are not identical to the dose-response functions estimated for men. All relationships presented here have been estimated for women aged 45 to 74 years old using information from Shurtleff (1974). First-time CHD in women can be estimated from the following equation:

$$\Delta Pr(CHD_{women}) = \frac{1}{1 + e^{6.9401 - 0.03072*DBP_1)}} - \frac{1}{1 + e^{6.9401 - 0.03072*DBP_2)}} \qquad (22)$$

where:

$\Delta Pr(CHD_{women})$ = change in 2 year probability of occurrence of CHD event for women aged 45-74;
DBP_1 = mean diastolic blood pressure in the control scenario; and
DBP_2 = mean diastolic blood pressure in the no-control scenario.

Again, non-fatal CHD events were estimated by assuming that two-thirds of the estimated events were not fatal (Shurtleff, 1974).

Valuing reductions in CHD events

Values of reducing CHD events for women are assumed to be equal to those calculated for men (above): $52,000 per CHD event.

Changes in Atherothrombotic Brain Infarctions and Initial Cerebrovascular Accidents

Quantifying the relationship between blood pressure and first-time stroke

For initial atherothrombotic brain infarctions in women, the logistic equation is:

$$\Delta Pr(BI_{women}) = \frac{1}{1 + e^{10.6716 - 0.0544*DBP_1)}} - \frac{1}{1 + e^{10.6716 - 0.0544*DBP_2)}} \qquad (23)$$

where:

$\Delta Pr(BI_{women})$ = change in 2 year probability of brain infarction in women aged 45-74;
DBP_1 = mean diastolic blood pressure in the control scenario; and
DBP_2 = mean diastolic blood pressure in the no-control scenario.

The relationship between BP and initial cerebrovascular accidents can be predicted by the following logistic equation:

$$\Delta Pr(CA_{women}) = \frac{1}{1 + e^{9.07737 - 0.04287*DBP_1)}} - \frac{1}{1 + e^{9.07737 - 0.04287*DBP_2)}} \qquad (24)$$

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[27] Schwartz, 1992b.

where:

$\Delta Pr(CA_{women})$ = change in 2 year probability of cerebrovascular accident in women aged 45-74;

DBP_1 = mean diastolic blood pressure in the control scenario; and

DBP_2 = mean diastolic blood pressure in the no-control scenario.

The predicted incidences of avoided BI and CA were multiplied by 70 percent to estimate only non-fatal strokes (Shurtleff, 1974).

Valuing reductions in strokes

The value of avoiding an initial cerebrovascular accident or an initial atherothrombotic brain infarction for women was calculated in the same way as for men (see above). Of women in the United States between the ages of 45 and 74 (the age group for which lead-related stroke was predicted), 38.2 percent are ages 45-54 and the remaining 61.8 percent are ages 55-74. Using these percentages, and the gender- and age-specific values in Taylor et al. (1996) the average value among women ages 45-74 of avoiding either type of stroke was estimated to be about $150,000.

Changes in Premature Mortality

Quantifying the relationship between blood pressure and premature mortality

The risk of premature mortality in women can be estimated by the following equation:

$$\Delta Pr(MORT_{women}) = \frac{1}{1 + e^{5.40374 - 0.01511 * DBP_1)}} - \frac{1}{1 + e^{5.40374 - 0.01511 * DBP_2)}} \quad (25)$$

where:

$\Delta Pr(MORT_{women})$ = the change in 2 year probability of death for women aged 45-74;

DBP_1 = mean diastolic blood pressure in the control scenario; and

DBP_2 = mean diastolic blood pressure in the no-control scenario.

Valuing reductions in premature mortality

The value of reducing premature mortality for women is assumed to be equal to that estimated for all premature mortality, $4.8 million per incident (see Appendix I).

Quantifying Uncertainty

Characterizing Uncertainty Surrounding the Dose-Response Relationships

The dose-response functions described for each health endpoint considered above generally quantify the adverse health effects expected due to increased exposure to lead. For children, these relationships are described directly in terms of changes in blood lead. For adults, effects are estimated in terms of changes in blood pressure (which are related to changes in blood lead levels). As with any estimate, uncertainty is associated with the dose-response relationships.

Consistent with the approach outlined in Appendix D for the non-lead criteria air pollutants, this analysis attempts to capture the uncertainty associated with these relationships. This is accomplished by estimating a distribution associated with each dose-response coefficient using the information reported in the literature. This analysis assumes these distributions to be normal. For each of the coefficients used to relate adverse health effects to lead exposure, Table G-2 summarizes the means and standard deviations of the normal distributions used in this analysis.

Characterizing Uncertainty Surrounding the Valuation Estimates

The procedure for quantifying uncertainty associated with the valuation estimates is similar to that used to characterize the dose-response coefficient estimates. The valuation distributions for health effects considered in the lead analysis are documented in Appendix I.

Table G-2. Uncertainty Analysis: Distributions Associated With Dose-Response Coefficients Used to Estimate Lead Health Effects.

Health Effect	Parameters of Normal distributions describing Dose-Response Coefficients	
	Mean	Standard Deviation
Blood Lead-Blood Pressure Coefficient (Adults)	1.44	0.85
Adult Males		
Mortality (ages 40-54)	0.03516	0.16596
Mortality (ages 55-64)	0.01866	0.00533
Mortality (ages 65-74)	0.00547	0.00667
Chronic Heart Disease (ages 40-59)	0.030365	0.003586
Chronic Heart Disease (ages 60-64)	0.02351	0.028
Chronic Heart Disease (ages 65-74)	0.02031	0.00901
Cerebrovascular Accidents	0.04066	0.00711
Atherothrombotic Brain Infarctions	0.0484	0.00938
Hypertension	0.793	not available
Adult Females		
Mortality (ages 45-74)	0.01511	0.00419
Chronic Heart Disease	0.03072	0.00385
Cerebrovascular Accidents	0.04287	0.00637
Atherothrombotic Brain Infarctions	0.0544	0.00754
Children		
Infant Mortality	0.0001	not available
Lost IQ Points	0.245	0.039
IQ<70 (cases)	relies on Lost IQ Point distribution	

Industrial Processes and Boilers and Electric Utilities

This section describes the methods and data sources used to estimate changes in blood lead levels due to changes in lead emissions from industrial processes and boilers between 1970 and 1990 and from electric utilities between 1975 and 1990. The estimates of the changes in health effects resulting from changes in lead emissions due to the CAA are also presented.

Methods Used to Determine Changes in Lead Emissions from Industrial Processes from 1970 to 1990

This analysis used several sources to determine the changes in facility-specific emissions of lead from industrial processes. To summarize, the analysis extracted 1990 facility-specific lead emissions data from the Toxics Release Inventory (TRI), which provides recent emissions data for over 20,000 U.S. manufacturing facilities. This study then adjusted these data by the relative changes in lead emissions between 1970 and 1990; these relative changes were derived from several data sources described below. This method yielded facility-specific emissions for five year intervals between 1970 and 1990 for both the controlled and uncontrolled scenarios. The five-year values were interpolated to derive annual changes for each year between 1970 and 1990. Specific details on this approach are given below.

TRI Data

The Toxics Release Inventory (TRI) is mandated by the Superfund Amendment Reauthorization Act (SARA) Title III Section 313 and requires that U.S. manufacturing facilities with more than 10 employees file annual reports documenting multimedia environmental releases and off-site transfers for over 300 chemicals. Facilities report both stack and fugitive releases to air. Reported releases are generally estimates rather than precise quantifications. Emissions data can be presented as numerical point estimates, or, if releases are below 1,000 pounds, as an estimated range of emissions.

From the TRI data base, this analysis extracted data from the reporting year 1990 for all facilities reporting emissions of lead to air, as either stack or fugitive emissions. Data were reported as annual emissions (in pounds per year). Where emissions are reported as a range, this analysis used the upper bound of the range to represent the emissions.[28] TRI facilities also report their location by latitude and longitude. In order to later match facilities emitting lead with Census data on surrounding exposed populations, this analysis uses the latitudes and longitudes of lead-emitting facilities.

Derivation of Industrial Process Emissions Differentials 1970-1990

The TRI database is the Agency's single best source of consistently reported release data; however, the database does not include information for most of the years modeled in this analysis. Furthermore, this analysis required estimates of hypothetical emissions in the absence of the CAA. Therefore, estimates were created for the emissions of lead from industrial sources under the CAA, and in the absence of the CAA, for the years 1970, 1975, 1980, 1985, and 1990. The *percent changes*, or differentials, reflected by these estimates were then applied to the 1990 TRI data to obtain facility-level release estimates for the years of interest for the control and no-control scenarios.

The method for creating these differentials captured the two potential causes of the differences between emissions from industrial sources regulated by the CAA and emissions from those same sources in the absence of the CAA. The first cause of the difference in emissions is a change in overall industrial output, resulting from the macroeconomic impact of the CAA. The second element is a change in emissions per unit of output, which results from the adoption of cleaner processes and the application of emissions control technology mandated by the CAA. The methods used to project the effects of these two causes, described below, were designed to be as consistent as possible with other emissions projection methods for other segments of the CAA retrospective analysis.

Data sources

Data for the differentials estimates were taken from the following sources:

- the Jorgenson/Wilcoxen (J/W) model projections, conducted as part of the section 812 analysis. This data source addresses the first cause of changes in emissions: the macroeconomic changes that resulted from the implementation of the 1970 CAA. The J/W model calculated the change in economic output for each of thirty-five industrial sectors, roughly analogous to two-digit standard industrial classification (SIC) codes, that resulted from the CAA's implementation. The specific output used from the J/W model in this analysis was the percentage change in economic output for the various industrial sectors, rather than any absolute measure of economic activity.

- the 1991 OAQPS Trends database. This data base is an emissions projection system that was used to produce the report, "The National Air Pollutant Emission Estimates, 1940-1990." It contains information on economic activity, national level emissions and emission controls, by industrial process, from 1970 through 1990. Three different elements were extracted from the Trends database: the emissions of lead per unit economic output for various industrial processes for the years 1970-1990; annual economic output data for these industrial processes; and the emission calculation formula.

- the National Energy Accounts (NEA), compiled by the Bureau of Economic Analysis. This database records the historical levels of industrial energy consumption, disaggregated by fuel type at the approximately three-digit SIC code level.

The manner in which these data were combined to derive lead emissions estimates is described below.

Estimates of industrial process emissions in the control scenario

Emissions data for industrial processes were estimated for the years 1970, 1975, 1980, 1985, and 1990. For each of these years, this analysis extracted an emission factor and a control efficiency for each lead-emitting industrial process in the Trends database. Emissions factors are expressed as amount of lead emitted per unit of economic activity, and control efficiencies are reported as the percent that emissions are reduced through the application of pollution control technology to the process. The year-specific emission factors and control efficiencies were multiplied by the economic activity data for that year, for that process, as reported in the Trends database, using the following equation found in the Trends report:

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[28] Ranges are infrequently reported and are either reported as 0-500 lbs. or 500-1000 lbs. The infrequency of the incidence of a facility reporting a range and the relatively small quantities of lead released by those facilities means any overestimation of benefits that results from using the upper limit of the range is extremely minor.

$$\begin{aligned}&\textit{Emissions} = (\textit{Economic Activity}) \times \\ &\quad (\textit{Emission Factor}) \times (1 - \textit{Control Efficiency}) \qquad (26)\end{aligned}$$

This calculation yielded the estimated control scenario emissions, by industrial process. Industrial processes were then assigned to an NEA code. Finally, all processes assigned to a given NEA code were summed to give a total emissions estimate for that NEA code.

Estimates of industrial process emissions in the no-control scenario

The results from the J/W model were used to estimate process emissions in the no-control scenario. As stated above, the J/W model provides percent changes in economic outputs by industrial sector. To use these values, lead-emitting industrial processes (in the Trends database) were assigned to a J/W sector. The percent change for that sector from the J/W model was then used to adjust the economic activity data for that process from the Trends database. These adjusted economic output figures were used together with 1970 emission factors and control efficiencies to derive the estimated lead emissions for each industrial process in the no-control scenario. The 1970 emission factors and control efficiencies were used for all years in the analysis (1970, 1975, 1980, 1985 and 1990) in the no-control scenario; this assumes that emissions per unit economic output and control efficiencies would have been constant over time in the absence of the CAA. This is the same approach that was used to project the changes in emissions from industrial processes for other criteria pollutants in other portions of the CAA retrospective analysis. The process-level emissions were then aggregated to the NEA-code level, as in the controlled scenario.

Matching TRI Data to Industrial Process Emissions Differentials

The methods described in the preceding section yielded emissions estimates from industrial processes in the control and no-control scenarios, by NEA code. We used these estimates to derive percent changes in emissions between control and no-control scenarios, by NEA code, for application to the TRI emissions data. However, since TRI data are reported by SIC code, we first mapped” NEA codes to the appropriate SIC codes, and used the percent change for each NEA code to represent the percent change for all SIC codes covered by that NEA code.

It should be noted that the Trends data base covers only the most important sources of lead in air, not all sources; as a result, not all SIC codes reporting lead emissions in TRI correspond to an NEA code for which emission differentials have been estimated. However, we assume that the TRI emissions sources that have a match are the most important sources of lead air emissions. In fact, although only 48 out of 519 legitimate SIC codes reporting lead emissions in TRI have matching differentials, these SIC codes account for over 69 percent of the lead emissions reported in TRI. The remaining 31 percent of the emissions are distributed relatively evenly among the remaining 471 SIC codes, each of which contributes a small amount to total emissions.

For the 31 percent of the emissions without differentials, this analysis has no information regarding the change in the lead emissions over time or between the control and no-control scenarios; therefore, we are unable to predict benefits attributable to the CAA for these emission sources. Although excluding these sources may lead us to underestimate total benefits, we believe these sources are unlikely to contribute significantly to the difference between control and no-control scenarios. The Trends data focus on the point sources of lead emissions of greatest concern to the Project Team and of greatest regulatory activity. If a process within an SIC code does not appear in the Trends, it is unlikely to have had specific CAA controls instituted over the past 20 years. A lack of control efficiencies for smaller sources prevents them from being included.

It should also be noted that the total industrial process emissions of lead estimated in the 1990 Trends report actually exceeds the reported lead emissions in

TRI, despite the fact that TRI covers more SIC codes. This is probably attributable in part to the fact that TRI covers only a subset of the facilities contributing to economic output in an SIC code. TRI reporting rules only require facilities with greater than 10 employees and who use certain amounts of lead in their processes to submit information to TRI, while the Trends report attempted to estimate emissions from all sources contributing to the economic output for the industrial sector, regardless of size. However, the components of the Trends data base used in this analysis (i.e., emissions factors, economic output data) represent typical conditions at average facilities; they do not allow for the representation of the distribution of emissions across particular facilities. In contrast, a major strength of the TRI is its match of emissions data with geographical information. Because the distribution of emissions geographically determines the size of exposed populations, this analysis used the TRI data, rather than Trends data, to characterize lead release quantities, and used the Trends figures only to characterize relative emissions and changes over time, rather than to estimate total quantities.

Because the Trends data are intended only as an estimate of emissions using typical conditions at average facilities, and do not capture the differences in facility-level emissions, the data do not provide sufficient information to make specific quantitative adjustments to the TRI-based benefits estimates to account for the overall higher emissions estimates in Trends. However, since Trends does generally suggest that there are many more sources than are accounted for by TRI, it is possible that our benefits calculations may be underestimated.

Some additional assumptions were necessary when matching the TRI lead release data and the differentials from the Trends data. Ideally, we would like to know whether the facilities present at a given location, as reported in the 1990 TRI, were present and operating in earlier years; whether facilities operating in 1970 have ceased to operate; and whether new facilities would have been constructed in the no-control situation. Unfortunately, data do not exist in an accessible form at this level of detail for the years 1970 through 1990. Therefore, for the purposes of this exercise, we have assumed that the locations and numbers of the 1990 sources are the same as they were in 1970.

Methods Used to Determine Changes in Lead Emissions from Industrial Boilers from 1970 to 1990

Several sources were used to determine the change in lead emissions from industrial boilers. TRI locational data, Trends database national fuel consumption levels and emissions factors, and NEA and SIC codes were used to derive the emissions for the control and no-control scenarios.

TRI Data

The TRI does not appear generally to contain combustion emissions data. In general, the emissions data are from process sources. We reached this conclusion based on two pieces of information:

(1) TRI reporting requirements: TRI has three reporting requirements: (a) the facility must fall in SIC codes 20-39; (b) the facility must employ more than 10 persons; and (c) the facility must manufacture or process more than 25,000 pounds of a TRI chemical, or otherwise use more than 10,000 pounds Firms must submit reports only for the chemical that exceeds the thresholds given in item (c), but they must report all releases of that chemical, including releases from uses that would not qualify alone. If the TRI chemical is part of a blended substance and the quantity of the TRI chemical in the blend exceeds the threshold, it must be reported. For industrial boilers, if the amount of lead in the fuel were to exceed the 10,000 pounds threshold, then the firm would be required to report all emissions of lead from combustion of fuel. There is an exemption, however, for ingredients present in small proportions. If the amount of lead in the oil were less than 0.1 percent (1,000 ppm), then the firm would not be required to report the emissions.

The conclusion from the above information is that most firms burning used oil are probably not reporting lead combustion emissions to TRI because these releases fall outside the TRI reporting requirements. The concentration at which lead is typically found is used oil (100 ppm) (NRDC, 1991) is much less than the minimum concentration required for reporting (1,000 ppm).

(2) Use data from the TRI data base: The hypothesis that firms do not report lead combustion was confirmed by an analysis of the data submitted by the

firms reporting lead use to TRI. On the TRI submission forms, firms must indicate how the chemical is used. Our analysis of category codes submitted by firms reporting lead emissions showed the following four use category reports: as a formulation component; as a reactant; as an article component; and repackaging only. None of these category codes suggest that the source of the reported lead release is combustion. Therefore, we may conclude that all of the lead emissions reported in TRI are process emissions.

Based on these analyses, the Project Team could not use the TRI release data to evaluate releases of lead from industrial combustion. However, the TRI geographical information was used to locate industrial facilities by longitude and latitude in order to combine combustion data with population information. For combustion emissions, the calculations included all TRI reporting facilities, not just those who reported lead emissions. The assignment of combustion emissions to these facilities is described below.

Derivation of Industrial Combustion Emissions 1970-1990

As with industrial process emissions, estimates were created for the emissions of lead from industrial combustion under the CAA, and in the absence of the CAA, for the years 1970, 1975, 1980, 1985, and 1990. These emissions estimates were used, in combination with the TRI data base geographic information, to obtain facility-level release estimates for the years of interest for the control and no-control scenarios. The method for deriving these emissions estimates included both the macroeconomic impact of the CAA and the change in emissions per unit of output that resulted from specific pollution control mandates of the CAA. The same data sources were used to derive combustion differentials as were used to derive process differentials. The particular data elements and the methods by which these data were combined to derive lead emissions estimates from industrial combustion are described below.

Estimates of combustion emissions under the control scenario

The Trends database contains a national aggregate industrial fuel consumption estimate, by fuel type (coal, natural gas, oil). For each fuel type, the fuel consumption estimate was disaggregated by the share of that fuel used by each NEA industrial category, using the NEA data base. It should be noted that the NEA includes data only for the years 1970 through 1985. For 1990, the 1985 figures were used to disaggregate the national-level consumption figure into NEA industrial categories.

The Trends database also contains emissions factors for industrial fuel use, by fuel type, as well as control efficiencies. The lead emissions from industrial combustion for each NEA category was derived by multiplying the fuel-specific combustion estimate for each NEA category by the emission factor and control efficiency for that fuel type. The result was emissions of lead by NEA code and by fuel type. Emissions from all fuel types were then summed by NEA code. By using the NEA data to disaggregate the industrial fuel consumption figures, the analysis assumes that the industrial combustion emissions are the same among all industries covered by a given NEA code, an assumption which may bias the analysis.

Estimates of combustion emissions under the no-control scenario

As in the control scenario, the national aggregate industrial fuel consumption estimate, by fuel type (coal, natural gas, oil), was disaggregated by the share of that fuel used by each NEA industrial category. The fuel use was then adjusted by one of two factors: (1) seven of the NEA codes were specifically modeled by the Industrial Combustion Emissions (ICE) model — for these sectors, the ICE modeled percent changes were used instead of J/W percent changes; or (2) the remaining NEA codes were matched to J/W sectors — the J/W percent changes were then applied to those matched NEA codes. These fuel use estimates were then combined with the 1970 emission factors and control efficiencies for industrial combustion by fuel type from the Trends database to obtain combustion-related lead emissions from industrial boilers in the no-control scenario, by NEA code.

The process-specific data in the Trends database, and the energy use data in the NEA, are much more disaggregated than the J/W sectoral projections. For the purpose of the analysis, it was assumed that all of the specific industrial processes in the Trends database and industrial categories in the NEA data set assigned to a given J/W sector changed at the same rate as the entire J/W sector. For example, if the economic activity in the J/W Sector 20, “Primary Metals,” changed by one percent between the control and no-control scenarios, then the analysis assumed that economic activity in each industrial process assigned to the Primary Metals sector also increased by one percent. This approach assumes that the economic activities of specific industries within a sector are equally affected by the imposition of the CAA. This assumption is consistent with the projection of the change in emissions from industrial processes for the other criteria air pollutants, which were calculated using a similar process.

Matching TRI Data to Industrial Combustion Emissions Data

Because of the structure of the TRI reporting requirements, it does not appear that TRI generally contains releases from combustion sources. Although TRI may incidentally contain lead combustion emissions, TRI would contain data on such releases only if the reporting facility also used more than 10,000 pounds of lead per year for manufacturing or processing. As a result, the combustion releases, estimated using the methods described above, do not have corresponding data in the TRI data base. Therefore, we devised a different method for estimating benefits from changes in combustion releases.

The first step in the method was to divide the estimates of total releases of lead from industrial combustion, by NEA code, by an estimate of the number of facilities in each NEA code. The number of facilities in each NEA category was estimated using the 1987 Census of Manufactures. This Census, conducted by the U.S. Department of Commerce, tallies the number of facilities by four-digit SIC code; these SIC codes were matched to the NEA codes.

Dividing total lead emissions emitted by number of facilities yielded the average yearly lead emissions from industrial combustion for each SIC code. We then assigned this average value to *all reporting TRI facilities* in the SIC code. The consequence of this approach is that the modeling of combustion from industrial facilities includes substantially more sources than the modeling of industrial process emissions; combustion emissions are assigned to essentially all facilities reporting to TRI, while the process emissions are only evaluated for facilities actually reporting lead air emissions from processes.

One unavoidable drawback to this approach is that it cannot capture differences in release quantities among facilities within an SIC code. Furthermore, this approach does not capture all combustion emissions because we assign average emissions only to facilities that report to TRI. TRI facilities account for between two percent and 50 percent of all facilities listed in the Census of Manufacturers, depending on the SIC code. Because of the inability to place the remaining facilities geographically, this analysis excludes the consideration of emissions from non-TRI facilities.

Methods Used to Determine Changes in Lead Emissions from Electric Utilities from 1975 to 1990

The estimation of lead emissions from electric utilities required data from three different sources. Energy use data for the control and no-control scenarios were obtained from the national coal use estimates prepared for the section 812 analysis by ICF Incorporated. The OAQPS Trends Database provided emissions factors and control efficiencies. Individual plant latitudes, longitudes, and stack information were collected from the EPA Interim Emissions Inventory. This analysis combines these three sets of data and estimates annual lead emissions at the plant level for coal burning electric utilities in the control and no-control scenarios. This section describes the sources and the methods used to create the final data set.

Coal-Use Data

The energy use data obtained from national coal-use estimates provide plant level energy consumption information for 822 electric utilities. The data set were separated into four distinct sets for the years 1975, 1980, 1985, and 1990. Each set of data provided the state where the plants are located, the plant names, and the amount of coal consumed, for both the control and no-control scenarios. The four data sets were combined into one comprehensive set by matching the plants' names and states.

The EPA Interim Emissions Inventory

The EPA Office of Air Quality Planning and Standards Technical Support Division provided the 1991 EPA Interim Emissions Inventory. The Interim Inventory contains data for all electric utility and industrial plants in the United States including latitude, longitude, stack height, stack diameter, stack velocity, and stack temperature. The additional stack parameter data allowed the use of plant-specific parameters in the air modeling for electric utilities rather than average parameters for all facilities as was done for industrial emissions.

Matching the Coal-Use Data to the Interim Emissions Inventory

The combination of the Interim Emissions Inventory and the coal-use data required two steps. First, the Interim Emissions Inventory had to be pared down to include only electric utility data, and to narrow the information provided for each utility. Second, the two databases had to be combined. One difficulty in combining them was the lack of a common data field that would allow a quick and complete matching process.

Electric utility plants were identified in the Interim Emissions Inventory by SIC code (code 4911). The associated stack information file, which lists the size of every stack on every plant, was reduced to include only the tallest stack for each plant. This provides a reasonable estimate of the stack height at which most emissions occur. The air modeling assumes that each electric utility releases its emissions from the largest stack that exists at that plant.

Next, the procedure matched the abridged Interim Emissions Inventory file with the coal use data. Due to the lack of a common data field between the two sets, this process required several phases. Both data sets had name fields, but these fields utilized different naming conventions for the plants. Therefore the name fields were matched directly, with individual words in the names, and then with abridged words from the names. Abridged word matches were double checked by ensuring that the names were indeed similar and by verifying that the state fields matched. Finally some matches were made by hand.

Only 27 unmatched plants with positive coal use remained. There were 493 matched plants with positive coal usage and these were included in the final data set.[29] To eliminate under-counting of emissions, the emissions from the 27 unmatched plants were allocated to matched plants within the states where the unmatched plants were located. Allocations were weighted according to the emission level for each matched plant within that state in the year in which the allocation was being made.

Emissions Factors and Control Efficiencies

At this stage, the electric utilities data set contained coal consumption by plant by year in the control and no-control cases as well as air modeling parameters. Using emission factors for lead and control efficiencies for electric utilities, estimates of lead emissions per plant per year could now be calculated. As in the industrial source analysis, the emission factors and control efficiencies come from the 1991 OAQPS Trends database.

Control efficiencies are available for coal-fired electric utilities in each year between 1975 and 1990. As in the industrial source analysis, it is assumed that pollution control on coal-burning power plants without the CAA would be the same as the pollution control level in 1970. Therefore, the control efficiency from 1970 is used in the no-control analysis.

The emission factor obtained from the Trends database is expressed in terms of lead emitted per ton of coal burned (6,050 grams per 1,000 tons).[30] The combined data set, though, contains quantity of coal burned per plant per year in energy units (trillions of BTUs). To reconcile this difference, a conversion factor was obtained from a 1992 DOE report titled *Cost and Quality of Fuels for Electric Utility Plants 1991*. The conversion factor used (20.93 million BTUs per ton of coal) is the average BTU per pound of coal burned for all domestic electric utility plants in 1990. Data for a small subset of other years were also provided in the DOE report, but they did not differ significantly from the 1990 number. Therefore, the 1990 conversion factor (637.3 pounds of lead per trillion BTU) is assumed valid over the entire study period. The final equation for lead emissions looks quite similar to the equation used in the industrial source analysis.[31] The only change is that “Economic Activity” has been replaced by “Coal Consumed” for this particular analysis:

$$\begin{aligned}&\textit{Emissions} = (\textit{Coal Consumed}) \times \\ &\quad (\textit{Emission Factor}) \times (1 - \textit{Control Efficiency}) \qquad (26)\end{aligned}$$

This equation produces estimates of the emissions per plant per year in both the control and the no-control scenarios.

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[29] Plants with zero coal usage were not immediately excluded from the analysis due to the possibility of analyzing lead emissions from oil combustion at these plants. However, OAQPS has suggested that oil combustion comprises under two percent of the total lead emitted from electric utilities. For this reason, the electric utility analysis focused entirely on coal.

[30] The actual figure cited is 12.1 metric pounds per 1,000 tons. A metric pound is one two-thousandth of a metric ton.

[31] U.S. EPA, 1991a

Use of Air Dispersion Modeling to Estimate Ambient Air Lead Levels

To link estimates of lead emissions to blood lead levels of populations living in the vicinity of a facility, the lead benefits model first uses air dispersion modeling to estimate air lead concentrations surrounding facilities that emit lead into the air. The air concentrations are then linked to blood lead levels.

This analysis uses the Industrial Source Complex Long Term (ISCLT) air dispersion model, a steady-state Gaussian plume model, to estimate long-term lead concentrations downwind of a source. The concentration is modeled as a function of site parameters (stack height, stack velocity).[32] The general form of the concentration equation from a point source at a distance *r* greater than one meter away is as follows:[33]

$$C_{air,r_{ijk}} = \frac{2K}{\sqrt{2\pi}\, r\Theta} - \frac{Q\ f\ S\ V\ D}{u\sigma_z} \qquad (28)$$

where,

C_{air} = concentration at distance *r* ($\mu g/m^3$),
Q = pollutant emission rate (g/sec),
f = frequency of occurrence of wind speed and direction,
Θ = sector width (radians),
S = smoothing function used to smooth discontinuities at sector boundaries,
u = mean wind speed (m/sec),
σ_z = standard deviation of vertical concentration distribution (m),
V = vertical term (m),
K = scaling coefficient for unit agreement.

For each facility modeled in the lead benefits model, a 21 by 21 kilometer grid around the source is specified. The model stores data in 1 km by 1 km cells and calculates the air lead concentrations for each of the 441 cells surrounding a given facility. Fugitive sources are modeled similarly, the only difference being a modified form of Equation 28.

For facility-specific weather data, the model used Stability Array (STAR) data. The STAR data contain information on typical wind speed and direction for thousands of weather stations in the U.S. For each facility, the model accesses the STAR data for the weather station nearest the source. Standard default parameters are used for the other parameters because facility-specific data are not available for them (except for utilities). Table G-3 lists default parameters for the ISCLT, and summarizes sources for other parameters.

Industrial process emissions were modeled as either point or fugitive sources, depending on how they were reported in TRI. All industrial combustion emissions were modeled as “fugitive” emissions. This is a more appropriate model scenario for boiler emissions than a 10 meter stack scenario. All electric utility sources were modeled as point sources.

The model tracks all lead emissions to a given grid cell. That is, if the plumes of two or more sources overlap in a given cell, the air concentration in the given cell is determined from the sum of all of the contributing sources.

Determination of Blood Lead Levels from Air Lead Concentrations

Once the air lead concentrations surrounding a given plant are estimated, the model estimates blood lead levels for children and adults living in those areas. This section describes the methods and data sources used to derive blood lead levels from estimated air lead concentrations.

Relationship Between Air Lead Concentrations and Blood Lead Levels

The rates at which lead is absorbed from air depend on the age of the exposed individual, distance from the facility, the initial concentration of blood lead, and other factors. In addition, rates determined from empirical data may differ depending on whether or not the analyses from which rates are derived have controlled for factors such as lead exposure through deposition on dust and soil (i.e., “indirect exposure”). Especially when children constitute the exposed group, the inclusion of indirect exposure results in higher air lead to blood lead slopes. In both cases, the slope re-

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[32] Ideally, reported stack and fugitive air releases would be modeled using site-specific data (such as source area or stack height). However, since TRI does not contain such facility-specific information, default values are used to model TRI facilities.

[33] This equation is from U.S. EPA (1992). The equation is for a specific wind speed, direction, and category (*ijk*). Each facility has several combinations of these that must be added to arrive at a total concentration at that point. The equation for area sources is similar.

Table G-3. Air Modeling Parameters.

Parameter	Industrial Source Value	Electric Utility Value	Source/ Comment
Stack height	10 m	site-specific or 115.0 m*	Industrial – U.S. EPA (1992) Utilities -- U.S. EPA (1991b)
Exit velocity	0.01 m/s	site-specific or 22.5 m/s*	Industrial – U.S. EPA (1992) Utilities -- U.S. EPA (1991b)
Stack diameter	1 m	site-specific or 5.15 m*	Industrial – U.S. EPA (1992) Utilities -- U.S. EPA (1991b)
Exit gas temperature	293° K	site-specific or 427.5*	Industrial – U.S. EPA (1992) Utilities -- U.S. EPA (1991b)
Area source size	10 m^2	10 m^2	U.S. EPA (1992)
Area source height	3 m	3 m	U.S. EPA (1992)
Lead emission rate	site-specific	site-specific	Industrial – TRIS (lbs/yr) Utilities -- SAI & OAQPS (lbs/yr)
Frequency of wind speed and direction	site-specific	site-specific	STAR data
Sector width	22.5°	22.5°	360° divided by 16 wind directions
Wind speed	site-specific	site-specific	STAR data (m/sec)
Smoothing function	calculated	calculated	
Vertical term	calculated	calculated	

* average value for electric utilities, utilized for utilities without this information

lationship is expressed as the change in blood lead (µg/dL) per change in air concentration (µg/m^3).

In performing this analysis, a choice had to be made between the use of air lead:blood lead relationships that account for inhalation exposure ("direct" slopes) and those that account for exposure to lead deposited from air onto soil and dust ("indirect" slopes). The choice of which slopes to use considered both the effects on the estimate of benefits over time (from 1970 to 1990) and the estimate of the difference in benefits between the control and no-control scenarios. The indirect slope is more comprehensive in its coverage of the types of exposures that will result from air releases, and thus captures more of the health effects predicted to occur from lead exposures, especially to children. For this reason, indirect slopes are preferred to direct slopes, especially when comparing the control and no-control scenarios: using only the direct slope would underestimate the benefits of avoiding deposition that controls confer. However, indirect slopes may capture effects from exposure to soil and dust lead deposited from both current air releases and historic air releases. Since lead's dissipation from soil is slow relative to its removal from air, the reservoir of lead in soil and dust is unlikely to change at the same rate as the reductions in air lead concentrations. Therefore, using indirect slopes to represent a change in blood lead over time due to reduced air emissions may overestimate the change in blood lead, and thus overestimate the benefits of reductions over time, to the extent that the indirect slope captures exposure to the total reservoir of soil and dust lead, rather than only recently deposited lead.

Given that the focus of this analysis is the *difference* between the control and no-control scenarios, it is important to capture both the benefits from reduced lead deposition that result from the CAA, and the direct benefits from reduced air concentrations. Therefore, this analysis modeled changes in blood lead levels using indirect slopes. It should be kept in mind that this choice may overestimate blood lead changes over time for both the control and no-control scenarios.

The relationship between concentrations of lead in ambient air and blood lead concentrations has been evaluated by a variety of methods. These include experimental studies of adult volunteers, as well as epidemiological studies of different populations of children and adults. The discussion below describes the slopes used in this analysis for children and adults, and for individuals with blood lead values greater than 30 µg/dL.

Children

U.S. EPA (1986b) reports that slopes which include both direct (inhalation) and indirect (via soil, dust, etc.) air lead contributions vary widely, but typically range from three to five µg/dL increment in children's blood lead per µg/m^3 increment in air lead concentration (roughly double the slope due to inhaled air lead alone). Since hand dust levels can play a significant role in blood lead levels (U.S. EPA, 1986b), this higher slope may be due to mouthing behavior of children that brings them into contact with dust and soil.

Specific values for estimating contribution of air lead to blood lead, including indirect pathways, are cited in U.S. EPA (1986b); slope values (ranging from -2.63 to 31.2) and data sources for these values are presented in Table 11-36 of U.S. EPA (1986b). The median of these values is 4.0 µg/dL per µg/m^3, which matches the midpoint of the range of typical slope values. This analysis used this value to represent the relationship between air lead concentrations and blood lead concentrations for children living in the vicinity of point sources of lead emissions.

The use of this slope assumes that indirect exposure" principally measures indirect effects of lead emissions to air (through deposition to dust and soil). However, it is possible that these slopes include other exposures not related to air lead. In many cases researchers have measured other possible exposures, such as water and food, and have confirmed that the most significant contribution comes from soil and dust lead, which is assumed to result from air deposition of lead. Those studies that measured lead in tap water showed that mean levels were generally low or not significantly related to blood lead. Landrigan et al. (1975) measured lead in pottery and food; lead in pottery was found in only 2.8 percent of homes, and food and water made no more than a negligible contribution to lead uptake. Lead in paint was measured in some studies.[34] Landrigan and Baker (1981) measured lead in paint at levels greater than one percent in about one fourth to one third of the houses in each area studied. Brunekreef et al. (1981) measured high levels of paint in some houses, but excluded these data points from the analysis.

Despite the possibility of confounding factors, this analysis uses the median value determined above (4.0 µg/dL per µg/m^3) as the appropriate slope for children living within five kilometers of the point source. Five kilometers is chosen as the cut off point because the data from most of the studies cited collected the majority of their data points near lead smelters.[35] Furthermore, these slopes, although measured primarily in the vicinity of smelters, are assumed applicable to all point sources that emit lead into the air.

Adults

For adult males and females, the air lead/ blood lead slopes that include indirect effects due to soil and dust differ very little from slopes that include only direct effects. This result is expected since the higher indirect slope values estimated for children are assumed to be as a result of mouthing behavior typical of young children.

U.S. EPA (1986b) describes several population studies that estimate indirect slopes for men; these slopes range from -0.1 to 3.1 µg/dL per µg/m^3.[36] Snee (1981) determined a weighted average of these studies and one other study.[37] The average slope, weighted by the inverse of each study's variance, is 1.0 µg/dL per µg/m^3. However, the Azar study measured the direct relationship between air lead and blood lead. Excluding the Azar study from the weighted average, the average slope is 1.1 µg/m^3. Excluding the highest and lowest slopes from this group (from Goldsmith,

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[34] Landrigan and Baker, 1981; Brunekreef et al., 1981.

[35] U.S. EPA, 1986b, Table 11-36.

[36] Johnson et al., 1976; Nordman, 1975; Goldsmith, 1974; Tsuchiya et al., 1975; Fugas et al., 1973.

[37] Azar et al., 1975.

1974 and Tsuchiya et al., 1975), both of which had difficulties,[38] the resulting slope is 1.4 µg/dL per µg/m^3.

Slopes for females range from 0.6 to 2.4 for general atmospheric conditions.[39] Snee determined an average slope for women of 0.9 µg/dL per µg/m^3, weighted by the inverse of the variances of the studies. Excluding the slope for women from Goldsmith (1974), the resulting slope for women is 1.0 µg/dL per µg/m^3.

These values are adjusted by a factor of 1.3 to account for the resorption of lead from bone tissue (according to Chamberlain, 1983), thus deriving an adjusted slope estimate of 1.8 µg/dL blood lead per µg/m^3 increment in air lead concentration for men and 1.3 for women. These are the slope estimates used in this analysis.

Individuals with initial blood lead levels of 30 µg/dL and greater

For individuals with high blood lead levels, the air lead to blood lead uptake slopes have been shown to be much shallower, as described by U.S. EPA (1986b). An appropriate change in blood lead per change in air lead is 0.5 µg/dL per µg/m^3 for individuals that have initial blood lead levels in the range of 30 to 40 µg/dL. This value is based on cross-sectional and experimental studies.[40] For individuals with initial blood lead levels greater than 40 µg/dL, an appropriate range of slopes is 0.03 to 0.2, as determined by occupational studies listed in Table 11-37 of U.S. EPA (1986b). The median value of these studies is 0.07. These two slopes (0.5 for the population with blood lead levels between 30 and 40 µg/dL and 0.07 for blood lead levels greater than 40 µg/dL) are used for both children and adults in this analysis. These relationships are summarized in Table G-4.

Estimates of Initial Blood Lead Concentrations

The benefits model requires an initial distribution of blood lead levels in the exposed populations to model health benefits of reducing lead air emissions. The model estimates the new distribution of blood lead levels that would exist after a given change in air concentrations using the slopes described above. Finally, the model estimates the difference between the two distributions. This analysis begins with an initial 1970, no-control scenario blood lead distribution from which all subsequent changes are modeled. This approach requires an estimate of the blood lead distributions in the U.S. population in 1970. Unfortunately, there are no actual national blood lead distribution estimates for 1970. Although the first NHANES study covered 1970, blood lead data were not collected in this study.[41] Nonetheless, a 1970 distribution of blood lead was estimated using NHANES II data (from 1976-1980), combined with estimates of typical changes in blood lead levels from 1970-1976 observed in localized screening studies.

Table G-4. Estimated Indirect Intake Slopes: Increment of Blood Lead Concentration (in µg/dL) per Unit of Air Lead Concentration (µg/m^3).

	Individuals with blood lead levels < 30 µg/dL	Individuals with blood lead levels 30-40 µg/dL	Individuals with blood lead levels > 40 µg/dL
Adult Males	1.8	0.5	0.07
Adult Females	1.3	0.5	0.07
Children	4.0	0.5	0.07

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[38] Goldsmith (1974) refrigerated (rather than froze) the blood samples, and did not analyze the samples until 8 or 9 months after they were taken, and restricted the analysis to one determination for each blood sample. Tsuchiya et al. (1975) measured air lead concentrations after blood samples were taken; blood was drawn in August and September of 1971, whereas air samples were taken during the 13 month period from September 1971 to September 1972.

[39] Tepper and Levin, 1975; Johnson et al., 1976; Nordman, 1975; Goldsmith, 1974; Daines et al., 1972.

[40] U.S. EPA, 1986b.

[41] NCHS, 1993a.

A major drawback to this approach is the uncertainty in deriving the 1970 estimates. Another drawback to beginning with the 1970 level and modeling changes from that point is the analysis only represents changes in lead exposure from air; reductions from other sources of lead exposure are not accounted for. The purpose of this analysis is to identify changes attributable to the CAA mandates; changes from other sources of lead exposure should not be considered. However, due to nonlinear nature of the lead concentration-response functions (see above), the overall exposure context in which the air lead exposure reductions take place will influence the estimate of benefits from those reductions. Specifically, at higher blood lead levels, the slope of the concentration-response curve is shallower than at lower levels. As a result, a given change in the mean blood lead level may result in a smaller change in the health effect if the change occurs from a relatively high starting level. On the other hand, if one accounts for the fact that other sources of lead exposure are reduced at the same time that the given air reductions occur, then those air emissions reductions may result in greater changes in health risk.

This issue is of concern even though the analysis focuses on the difference between the control and no-control scenarios, since the health benefit implications of the emissions differentials between the scenarios will depend on the point on the blood lead distribution curve at which the differences are considered. That is, a difference between a mean blood lead of 25 µg/dL and one of 20 µg/dL may have different health implications than a difference between 15 µg/dL and 10 µg/dL, even though the absolute value of the difference is the same (5 µg/dL).

An alternative method is to “start” with a 1990 blood lead level and to “back-calculate” benefits by representing the differentials as increases over the 1990 levels, rather than decreases from 1970 levels. The advantage of this approach is that it accounts for reductions in lead exposure from other sources, as represented by current blood lead levels. Its disadvantage is that it holds other sources constant to (lower) 1990 levels, and thus the modeling may underestimate actual blood lead distributions in earlier years, and thereby overestimate benefits from controls during those years. This analysis presents the results of both approaches, indicated as “forward-looking” and “backward-looking”.

Combination of Air Concentration Estimates with Population Data

The modeled air lead concentrations at various distances from the sources were combined with population data from the Census Bureau to arrive at an estimate of the number of cases of health effects for each of the years from 1970 to 1990 in both the control and no-control scenarios. The primary census information was accessed from the Graphical Exposure Modeling System Database (GEMS), an EPA mainframe database system. The following data were obtained from GEMS for the years 1970, 1980, and 1990: total population for each Block Group/Enumeration District (BG/ED); state and county FIPS codes associated with each BG/ED; latitude and longitude of each BG/ED; and population of males under 5 and females under 5 for each BG/ED. The intervening five year intervals (1975 and 1985) were estimated using the Intercensal County Estimates from the Census, which estimate annual populations on a county by county basis. The decennial Census data and the Intercensal County Estimates data sets were related by county FIPS codes; the population in each BG/ED was assumed to grow or shrink at the same rate as the county population as a whole.

Since the concentration-response data are particular to specific sex and adult age groups, additional population data were also required to determine the sizes of affected subpopulations. For 1990 age and sex, the U.S. Census, 1992 was used, with age groups tallied as necessary. For 1980 age and sex, the U.S. Census, 1982 was used, with age groups also tallied as necessary. The 1970 age and sex breakdowns were obtained through personal communication with the Census Bureau.[42] The age and sex percentages were interpolated for intervening years.

Pregnant women are often a subpopulation of interest for lead effects. Although pregnant women themselves may be harmed by exposure to lead, this analysis was concerned with pregnant women because of possible effects on their fetuses who will be born and evince effects as young children. To estimate the number of exposed fetuses who were born during the years of interest,[43] birth rates for 1970, 1980 and 1990 were obtained from the Census Bureau.[44] These birth rates were used to interpolate for years between 1970 and 1980, and for the years between 1980 and 1990.

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[42] Personal communication, Karl Kuellmer, Abt Associates and the Bureau of Census, Population, Age and Sex telephone staff, March, 1994.

Results

For both the control and no-control scenarios, Table G-5 shows estimated lead emissions from electric utilities, industrial processes, and industrial combustion. Tables G-6 and G-7 show the differences in health impacts between the two scenarios (for industrial processes, industrial combustion and electric utilities only) for the "forward-looking" and "backward-looking" analyses. The modeled population for each year is also presented.

Table G-5. Estimated Lead Emissions from Electric Utilities, Industrial Processes, and Industrial Combustion (in Tons).

	1970	1975	1980	1985	1990
Electric Utilities[a] Control Scenario		1,351	636	175	190
Electric Utilities[a] No-control Scenario		2,309	3,143	3,670	3,864
Industrial Processes Control Scenario	7,789	3,317	1,032	670	658
Industrial Processes No-control Scenario	7,789	7,124	6,550	5,696	5,305
Industrial Combustion Control Scenario	4,329	4,354	1,880	190	187
Industrial Combustion No-control Scenario	4,329	4,457	4,653	4,584	4,596

[a] Appropriate data on electric utilities do not exist for years prior to 1975.

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[43] Note that we do not record the number of pregnancies, since the valuation only applies if the child is born and lives to exhibit the effect. Neither are we concerned with whether the births are single or multiple births, since each fetus is at risk, whether a pregnant women carries one or more fetuses.

[44] Personal communication, Karl Kuellmer, Abt Associates and the Bureau of Census, Population, Fertility/Births telephone staff.

Table G-6. Yearly Differences in Number of Health Effects Between the Control and No-control Scenarios: Industrial Processes, Boilers, and Electric Utilities (Holding Other Lead Sources at Constant 1970 Levels).

Health Effect	**1975**	**1980**	**1985**	**1990**
Mortality				
Men (40-54)	0.1	1.5	2.5	2.7
Men (55-64)	0.0	1.1	1.8	1.8
Men (65-74)	0.0	0.4	0.7	0.8
Women (45-74)	0	0.8	1.3	1.4
Infants	0	0.001	0.002	0.002
Total	0.1	3.9	6.3	6.7
Coronary Heart Disease				
Men (40-54)	0.1	1.8	3.0	3.3
Men (55-64)	0.0	0.7	1.2	1.2
Men (65-74)	0.0	1.0	1.6	1.7
Women (45-74)	0.1	1.3	2.1	2.1
Total	0.2	4.8	8.0	8.3
Strokes				
Cerebrovascular Accident (men 45-74)	0.1	1.1	1.8	1.8
Cerebrovascular Accident (women 45-74)	0	0.5	0.9	0.9
Brain Infarction (men 45-74)	0	0.7	1.1	1.1
Brain Infarction (women 45-74)	0	0.4	0.6	0.6
Total	0.1	2.7	4.4	4.4
Hypertension (men 20-74)	149	3,790	6,350	6,670
IQ Decrement				
Lost IQ Points	630	14,300	22,700	23,900
IQ<70 (cases)	3	60	120	125
Population Exposed (millions)	188	197	207	217

Table G-7. Yearly Differences in Number of Health Effects Between the Control and No-control Scenarios: Industrial Processes, Boilers, and Electric Utilities (Holding Other Lead Sources at Constant 1990 Levels).

Health Effect	**1975**	**1980**	**1985**	**1990**
Mortality				
Men (40-54)	0.3	6.9	11.5	12.5
Men (55-64)	0.2	5.1	8.3	8.2
Men (65-74)	0.1	2.0	3.5	3.9
Women (45-74)	0.2	3.9	6.4	6.4
Infants	0	0.001	0.002	0.002
Total	0.8	17.9	29.7	31.0
Coronary Heart Disease				
Men (40-54)	0.4	8.3	13.8	15.0
Men (55-64)	0.1	3.4	5.6	5.6
Men (65-74)	0.2	4.4	7.6	8.0
Women (45-74)	0.2	5.9	9.6	9.7
Total	0.9	22.1	36.6	38.3
Strokes				
Cerebrovascular Accident (men 45-74)	0.2	5.0	8.1	8.2
Cerebrovascular Accident (women 45-74)	0.1	2.6	4.1	4.2
Brain Infarction (men 45-74)	0.1	2.8	4.6	4.7
Brain Infarction (women 45-74)	0.1	1.6	2.7	2.7
Total	0.5	12.0	19.5	19.8
Hypertension (men 20-74)	422	10,800	18,100	19,000
IQ Decrement				
Lost IQ Points	630	14,300	22,700	23,900
IQ<70 (cases)	0	31	50	61
Population Exposed (millions)	188	197	207	217

Reduction in Health Effects Attributable to Gasoline Lead Reductions

Estimating Changes in Amount of Lead in Gasoline from 1970 to 1990

The relationship between the national mean blood lead level and lead in gasoline is calculated as a function of the amount of lead in gasoline consumed. Thus, to calculate the health benefits from gasoline lead reductions, necessary inputs are estimates of lead in gasoline consumed over the period 1970 to 1990 and the amount of lead in gasoline that would have been consumed in the absence of the Clean Air Act. These values are calculated using the quantity of both leaded and unleaded gasoline sold each year and the concentration of lead in leaded and unleaded gasoline for each year in the period of interest. For each year, the relationship is expressed as:

$$LEAD = \left(\frac{SOLD}{365\ days}\right) \times [FRAC_{Pb} \times PB_{leaded} + (1 - FRAC_{Pb}) \times PB_{unleaded}] \qquad (29)$$

where:

$LEAD$ = average lead per day in gasoline sold in a given year (metric tons/day),

$SOLD$ = total quantity of gasoline sold (million gal/yr),

$FRAC_{Pb}$ = fraction of total gasoline sales represented by leaded gasoline (dimensionless),

Pb_{leaded} = lead content of leaded gasoline (g/gal), and

$Pb_{unleaded}$ = lead content of unleaded gasoline (g/gal).

Gasoline Sales (SOLD): Data on annual gasoline sales were taken from a report by Argonne National Laboratories (1993) which presented gasoline sales for each state in five year intervals over the period 1970-1990. This analysis used linear interpolation to estimate the gasoline sales for years between the reported years. These data were summed to obtain national sales figures.

Fraction of Total Sales Comprised of Leaded Gasoline ($FRAC_{Pb}$): For the control scenario, this analysis used information reported by Kolb and Longo (1991) for the fraction of the gasoline sales represented by leaded gasoline for the years 1970 through 1988. For 1989 and 1990, data were taken from DOE (1990 and 1991, respectively). For the no-control scenario, all of the gasoline sold was assumed to be leaded for all years.

Lead Content of Gasoline (Pb_{leaded} and $Pb_{unleaded}$): Argonne National Laboratory in Argonne, Illinois was the source for the data on the lead content of leaded and unleaded gasoline for the period 1974-1990. Argonne compiled these data from historical sales data submitted to EPA, from Clean Air Act regulations on lead content, and from recent Motor Vehicle Manufacturers Association (MVMA) surveys. For 1970 through 1973, this analysis assumed the lead content of gasoline to be at the 1974 level. For the no-control scenario, this analysis used the 1974 lead content in leaded gasoline as the lead content in all gasoline for each year.

Estimating the Change in Blood Lead Levels from the Change in the Amount of Lead in Gasoline

Several studies have found positive correlations between gasoline lead content and blood lead levels.[45] Data from the National Health and Nutrition Examination Survey (NHANES II) have been used by other researchers who determined similar positive correlations between gasoline lead and blood lead levels.[46]

The current analysis used a direct relationship between consumption of lead in gasoline and blood lead levels to estimate changes in blood lead levels resulting from Clean Air Act regulation of the lead content of gasoline. This relationship was based on regression analyses of the reduction of leaded gasoline presented in the 1985 Regulatory Impact Analysis (RIA).[47] Several multiple regressions were performed in the RIA to relate gasoline usage with individuals' blood lead levels, which were taken from NHANES II. These regressions of blood lead on gasoline usage controlled for such variables as age, sex, degree of urbanization, alcohol consumption, smoking, occupational exposure, dietary factors, region of the country, educational attainment, and income. The regressions suggested that a decrease of 100 metric tons per day (MTD) of lead used in gasoline is associated with a decrease in mean blood lead concentration of 2.14 µg/dL for whites and 2.04 µg/dL for blacks. In both of these regressions, gasoline use was found to be a highly significant predictor of blood lead ($p < 0.0001$).[48]

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[45] U.S. EPA, 1985; Billick et al., 1979; Billick et al., 1982.

[46] Janney, 1982; Annest et al., 1983; Centers for Disease Control, 1993; National Center for Health Statistics, 1993b.

[47] U.S. EPA, 1985.

To determine a single gasoline usage-blood lead slope for the entire population of the U.S., this analysis used the average of the slopes for blacks and for whites, weighted by the percentage of blacks and whites in the U.S. during the time period of the analysis.[49] The resulting relationship is 2.13 µg/dL blood lead per 100 metric tons of lead in gasoline consumed per day. The same relationship was used to model changes in both children's and adults' blood lead levels. The U.S. EPA (1985) analyzed data from a study of black children in Chicago during the time period 1976 to 1980 and determined a slope of 2.08 µg/dL per 100 MTD. This slope for children is very similar to the one used in this analysis.

1970-Forward and 1990-Backward Approaches

As with the industrial processes and boilers analysis, this analysis used two different approaches to determine mean blood lead levels based on changes in lead concentrations in gasoline. In the 1970-forward approach, the calculations began with the estimated blood lead level for 1970. The change in blood lead level from one year to the next was based upon the change in the amount of lead in gasoline sold, as discussed above, for both the control and no-control scenarios. For example, to calculate the blood lead level for 1971, the calculated change in blood lead from 1970 to 1971 was added to the 1970 value. This process was repeated for each succeeding year up to 1990.

The 1990-backward approach began with a mean blood lead level in 1990 for the control scenario. For the no-control scenario, the starting blood lead was estimated from the 1990 level used in the control scenario, plus an additional blood lead increment resulting from the difference between the 1990 consumption of lead in gasoline under the two scenarios. Again, the difference in mean blood lead levels from one year to the next was based on the change in gasoline lead for the corresponding years. For example, the difference in blood lead levels between 1990 and 1989 was subtracted from the 1990 level to determine the 1989 level. The process was continued for each year back to 1970.

Relating Blood Lead Levels to Population Health Effects

The mean blood lead levels calculated using the methods described above were used in the dose-response functions for various health effects (e.g., hypertension, chronic heart disease, mortality). This information was then combined with data on the resident population of the 48 conterminous states in each year to determine the total incidence of these health effects attributable to lead in gasoline. A Department of Commerce Publication (1991) was used to obtain the total population in 1970, 1980, and 1983-1990, while a different publication was the source of the 1975 population values.[50] Linear interpolation was used to estimate the populations in years for which specific data were not available.

For certain health effects, it was necessary to know the size of various age groups within the population. Two different sources were used to estimate the proportions of the population in the age groups of interest. A U.S. Census summary (U.S. Dept. of Commerce, 1990) was used for information for 1990 for children and adults and for 1980 for adults, and Census Telephone Staff (U.S. Dept. of Commerce, 1994) provided information for 1980 for children and 1970 for children and adults. The populations for the intervening years were estimated by linear interpolation.

Changes in Leaded Gasoline Emissions and Resulting Decreased Blood Lead Levels and Health Effects

Table G-8 shows the estimated quantity of lead burned in gasoline in the five year intervals from 1970 to 1990. Tables G-9 and G-10 show the difference in health impacts between the two scenarios (for lead in gasoline only) for the "forward-looking" and "backward-looking" analyses. In general, health effect benefits resulting from gasoline lead reductions exceed those predicted from lead reductions at the point sources examined (i.e., industrial processes and boilers and electric utilities) by three orders of magnitude.

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[48] U.S. EPA, 1985.

[49] U.S. Department of Commerce, 1992. Although the percentages of blacks and whites changed slightly over this time period (1970-1990), the change did not affect the value of the weighted slope.

[50] U.S. Dept. of Commerce, 1976.

Table G-8. Lead Burned in Gasoline (in tons).

	1970	1975	1980	1985	1990
Control Scenario	176,100	179,200	86,400	22,000	2,300
No-control Scenario	176,100	202,600	206,900	214,400	222,900

Table G-9. Yearly Differences in Number of Health Effects Between the Control and No-control Scenarios: Lead in Gasoline only (Holding Other Lead Sources at Constant 1970 Levels).

Health Effect	**1975**	**1980**	**1985**	**1990**
Mortality				
Men (40-54)	309	1,820	3,340	4,150
Men (55-64)	220	1,340	2,380	2,700
Men (65-74)	81	520	999	1,260
Women (45-74)	155	939	1,710	2,060
Infants	456	2,340	3,930	4,940
Total	1,220	6,960	12,400	15,100
Coronary Heart Disease				
Men (40-54)	230	1,360	2,540	3,280
Men (55-64)	92	563	1,030	1,220
Men (65-74)	113	723	1,380	1,750
Women (45-74)	73	442	805	965
Total	508	3,090	5,760	7,210
Strokes				
Cerebrovascular Accident (men 45-74)	147	884	1,610	1,960
Cerebrovascular Accident (women 45-74)	73	442	805	965
Brain Infarction (men 45-74)	85	508	927	1,130
Brain Infarction (women 45-74)	47	286	521	624
Total	352	2,120	3,862	4,679
Hypertension (men 20-74)	677,000	4,200,000	7,840,000	9,740,000
IQ Decrement				
Lost IQ Points	1,030,000	5,020,000	8,580,000	10,400,000
IQ<70 (cases)	3,780	20,100	36,500	45,300
Population Exposed (millions)	214	225	237	247

Table G-10. Yearly Differences in Number of Health Effects Between the Control and No-control Scenarios: Lead in Gasoline only (Holding Other Lead Sources at Constant 1990 Levels).

Health Effect	**1975**	**1980**	**1985**	**1990**
Mortality				
Men (40-54)	476	3,040	6,140	7,950
Men (55-64)	342	2,250	4,430	5,240
Men (65-74)	128	886	1,880	2,480
Women (45-74)	242	1,590	3,210	4,030
Infants	456	2,340	3,930	4,940
Total	1,640	10,100	19,600	24,600
Coronary Heart Disease				
Men (40-54)	356	2,280	4,690	6,310
Men (55-64)	142	945	1,910	2,370
Men (65-74)	176	1,220	2,570	3,380
Women (45-74)	113	740	1,490	1,860
Total	787	5,180	10,700	13,900
Strokes				
Cerebrovascular Accident (men 45-74)	225	1,460	2,940	3,720
Cerebrovascular Accident (women 45-74)	113	740	1,490	1,860
Brain Infarction (men 45-74)	129	837	1,680	2,120
Brain Infarction (women 45-74)	73	477	955	1,190
Total	540	3,514	7,065	8,890
Hypertension (men 20-74)	984,000	6,350,000	12,300,000	15,600,000
IQ Decrement				
Lost IQ Points	1,030,000	5,030,000	8,580,000	10,400,000
IQ<70 (cases)	3,790	20,100	36,500	45,300
Population Exposed (millions)	214	225	237	247

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Appendix H: Air Toxics

Introduction

Air toxics are defined as air pollutants other than those six criteria pollutants for which EPA sets acceptable concentrations in ambient air. The SARA 313 Toxic Release Inventory (TRI), covering 328 of the approximately 3000 potentially hazardous compounds detected in air, estimated that approximately 1.2 million tons of air toxics were released to the atmosphere in 1987 from U.S. stationary sources alone. While the TRI estimate tends to understate emissions of toxics for a number of reasons, it does show that large quantities of toxics are emitted into the atmosphere annually.

Effects of air toxics emissions are divided into three categories for study and assessment: cancer; "noncancer" effects, e.g. a wide variety of serious health effects such as abnormal development, birth defects, neurological impairment, or reproductive impairment, etc.; and ecological effects. Each year, these air toxics emissions contribute to significant adverse effects on human health, human welfare, and ecosystems. In EPA's 1987 *Unfinished Business Report*[1] cancer and noncancer air toxics risk estimates were considered sufficiently high, relative to risks addressed by other EPA programs, that the air toxics program area was among the few rated "high risk".

Limited Scope of this Assessment

The effects of air toxics emissions are difficult to quantify. The adverse health effects of toxics are often irreversible, not mitigated or eliminated by reduction in ongoing exposure, and involve particularly painful and/or protracted disease. Therefore these effects are not readily studied and quantified in human clinical studies, in contrast to, for example, ambient ozone. In addition, epidemiological studies of effects in exposed populations are often confounded by simultaneous exposure of subjects to a variety of pollutants. Therefore, the effects of air toxics are often quantified by extrapolating data from animal studies to human exposure and expressed as risk per unit of exposure. Incidence of noncancer effects, for example, often are difficult to translate into monetized benefits.

Similarly, the quantification of ecological effects due to emissions of air toxics is hampered by lack of sufficient information regarding contribution of sources to exposure, associations between exposure to mixtures of toxics and various ecological endpoints, and economic valuation for ecological endpoints.

The air toxics portion of this study is, of necessity, separate and more qualitative in nature than the benefit analysis conducted for the criteria air pollutants. Limitations in the quantitative analyses of air toxics effects led the Project Team to decide to exclude the available quantitative results from the primary analysis of CAA costs and benefits. Table H-1 presents the range of potential human health and ecological effects that can occur due to air toxics exposure. As indicated, this appendix presents quantitative estimates of benefits of CAA air toxics control for the cancer mortality endpoint for only nonutility stationary source and mobile source categories. Noncancer effects and ecological effects are described qualitatively.

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[1] U.S. EPA. Office of Policy Planning and Evaluation. *Unfinished Business: A Comparative Assessment of Environmental Problems*. February 1987.

Table H-1. Health and Welfare Effects of Hazardous Air Pollutants.

Effect Category	**Quantified Effects**	**Unquantified Effects**	**Other Possible Effects**
Human Health	Cancer Mortality - nonutility stationary source - mobile source	Cancer Mortality - utility source - area source Noncancer effects - neurological - respiratory - reproductive - hematopoetic - developmental - immunological - organ toxicity	
Human Welfare		Decreased income and recreation opportunities due to fish advisories Odors	Decreased income resulting from decreased physical performance
Ecological		Effects on wildlife Effects on plants Ecosystem effects Loss of biological diversity	Effects on global climate
Other Welfare		Visibility Materials Damage	

History of Air Toxics Standards under the Clean Air Act of 1970

The 1970 Clean Air Act required the EPA to list a chemical as a hazardous air pollutant if it met the legislative definition provided:

> *"The term 'hazardous air pollutant' means an air pollutant to which no ambient air quality standard is applicable and which in the judgment of the Administrator may cause, or contribute to, an increase in mortality or an increase in serious irreversible, or incapacitating reversible, illness."*[2]

Once a HAP was listed, the EPA Administrator was required to:

> *"establish any such standard at the level which in his judgment provides an ample margin of safety to protect the public health from such hazardous air pollutant."*[3]

In other words the EPA had to first determine that a chemical was a HAP, and then regulate the emissions of each HAP based solely on human health effects and with an ample margin of safety. This regulatory mandate proved extremely difficult for EPA to fulfill, for reasons discussed below, and the result was that only seven HAPs were regulated over a period of 20 years.

Listing chemicals became a difficult task because of debates within and outside of the EPA surrounding issues of how much data are needed and which methodologies should be used to list a chemical as a HAP. An even more difficult issue was how to define the Congressional mandate to provide an "ample margin of safety." For carcinogens, there is generally no threshold of exposure considered to be without risk. What level of risk, then, is acceptable, and how should it be calculated? The EPA struggled to provide answers to these questions, and was challenged in court. The end result was a 1987 ruling by the D.C. Circuit Court that provided the EPA with a legal framework with which to determine an "ample margin of safety." This framework was interpreted and used by the EPA in its 1989 benzene regulations.

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[2] 42 U.S.C. §1857(a)(1).

[3] 42 U.S.C. §1857(b).

Quantifiable Stationary Source Air Toxics Benefits

One might be tempted to presume that the few federal HAP standards set would have achieved relatively substantial reductions in quantifiable risk. While some standards set under section 112 of the Clean Air Act appear to have achieved significant reductions in cancer incidence, the coverage, quantification, and monetization of the full range of potential adverse effects remains severely limited. This fact serves to highlight the inadequacy of current methods of evaluating HAP control benefits. This limited ability to estimate the total human health and ecological benefits of HAP reductions is an important area for future research. Thus the quantifiable benefits for CAA air toxics control presented here are limited in scope.

There are three sources of information that provide a picture of potential stationary source air toxics benefits of the CAA. EPA's Cancer Risk studies attempted to broadly assess the magnitude and nature of the air toxics problem by developing quantitative estimates of cancer risks posed by selected air toxics and their sources. Secondly, risk assessments conducted in conjunction with the promulgation of National Emissions Standards for Hazardous Air Pollutants (NESHAPs) offer a snapshot of potential monetized cancer mortality benefits. Finally, the Project Team attempted to estimate historical non-utility stationary source HAP-related direct inhalation cancer incidence reductions. Results from each of these studies are presented below.

EPA Analyses of Cancer Risks from Selected Air Toxic Pollutants

The Agency conducted two efforts to broadly assess the magnitude and nature of the air toxics problem. The 1985 report entitled, "The Air Toxics Problem in the United States: An Analysis of Cancer Risks for Selected Pollutants"[4] otherwise known as the "Six Month Study," was intended to serve as a "scoping" study to provide a quick assessment of the air toxics problem utilizing only readily available data on compound potencies, emissions, and ambient pollutant concentrations. The Agency updated this analysis of cancer risks in the 1990 report entitled "Cancer Risk from Outdoor Exposure to Air Toxics" referred to here as the "1990 Cancer Risk study."[5]

For the pollutant and source categories examined, the 1990 Cancer Risk study estimated the total nationwide cancer incidence due to outdoor concentrations of air toxics to range from 1,700 to as many as 2,700 excess cancer cases per year, with 14 compounds accounting for approximately 95 percent of the annual cancer cases. Additionally, point sources contribute 25 percent of annual cases and area sources contribute 75 percent of annual cases. Mobile sources account for 56 percent of the nationwide total.[6]

The Six Month study indicates that the criteria air pollutant programs appear to have done more to reduce air toxics levels during the 1970 to 1990 period than have regulatory actions aimed at specific toxic compounds promulgated during the same period. Metals and polynuclear compounds usually are emitted as particulate matter and most of the volatile organic compounds are ozone precursors. As such, they are regulated under State Implementation Plan (SIP) and New Source Performance Standard (NSPS) programs and Title II motor vehicle regulations. A number of reports cited indicate significant reductions in air toxics emissions attributable to actions taken under SIP, NSPS and mobile source programs. Additionally, EPA conducted a comparison of air quality and emissions data for 1970 with the estimates of cancer incidence for 1980.[7] Methods, assumptions and pollutants included were held constant over the period. The analysis showed a significant decrease in incidence during the decade due to improvements in air quality, presumably related to general regulatory programs. For the 16 pollutants studied, estimated nationwide cancer incidence decreased from 3600 in 1970 to 1600 in 1980. The 1990 Cancer Risk Study did not attempt to update this analysis.

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[4] U.S. EPA. Office of Air Quality Planning and Standards. *The Air Toxics Problem in the United States: An Analysis of Cancer Risks for Selected Pollutants*. May 1985. EPA-450/1-85-001.

[5] U.S. EPA. Office of Air Quality Planning and Standards. *Cancer Risk from Outdoor Exposure to Air Toxics*. September 1990. EPA-450/1-90-004a.

[6] The 1990 Cancer Risk study reported approximately 500 - 900 more cancer cases per year than the Six Month Study due primarily to the inclusion of more pollutants, better accounting of emissions sources, and, in some cases, increases in unit risk estimates.

Although it is difficult to draw quantitative conclusions from these two studies regarding the benefits of CAA air toxics control, it is apparent that the pollutant-specific and source category-specific NESHAPs were not structured to reduce significant air toxic emissions from area and mobile sources. In fact, the 1990 Cancer Risk Study indicates that considerable cancer risk remained prior to passage of the 1990 CAA Amendments: as many as 2,700 excess cancer cases annually. Some studies indicate that the criteria air pollutant program played a critical role during the 1970 to 1990 period in achieving air toxic emission reductions and therefore decreasing cancer risk.

Cancer Risk Estimates from NESHAP Risk Assessments

In looking back at the estimated effects of the HAP standards, EPA found that the effects of the NESHAPs were not quantified completely. These estimates occurred at a time when emission estimation and risk assessment methodologies for HAPs were first being developed. One consequence is that because emissions were not fully characterized, air toxics exposures could not be completely assessed. Additionally, most assessments only focused on the specific HAP being listed under the CAA and did not assess the reduction of other pollutants, which are currently considered HAPs. For example, while the vinyl chloride standard reduces emissions of ethylene dichloride, these emission reductions were not assessed in the risk assessment. In a different context, reductions of HAP may also achieve reductions of VOC and PM. The benefits of such reductions generally were also not evaluated. In addition, EPA generally did not assess the potential exposure to high, short-term concentrations of HAP and therefore did not know whether toxic effects from acute exposures would have been predicted and possibly addressed by the HAP standards.

In addition, people living near emission sources of concern are often exposed to a mix of pollutants at once. Some pollutants have been shown to act synergistically together to create a health risk greater than the risk that would be expected by simply adding the two exposure levels together. More research is needed to understand the effects of multiple-pollutant exposures. Finally, HAP risks tend to be distributed unevenly across exposed populations, with particularly high exposures occurring closest to emission sources. It should be noted that HAP exposure to specific populations may tend to fall disproportionately among the poor and minorities, who are more likely to live in close proximity to emitting facilities.

With the above caveats in mind, Table H-2 provides information about maximum individual risk taken from the Federal Register notices for the NESHAPs promulgated before the 1990 amendments to the Clean Air Act. The benefits are calculated by multiplying the estimated annual incidence reduction by the $4.8 million valuation per statistical life (1990 dollars). These benefit estimates provide a snapshot of potential monetized benefits for the year in which each NESHAP was promulgated. Of course these estimates do not include air toxics benefits for other health and ecological benefit categories, or air toxics benefits from co-control of criteria air pollutants. All uncertainties associated with the original estimates remain.

Non-utility Stationary Source Cancer Incidence Reductions

The Project Team commissioned two studies to estimate reductions in cancer incidence due to pre-1990 NESHAPs: the PES Study and the ICF Re-analysis. The methodology used for most air pollutant evaluations involved a “back calculation” for the estimation of incidence reductions. However, the EPA has elected not to rely on the results of this analysis given critical methodological flaws. Despite the Project Team’s concerns, the methodology and results of the two studies are presented below in the interest of full disclosure and to guide efforts to develop a more valid

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[7] Hunt, W.F., Faoro, R.B. and Curran, T.C., “Estimation of Cancer Incidence Cases and Rates for Selected Toxic Air Pollutants Using Ambient Air Pollution Data, 1970 vs. 1980”. U.S. EPA. April 1985.

Table H-2. Cancer Incidence Reductions and Monetized Benefits for NESHAPs.

Pollutant	Source Category	Year Promulgated	Pre-Reg Maximum Individual Risk	Post-Reg Maximum Individual Risk	Reduction in Cancer Incidence (per year)	Benefits in $million per year (1990$)
benzene		1985	1.5x10^{-3}	4.5x10^{-4}	.31	1.5
benzene	coke by-product	1984	7x10^{-3}	2x10^{-4}	1.95	9.4
benzene	storage vessels	1982	4.5x10^{-4}	3x10^{-5}	0.01 to 0.06	0.05 to 0.3
benzene	waste operations	1986	2x10^{-3}	5x10^{-5}	0.55	2.6
benzene	transfer operations	1987	6x10^{-3}	4x10^{-5}	0.98	4.7
arsenic	primary copper	1986	1.3x10^{-3} to 5x10^{-6}	1.2x10^{-3} to 3x10^{-6}	0.09	0.4
arsenic	glass manuf.	1986	7x10^{-4} to 3x10^{-5}	1.7x10^{-4} to 6x10^{-6}	0.117 to 0.0034	0.02 to 0.6
asbestos	demolition	1973			100	480
vinyl chloride	PVC production	1975			10.5	50.4

and reliable analysis of the health-related benefits of HAP reductions in the upcoming section 812 Prospective studies.

PES Study

Methodology

The first attempt to estimate, for this study, historical non-utility stationary source HAP-related direct inhalation cancer incidence reductions was conducted by Pacific Environmental Services (PES). The basic approach used in the PES study was to adjust the cancer incidence estimates developed for EPA's 1990 Cancer Risk study to reflect the changes in emissions of, and exposures to, 14 key HAPs: arsenic, asbestos, benzene, 1,3-butadiene, carbon tetrachloride, chloroform, hexavalent chromium, dioxin, ethylene dichloride, ethylene dibromide, formaldehyde, gasoline vapors, products of incomplete combustion (PICs), and vinyl chloride.

The first step was to compile baseline incidence levels, defined as cancer cases per million population, for each of the 14 pollutants. The point estimates of incidence from the 1990 Cancer Risk study were used for this purpose. For some source categories, the "best point estimate" from the 1990 Cancer Risk study was used, for others a mid-point was selected.[8] These baseline incidence levels were based on measured ambient concentrations of the pollutant, modeled concentrations, or both.

The second step involved allocating baseline incidence levels to the individual source categories known to emit the relevant pollutant. In some cases, adjustments were made to reflect differences among the vintages of source category-specific data.[9] All baseline incidence estimates were ultimately expressed relative to a 1985 base year.[10] The assumption was then made that source-category incidence rates were proportional to the level of emissions from that source category.

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[8] For some of the source categories, the original NESHAP/Air Toxic Exposure and Risk Information System (NESHAP/ATERIS) estimates of incidence were not available, in which case the baseline incidence was obtained from the 1989 National Air Toxics Information Clearinghouse(NATICH) Database Report. (See PES, "Draft Summary of Methodology Used for Cancer from Stationary Sources," memorandum from Ken Meardon, PES to Vasu Kilaru, US EPA, March 22, 1993, p. 2.)

Next, levels of control for each source category-specific incidence rate were estimated for each of the target years of the present analysis (i.e., 1970, 1975, 1980, 1985, and 1990).[11] Source category-specific activity level indicators were then established and linked to changes in corresponding activity indicators provided by the J/W macroeconomic modeling results. Activity levels were estimated for each source category, for each of the target years, and for each of the two scenarios.

Finally, source category/pollutant combination incidence levels for both the control and no-control scenarios were developed. These incidence levels were developed based on the baseline incidence levels, the activity indicators, and the control levels for each year. Both of these latter two factors varied between the control and no-control scenarios. The activity levels differed based on the specific levels of related sector economic activity predicted by the J/W model for the control and no-control scenario. The control levels prevailing in each of the target years were used for the control scenario, and the 1970 control level was applied throughout the 1970 to 1990 period for the no-control scenario.[12] The formula used for these calculations was as follows:[13]

$$I_{ty} = I_{by} \times [\frac{A_{ty}}{A_{by}}] \times [\frac{P_{ty}}{P_{by}}] \times [\frac{(1 - C_{ty})}{(1 - C_{by})}] \qquad (1)$$

where:

I = cancer incidence for a source category-pollutant combination
A = activity level for a source category
P = population
C = control level for a source category-pollutant combination
ty = target year (1970 ... 1990)
by = base year

Findings

The PES analysis concluded that substantial reductions in HAP-related cancer cases were achieved during the reference period of the present study. The vast majority of these estimated reductions were attributable to reduced exposures to asbestos, particularly from manufacturing and fabricating sources.[14] In fact, roughly 75 percent of the total reduction in cancer cases averaged over the 1970 to 1990 period were attributed to asbestos control.[15] Figure H-1 summarizes the PES study overall cancer incidence reductions and the relative contribution of asbestos-related reductions over the study period.

The Project Team had several concerns about the PES results. First and foremost, the reductions in asbestos-related cancer cases appeared to be substantially higher than expected, particularly in the earlier target years. Second, the control scenario activity level indicators for several sources with which Project Team members were familiar did not appear to be even remotely consistent with actual historical activity patterns.[16] Finally, the level of documentation of the analytical methodologies, assumptions, and results was insufficient to ascertain the validity and reliability of

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[9] For example, six discrete sources for vinyl chloride were identified in the Six-Month Study Update. Point estimate incidences for each of these source categories came from separate references with databases corresponding to different years. (See PES, "retrospective analysis for section 812(a) Benefits Study," September 30, 1992, p. 8.)

[10] See PES, March 22, 1993 memorandum, p. 3.

[11] Control level estimates were based on one of the following: control efficiencies for related criteria pollutants defined in the criteria pollutant analysis, reference documents such as Control Technology Guidelines (CTGs) or Background Information Documents (BIDs), preambles for related regulations, or EPA experts. (See PES, March 22, 1993 memorandum, p. 3.)

[12] More detailed descriptions of the methodology and associated uncertainties are provided in "Retrospective Analysis for section 812(a) Benefits Study," a September 30, 1992 memorandum from Ken Meardon, PES to Vasu Kilaru, US EPA.

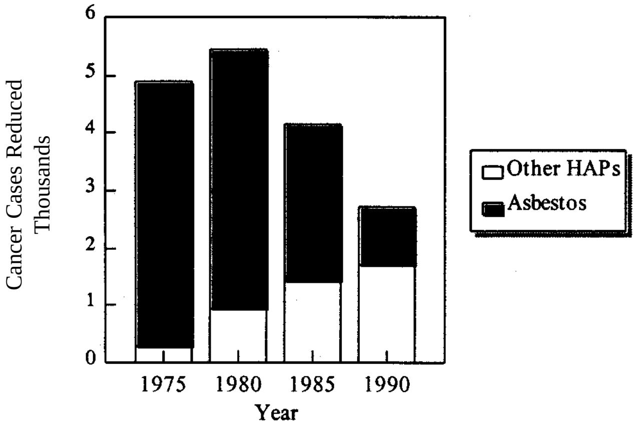
[13] See PES, March 22, 1993 memorandum, p. 4.

[14] PES, "Cancer Risk Estimates from Stationary Sources," memorandum from Ken Meardon, PES to Vasu Kilaru, US EPA, March 5, 1993.

[15] ICF, "Direct Inhalation Incidence Benefits," Draft Report, November 11, 1994, p. 10.

[16] For example, the activity indicators for Municipal Waste Combustors (MWCs) incorporated in the PES analysis decline dramatically throughout the 1975 to 1990 period. (See PES, March 5, 1993 memorandum to Vasu Kilaru, p. 10). In reality, overall MWC capacity and throughput increased significantly over this period.

Figure H-1. PES Estimated Reductions in HAP-Related Cancer Cases.



the results. Ultimately, the Project Team determined that it was necessary to conduct a formal review and re-analysis of the cancer incidence reductions associated with non-utility stationary source HAP controls. The results of the PES analysis remain a relevant part of the record of the present study, however, since they provided a substantial basis for the subsequent re-analysis by ICF Incorporated.

ICF Re-analysis

Methodology

The purposes of the ICF Re-analysis were to examine the methodology and results of the PES study, particularly to address the aforementioned concerns of the Project Team, and to develop a revised set of estimates. Due to significant constraints on the resources remaining for HAP analysis in the section 812 study, however, only a few key HAPs could be investigated in depth and many important issues could not be addressed.[17] Furthermore, the effects of two early and potentially important HAP standards –the Beryllium and Mercury NESHAPs– could not be evaluated. Nevertheless, the ICF Re-analysis clarified some potential sources of uncertainty in the PES results and provided revised cancer incidence reduction estimates for several HAPs.

A key uncertainty in the PES results was associated with the use of a "back-calculation" technique to estimate incidence reductions for some HAPs. The back-calculation technique estimates uncontrolled incidence by dividing residual incidence by the assumed control efficiency. This approach means uncontrolled incidence, and therefore incidence reductions, are highly sensitive to small changes in assumed control efficiency.[18] In some cases, the PES analysis may have used control efficiencies which were too high, resulting in overestimation of uncontrolled incidence and therefore incidence reductions attributable to the CAA.[19] The vinyl chloride incidence reduction estimates appear to be significantly influenced by the use of this back-calculation technique. Another important source of uncertainty identified by ICF involved the potential overestimation of incidence totals when source apportionment is based on measured ambient concentrations.[20] ICF was unable, however, to perform an extensive evaluation of the activity level indicators used in the PES study.[21]

The first step undertaken in the re-analysis was to conduct a screening test to identify the HAPs which accounted for the most significant estimated incidence reductions. Based on this screening analysis, ICF eliminated 1,3-butadiene, carbon tetrachloride, chloroform, gasoline vapors, chromium, formaldehyde, and PICs from the detailed re-analysis effort.

Detailed reviews were then conducted for the remaining HAPs: vinyl chloride, dioxins, ethylene dibromide (EDB), ethylene dichloride (EDC), benzene, asbestos, and arsenic. In the re-analysis of these HAPs, ICF determined whether a forward- or back-calculation technique was used for the relevant source categories of a given HAP, reviewed the regulatory history of the relevant source categories to re-evaluate the assumed control efficiencies, and reviewed the upper-bound unit risk factor for each HAP. Revised total incidence reduction estimates for each HAP and for each target year were then calculated using the same basic calculation procedure used by PES. Finally, ICF identified a number of residual deficiencies in the analysis which could only be addressed through additional research and analysis.[22]

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[17] For example, the Project Team sought to develop and apply a methodology for estimating a central tendency estimate for the total carcinogenic risk imposed by all the HAPs examined. The intent was to address concerns about potential overestimation of aggregate risk measures when combining upper bound risk estimates of multiple HAPs. Unfortunately, resources were insufficient to continue development of this methodology.

[18] An example of this back-calculation technique illustrating the sensitivity to the assumed control efficiency is presented on page 12 of the draft ICF report.

[19] See ICF Draft Report, p. 12.

[20] See ICF Draft Report, p. 9.

[21] See ICF Draft Report, p. 13.

Findings

The ICF Re-analysis largely affirmed the original results obtained by PES; primarily because the PES analysis itself served as the basis for the re-analysis and only minor adjustments were adopted for many critical variables. In particular, most Project Team concerns regarding the PES methodology could not be resolved, including uncertainties associated with activity levels, assumed control efficiencies, and the unexpectedly high estimated incidence reductions associated with asbestos. In fact, the ICF Re-analysis produced a revised upper bound estimate for vinyl chloride-related incidence reductions which were even higher than the asbestos benefits.

Several sets of results were developed by ICF and presented in either the November 1994 draft report or in briefing materials prepared for the Science Advisory Board Clean Air Act Compliance Analysis Council Physical Effects Subcommittee (SAB/ACCACAPERS) in May 1995. The first set of results is based on the assumption of 100 percent source compliance with HAP control requirements. An alternative set of results was developed assuming an 80 percent compliance rate with applicable standards. Given the linear effect of changes in compliance rates, these results were precisely 20 percent lower than the first set of estimates. At the May 1995 ACCACAPERS briefing, estimates based on the 100 percent compliance estimates were presented. For asbestos, the revised incidence reductions were presented and characterized as upper bound. The asbestos estimates were then combined with upper and lower bound estimates for vinyl chloride and for “all other compounds.” Figure H-2 presents the total cancer incidence reductions derived from the ICF Re-analysis, using the asbestos estimates combined with the lower bound estimates for non-asbestos HAPs.

Figure H-2. ICF Estimated Reductions in Total HAP-Related Cancer Cases Using Upper Bound Asbestos Incidence and Lower Bound Non-Asbestos HAP Incidence.

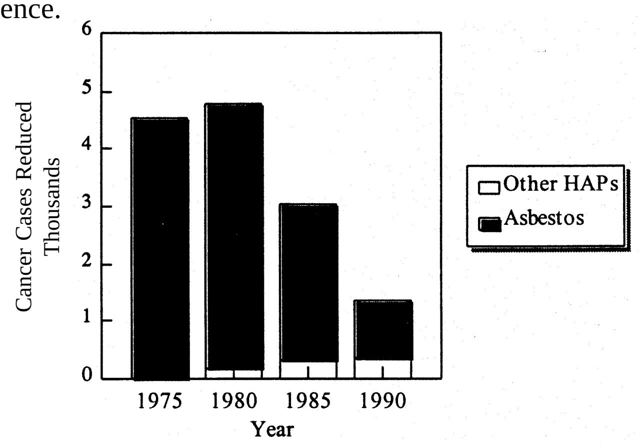

Figure H-3. ICF Estimated Reduction in Total HAP-Related Cancer Cases Using Upper Bound Incidence for All HAPs.

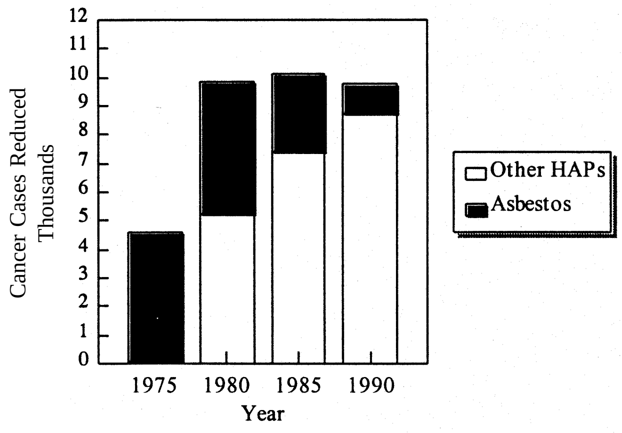

Figure H-3 presents a comparable compilation reflecting the upper bound estimates for all HAPs.

The Project Team remains concerned about these incidence reduction estimates, particularly given the doubts raised by the SAB/ACCACAPERS at the May 1995 presentation of these results. For instance, several critical assumptions are needed to make this analysis valid when applied to EPA’s NESHAPs. The flaws in these assumptions are described below.

(1) The risk estimates described in the 1990 Cancer Risk study, which served as the baseline for determining risk reductions, were accepted without question. There are myriad uncertainties in these estimates

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[22] Additional details of the ICF Re-analysis methodology can be found in ICF, “Direct Inhalation Incidence Benefits,” Draft Report, November 11, 1994.

that must be recognized, as the study was designed only to generate rough order-of-magnitude estimates of the extent of the air toxics cancer problem.

(2) The percent control efficiency for emission reductions, which is calculated in each standard, would have to apply across every facility. Typically, the emissions reductions vary between facilities; using a single average reduction could skew the results.

(3) There is a direct correlation between the number of tons of emissions reduced and incidence reduced by a specific regulation. Given the assumption of a linear, non-threshold dose-response curve (as is typically done for cancer), this is theoretically correct.

(4) Finally, the back calculation approach assumes that there is 100 percent compliance with the regulation.

EPA staff reviewed the "back calculation" approach for one of the more controversial aspects of the vinyl chloride (VC) NESHAP. The PES study estimates benefits at 426 cases reduced in 1990. The ICF Re-analysis resulted in an even higher estimate, between 1,000 and 7,000 cases annually. An analysis by EPA staff indicated that these vinyl chloride risk estimates are highly suspect given historical cancer incidence data for hepatic angiocarcinoma, a specific cancer that has been linked to vinyl chloride (Koppikar and Fegley, 1995). The following analysis demonstrates the inadequacies of the assumptions in the 1993 study.

(1) In the actual standard, no control technology was required for emissions from oxychlorination vents at ethylene dichloride (EDC)/VC plants. Applying "back calculation" for these emissions is inappropriate.

(2) In 1985, there were an estimated 8,000 fabrication plants which processed resins produced by PVC plants, thus resulting in VC emissions, which were exempt from the VC NESHAP. They emit very small quantities of VC and back calculation is not appropriate.

(3) The 1993 study uses a baseline estimate of 18 residual cases from the NESHAP/ATERIS data base. There is no evidence that these cases resulted only from emissions from PVC and EDC/VC plants.

(4) The risk analysis performed for the October 21, 1976 final VC regulation projected an incidence reduction of 11 cases per year.

In contrast, the PES study, using the "back calculation" method derived the following annual incidence reductions:

1980 - 250 cases
1985 - 360 cases
1990 - 430 cases

The subsequent back calculation conducted in the ICF Re-analysis resulted in incidence reductions as much as an order of magnitude higher than these.

Even considering the slightly different industrial output assumptions imposed by macroeconomic modeling, such a stark contrast is difficult to explain except for a critically flawed approach. Growth in activity and population nor other factors explain the difference in these two estimates. Given that the same general methodology was used for all of the air toxic pollutant assessments as was used for the VC NESHAP evaluation, there is reason to believe that cancer incidence results for the other air toxic pollutants are also flawed.

Mobile Source HAP Exposure Reductions

EPA's Cancer Risk report estimated that approximately 60 percent of the total carcinogenic risk posed by HAPs was attributed to mobile sources, with stationary sources contributing 15 percent and area sources contributing the remaining 25 percent.[23] The relative importance of mobile sources to total HAP exposure was a significant motivation behind EPA's subsequent effort to examine exposures and risks from mobile source HAPs.[24] Although available analytical resources were severely limited, the Project Team nevertheless decided it was necessary to perform at least an initial screening analysis to estimate the differences in mobile source HAP exposures between the control and no-control scenarios configured for the present study.

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[23] Cancer Risk report, Page ES-12.

[24] See US EPA/OAR/OMS, "Motor Vehicle-Related Air Toxics Study," EPA 420-R-93-005. April 1993.

Methodology

The approach used by ICF/SAI in conducting the mobile source HAP analysis closely followed the approach used in the EPA Motor Vehicle-Related Air Toxics Study (MVATS).[25] Recognizing the dearth of HAP ambient concentration and exposure data, both studies use carbon monoxide (CO) concentrations as the basis for estimating mobile source HAP concentrations and exposures. An important difference between the two studies, however, is that the ICF/SAI study adjusted the estimated change in ambient CO concentrations to take account of background[26] and non-mobile source[27] CO emissions. The HAP exposure function used in the ICF/SAI analysis is summarized by the following equation:

$$E = ((C \times A) - B) \times S \times M \times \frac{(VOC \times HAP)}{CO} \quad (2)$$

where :

E = exposure to motor vehicle-emitted HAP
C = annual ambient CO concentration to annual CO exposure concentration conversion factor
A = county-level annual average ambient CO concentration
B = background CO concentration
S = no-control to control scenario CO concentration adjustment factor (equals 1 for the control scenario)
M = total CO exposure to mobile source CO exposure conversion factor
VOC = VOC emissions by year, county, and scenario
HAP = VOC speciation factor by mobile source HAP
CO = CO emissions by year, county, and scenario

Details of the derivation of each of the variables applied in the above equation are provided in the ICF/SAI report. However, in essence, the calculation involves the following basic steps.

First, annual average county-level CO ambient monitoring data are compiled from the EPA Aerometric Information Retrieval System (AIRS) database. After adjusting for background and non-mobile source contributions, these annual average ambient CO concentrations are converted to annual average CO exposure concentrations. As in the EPA MVATS, this conversion is made based on the Hazardous Air Pollutant Exposure Model - Mobile Sources (HAPEM-MS) population exposure model, which takes account of time spent in five indoor and outdoor microenvironments: indoors at home, other indoor, in-vehicle, outdoors near roadway, and other outdoor.[28] After adjusting for CO exposures attributable to non-mobile sources of CO, the CO exposures are converted to exposures for each of the mobile source HAPs based on available VOC speciation data and the ratio of co-located VOC and CO emissions.[29] These calculations are repeated for the no-control scenario after adjusting for differences in CO ambient concentrations for each target year and for differences in fuel composition.

Results

By 1990, CAA controls resulted in significant reductions in exposure to motor vehicle HAPs. Figure H-4 summarizes the nationwide annual average exposure levels, in micrograms per cubic meter, for each of the five HAPs analyzed under the control and no-control scenarios. Additional detailed results, including breakdown by urban versus rural environments and comparisons with the EPA MVATS estimates, are provided in the ICF/SAI report.

Analytical resources to carry forward these exposure estimates to derive estimates of the changes in motor vehicle HAP-related adverse effects attributable to historical CAA programs were not available.

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[25] ICF/SAI, "Retrospective Analysis of Inhalation Exposure to Hazardous Air Pollutants from Motor Vehicles," October 1995, p. 4.

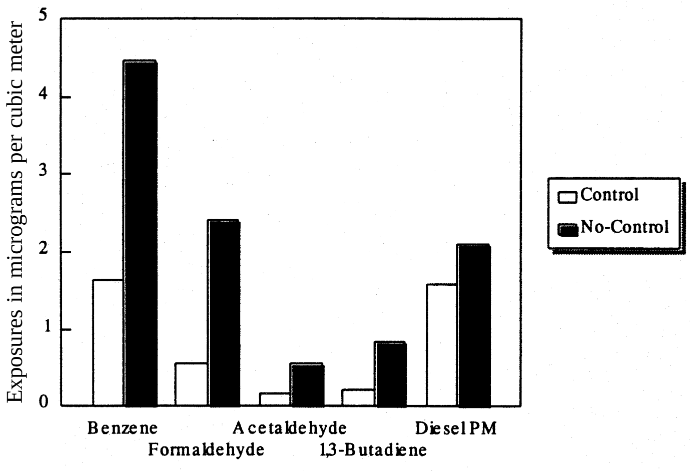
[26] Background CO is produced by the oxidation of biogenic hydrocarbons. See ICF/SAI, p. 7.

[27] The EPA MVATS attributed all measured CO to motor vehicles, resulting in an overestimation of motor-vehicle contributions to CO concentration changes. See ICF/SAI, p. 8. The MVATS assumption would also lead to a subsequent overestimation of changes in HAP exposures.

[28] See ICF/SAI, p. 3.

[29] The same HAP emission fractions used in the EPA MVATS were used herein, except for diesel PM which is not proportional to VOC emissions. Instead, diesel PM emission factors were developed using year-specific PART5 diesel PM emission factors and VMT estimates for diesel-powered vehicles.

Figure H-4. National Annual Average Motor Vehicle HAP Exposures (µg/m3).



Non-Cancer Health Effects

Broad gaps exist in the current state of knowledge about the quantifiable effects of air toxics exposure. This is particularly true for a wide range of health effects such as tumors, abnormal development, birth defects, neurological impairment, or reproductive impairment, etc. For example, the EPA's Non-Cancer Study[30] found that ambient concentrations for a substantial number of monitored and modeled HAPs exceeded one or more health benchmarks.[31] However no accepted methodology exists to quantify the effects of such exceedences. More data on health effects is needed for a broad range of chemicals.

Ecological Effects

Through the 1970s and 1980s, the adverse effects of toxic pollution on the Great Lakes became clear and undeniable. Over the same time period, scientists began collecting a convincing body of evidence that toxic chemicals released to the air can travel long distances and be deposited on land or water far from the original sources. An example of this evidence is the presence of such contaminants as PCBs, toxaphene, and other pesticides in fish in Lake Siskiwit, a lake on an island on upper Lake Superior, which has no waterborne sources of pollution. Toxaphene, a pesticide used primarily in the southeastern U.S. cotton belt, has been found as far away as the Arctic, with a decreasing air concentration gradient from the southeast toward the Great Lakes and the north Atlantic regions.

Similarly, a growing body of evidence showed that pollutants that were persistent (do not easily break down) and bioaccumulating (not significantly eliminated from the body) were magnifying up the food chain, such that top predator fish contained levels up to millions of times greater than the harmless levels in the water. As such, those who ate those large fish, such as humans, eagles, mink, and beluga whales could receive very high exposures to the pollutants. Wildlife were beginning to show adverse effects in the wild, that could be duplicated in the lab. In the Great Lakes, such chemicals as PCBs, mercury, dieldrin, hexachlorobenzene, Lindane, lead compounds, cadmium compounds, DDT/DDE, and others are of significant concern. In other places in the country, similar effects are being experienced, especially with mercury, which is transported primarily by air, but exposure to which is primarily through contaminated fish. It was this kind of information about DDT and toxaphene that led to their being banned in the U.S. under FIFRA.

While ecological and economical sciences are not yet sufficiently advanced to support the kind of comprehensive, quantitative evaluation of benefits needed for the present study, selected local and regional scale adverse ecological effects of HAPs, and their adverse consequences for human health and welfare, can and have been surveyed. In May 1994, the EPA issued its first "Report to Congress on Deposition of Air Pollutants to the Great Waters."[32] The Great Waters Report examined the pollutants contributing to adverse ecological effects, the potential significance of the contribution to pollutant loadings from deposition of airborne pollutants, and the potential adverse effects associated with these pollutant loadings. Key HAPs identified in the Great Waters Report include PCBs, mercury, dioxins, and other heavy metals and toxic organics.

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[30] U.S. Environmental Protection Agency, "Toxic Air Pollutants and Noncancer Risks: Screening Studies," External Review Draft, September, 1990.

[31] Relevant benchmarks include Acceptable Daily Intake (ADI), the estimate of daily exposure at which adverse health effects are unlikely; and Lowest Observed Actual Effect Level (LOAEL), which is the lowest exposure level at which significant adverse health effects are observed.

[32] USEPA/OAR/OAQPS, "Deposition of Air Pollutants to the Great Waters, First Report to Congress," EPA-453/R-93-055, May 1994.

Of particular relevance to the present assessment, the Great Waters Report demonstrated the significance of transport and transformation of HAPs through food webs, leading to increased toxicity and biomagnification. A prime example of adverse transport and transformation is mercury. Transformation from inorganic to methylated forms significantly increases the toxic effects of mercury in ecosystems. A prime example of biomagnification is PCBs. As noted in the Great Waters Report:

> *"Pollutants of concern [such as PCBs] accumulate in body tissues and magnify up the food web, with each level accumulating the toxics from its diet and passing the burden along to the animal in the next level of the food web. Top consumers in the food web, usually consumers of large fish, may accumulate chemical concentrations many millions of times greater than the concentrations present in the water...High risk groups...include breast-feeding mothers because breast-fed babies continue to accumulate [pollutants] from their mothers after birth. For example, they can have PCB levels four times higher than their mothers after six to nine months of breast feeding."*[33]

Because of the risk of significant exposure to infants and other high-risk groups, such as "sport anglers, Native Americans, and the urban poor,"[34] a substantial number of fish consumption advisories have been issued in recent years. Current fish advisories for the Great Lakes alone include widespread advisories for PCB's, chlordane, mercury and others, cautioning that nursing mothers, pregnant women, women who anticipate bearing children, female children of any age and male children age 15 and under not eat certain high-food chain fish species. It should be noted as well that 40 states have issued mercury advisories in some freshwater bodies, and nine states have issued mercury advisories for every freshwater waterbody in the state (these states are Maine, New Hampshire, Vermont, Massachusetts, New York, New Jersey, Missouri, Michigan, and Florida).

There is little evidence indicating that the CAA had much beneficial effect on air toxic deposition to water bodies. Since the early NESHAPs were based on direct inhalation, primarily cancer effects close to a plant, they did not address the issue of cumulative effects of persistent pollutants far from the source. It was for this reason that section 112(m) was included in the 1990 CAA Amendments, with requirements to study and document the atmospheric contribution of water pollutants, the adverse human health and environmental effects resulting and the sources that should be controlled to prevent adverse effects, and additionally, to promulgate regulations to prevent adverse effects.

Conclusions — Research Needs

As has been demonstrated, there are broad gaps in the current state of knowledge about the quantifiable effects of air toxics exposure for a wide range of both human health and environmental effects. The following discussion outlines areas in which further research is needed in order to adequately quantify the benefits of air toxics control.

Health Effects

- Develop health effects data on pollutants for which limited or no data currently exists. Such studies should be focused on pollutants with a relatively high probability of exposure and/ or potential adverse health effects.
- Understand mechanism of action of pollutants, for example through pharmacokinetic modeling. This will allow for a more accurate assessment of the effects of these pollutants on humans.
- Conduct research on factors that affect variations in susceptibility of human populations and determine the distribution of these factors in the U.S.
- Conduct research to better understand interactive effects of multiple pollutant exposures.
- Develop methodologies to derive alternative estimates of human cancer risk from existing upper-bound methods.
- Acquire data and develop dose-response relationships for critical noncancer effects such as developmental, neurotoxic, mutagenic, res-

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[33] EPA-453/R-93-055, May 1994, p. ix.

[34] EPA-453/R-93-055, May 1994, p. x.

piratory and other effects. In particular, design methodology to quantify effects of exposures above health benchmarks.

- Acquire data and develop methods to estimate effects from acute exposure.

Exposure Assessment

- Expand data collection efforts: pre- and post-control emissions; HAP speciation; facilities location; facility parameters (stack heights, distances from stacks to fencelines, etc.).
- Develop more comprehensive exposure models which incorporate activity patterns, indirect exposures, total body burden, ratios of time spent indoors to outdoors.
- Continue to refine uncertainty analysis methods.

Ecosystem Effects

- Reliable estimates/measures of the levels of persistent bioaccumulating toxics in different media (air, water column, soils and sediments)
- Work to correlate levels of persistent bioaccumulating toxics with exposures, biota concentrations/accumulation, and adverse effects, especially subtle effects such as wasting, behavioral effects, and developmental effects.
- Criteria for effects, such as a wildlife correlate to a RfD or dose-response curve. This work should be done to complement the mass balance efforts now being completed, which will model source emissions to water column concentrations, then design research to predict effects on living resources given those predicted levels.
- Work to determine the effects of mixtures of persistent bioaccumulating toxic pollutants, and to determine cause-effect relationships of exposures over long periods of time.
- Studies to evaluate toxic effects in less well understood terrestrial systems such as: soil organisms/invertebrates, food web effects, amphibian effects, effects on endangered species and phytotoxic effects.
- Work to improve understanding of effects of toxic air pollutants on wetland species and wetland functions.

Economic Valuation

- Develop valuation estimates for endpoints for which inadequate estimates currently exist. These valuation estimates must be consistent with the kinds of damages expected.
- Initiate broad-scope economic valuation of air toxics program using survey techniques.

Air Toxics References

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Appendix I: Valuation of Human Health and Welfare Effects of Criteria Pollutants

This appendix describes the derivations of the economic valuations for health and welfare endpoints considered in the benefits analysis. Valuation estimates were obtained from the literature and reported in dollars per case avoided for health effects, and dollars per unit of avoided damage for welfare effects. This appendix first introduces the method for monetizing improvements in health and welfare, followed by a summary of dollar estimates used to value benefits and detailed descriptions of the derivation of each estimate. These economic valuations are given both in terms of a central (point) estimate as well as a probability distribution which characterizes the uncertainty about the central estimate. All dollar values are rounded and are in 1990 dollars. Next, results of the economic benefits analysis are presented. Finally, uncertainties in valuing the benefits attributable to the CAA are explored.

Methods Used to Value Health and Welfare Effects

Willingness to pay (WTP) and willingness to accept (WTA) are the two measures commonly used to quantify the value an individual places on something, whether it is something that can be purchased in a market or not. Both WTP and WTA are measures of the amount of money such that the individual would be indifferent between having the good (or service) and having the money. Whether WTP or WTA is the appropriate measure depends largely on whether an increase or a decrease of the good is at issue. WTP is the amount of money an individual would be willing to pay to have a good (or a specific increase in the amount of the good) — i.e., the amount such that the individual would be indifferent between having the money and having the good (or having the specific increase in the good). WTA is the amount of money the individual would have to be compensated in order to be indifferent to the *loss* of the good (or a specific decrease in the amount of the good). WTP is the appropriate measure if the baseline case is that the individual does not have the good or when an increase in the amount of the good is at issue; WTA is the appropriate measure if the baseline case is that the individual has the good or when a decrease in the amount of the good is at issue. An important difference between WTP and WTA is that, in theory, WTP is limited by the individual's budget, whereas WTA is not. Nevertheless, while the underlying economic valuation literature is based on studies which elicited expressions of WTP and/or WTA, the remainder of this report refers to all valuation coefficients as WTP estimates. In some cases (e.g., stroke-related hospital admissions), neither WTA nor WTP estimates are available and WTP is approximated by cost of illness (COI) estimates, a clear underestimate of the true welfare change since important value components (e.g., pain and suffering associated with the stroke) are not reflected in the out-of-pocket costs for the hospital stay.

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 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, however, 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 days of cough avoided in a given year resulting from CAA reductions of pollutant concentrations, but the analysis does not estimate which individuals will be spared those days of coughing. The health benefits conferred on individuals by a reduction in pollution concentrations are, then, actually *reductions in the probabilities* of having to endure certain health problems. These benefits (reductions in probabilities) 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

$$\sum_{i=1}^{N} WTP_i(B_i) \qquad (1)$$

where B_i is the benefit (i.e., the reduction in probability of having to endure the health problem) conferred on the ith individual (out of a total of N) by the reduction in pollution concentrations, and $WTP_i(B_i)$ is the ith 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

$$\sum_{i=1}^{N}\sum_{j=1}^{J} WTP_i(B_{ij}) \qquad (2)$$

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

The reduction in probability 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 "WTP to avoid a statistical death," or, equivalently, "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 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

$$\sum_{i=1}^{N} \mathit{(number\ of\ units\ of\ risk\ reduction)}_i \times \mathit{(WTP\ per\ unit\ risk\ reduction)}_i \qquad (3)$$

where (number of units of risk reduction)$_i$ is the number of units of risk reduction conferred on the ith exposed individual as a result of the pollution reduction, (WTP per unit risk reduction)$_i$ is the ith individual's willingness to pay for a unit risk reduction, and N is the number of exposed individuals.

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 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. Derivation of the dollar value estimates discussed below was often limited by available information.

Valuation of Specific Health Endpoints

Valuation of Premature Mortality Avoided

As noted above, it is actually reductions in mortality risk that are 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. The "value of a premature death avoided," then, should be understood as shorthand for "the value of a *statistical* premature death avoided."

The value of a premature death avoided is based on an analysis of 26 policy-relevant value-of-life studies (see Table I-1). Five of the 26 studies are contingent valuation (CV) studies, which directly solicit WTP information from subjects; the rest are wage-risk studies, which base WTP estimates on estimates of the additional compensation demanded in the labor market for riskier jobs. Each of the 26 studies provided an estimate of the mean WTP to avoid a statistical premature death. Several plausible standard distributions were fit to the 26 estimates of mean WTP. A Weibull distribution, with a mean of $4.8 million and standard deviation of $3.24 million, provided the best fit to the 26 estimates. The central tendency estimate of the WTP to avoid a statistical premature death is the mean of this distribution, $4.8 million. The considerable uncertainty associated with this approach is discussed in detail below, in the subsection titled "The Economic Benefits Associated with Mortality," within the section titled "Uncertainties."

Table I-1. Summary of Mortality Valuation Estimates (millions of 1990 dollars).

Study	Type of Estimate	Valuation (millions 1990$)
Kneisner and Leeth (1991) (US)	Labor Market	0.6
Smith and Gilbert (1984)	Labor Market	0.7
Dillingham (1985)	Labor Market	0.9
Butler (1983)	Labor Market	1.1
Miller and Guria (1991)	Cont. Value	1.2
Moore and Viscusi (1988a)	Labor Market	2.5
Viscusi, Magat, and Huber (1991b)	Cont. Value	2.7
Gegax et al. (1985)	Cont. Value	3.3
Marin and Psacharopoulos (1982)	Labor Market	2.8
Kneisner and Leeth (1991) (Australia)	Labor Market	3.3
Gerking, de Haan, and Schulze (1988)	Cont. Value	3.4
Cousineau, Lacroix, and Girard (1988)	Labor Market	3.6
Jones-Lee (1989)	Cont. Value	3.8
Dillingham (1985)	Labor Market	3.9
Viscusi (1978, 1979)	Labor Market	4.1
R.S. Smith (1976)	Labor Market	4.6
V.K. Smith (1976)	Labor Market	4.7
Olson (1981)	Labor Market	5.2
Viscusi (1981)	Labor Market	6.5
R.S. Smith (1974)	Labor Market	7.2
Moore and Viscusi (1988a)	Labor Market	7.3
Kneisner and Leeth (1991) (Japan)	Labor Market	7.6
Herzog and Schlottman (1987)	Labor Market	9.1
Leigh and Folson (1984)	Labor Market	9.7
Leigh (1987)	Labor Market	10.4
Gaten (1988)	Labor Market	13.5

SOURCE: Viscusi, 1992

Life-years lost is a possible alternative measure of the mortality-related effect of pollution, as discussed in Appendix D. If life-years lost is the measure used, then the value of a statistical life-year lost, rather than the value of a statistical life lost would be needed. Moore and Viscusi (1988) suggest one approach for determining the value of a statistical life-year lost. They assume that the willingness to pay to save a statistical life is the value of a single year of life times the expected number of years of life remaining for an individual. They suggest that a typical respondent in a mortal risk study may have a life expectancy of an additional 35 years. Using a mean estimate of $4.8 million to save a statistical life, their approach would yield an estimate of $137,000 per life-year lost or saved. If an individual discounts future additional years using a standard discounting procedure, the value of each life-year lost must be greater than the value assuming no discounting. Using a 35 year life expectancy, a $4.8 million value of a statistical life, and a 5 percent discount rate, the implied value of each life-year lost is $293,000. The Moore and Viscusi procedure is identical to this approach, but uses a zero discount rate.

Using the value of a life-year lost and the expected number of years remaining (obtained from life expectancy tables), and assuming a given discount rate, values of age-specific premature mortality can be derived. Examples of valuations of pollution-related mortality using the life-years lost approach are given below, in the subsection titled "The Economic Benefits Associated with Mortality," within the section titled "Uncertainties."

Valuation of Hospital Admissions Avoided

In the case of hospital admissions, cost-of-illness (COI), or "costs avoided," estimates were used in lieu of WTP because of the lack of other information regarding willingness to pay to avoid illnesses that necessitate hospital admissions. For those hospital admissions which were specified to be the *initial* hospital admission (in particular, hospital admissions for coronary heart disease (CHD) events and stroke), COI estimates include, where possible, all costs of the illness, including the present discounted value of the stream of medical expenditures related to the illness, as well as the present discounted value of the stream of lost earnings related to the illness. (While an estimate of present discounted value of both medical expenditures and lost earnings was available for stroke, the best available estimate for CHD did not include lost earnings. The derivations of the COI estimates for CHD and stroke, both lead-induced effects, are discussed in Appendix G.)

In those cases for which it is unspecified whether the hospital admission is the initial one or not (that is, for all hospital admissions endpoints other than CHD and stroke), it is unclear what portion of medical expenditures and lost earnings after hospital discharge can reasonably be attributed to pollution exposure and what portion might have resulted from an individual's pre-existing condition even in the absence of a particular pollution-related hospital admission. In such cases, the COI estimates include only those costs associated with the hospital stay, including the hospital charge, the associated physician charge, and the lost earnings while in the hospital. The derivations of these costs are discussed in Abt Associates Inc., 1996.

These COI estimates are likely to substantially understate total WTP to avoid an illness that began with a hospital admission or to avoid a particular hospital admission itself. First, most of the COI estimates fall short of being full COI estimates either because of insufficient information or because of ambiguities concerning what portion of post-hospital costs should be attributed to pollution exposure. Even full COI estimates will understate total WTP, however, because they do not include the value of avoiding the pain and suffering associated with the illness for which the individual entered the hospital.

Valuation of Chronic Bronchitis Avoided

Although the severity of cases of chronic bronchitis valued in some studies approaches that of chronic obstructive pulmonary disease, to maintain consistency with the existing literature we do not treat those cases separately for the purposes of this analysis. Chronic bronchitis is one of the only morbidity endpoints that may be expected to last from the initial onset of the illness throughout the rest of the individual's life. WTP to avoid chronic bronchitis would therefore be expected to incorporate the present discounted value of a potentially long stream of costs (e.g., medical expenditures and lost earnings) associated with the illness. Two studies, Viscusi et al. (1991) and Krupnick and Cropper (1992) provide estimates of WTP to avoid a case of chronic bronchitis. The study by Viscusi et al., however, uses a sample that is larger and more representative of the general population than the study by Krupnick and Cropper (which selects people who have a relative with the disease). The valuation of chronic bronchitis in this analysis is therefore based on the distribution of WTP responses from Viscusi et al. (1991).

Both Viscusi et al. (1991) and Krupnick and Cropper (1992), however, defined a case of severe chronic bronchitis. It is unclear what proportion of the cases of chronic bronchitis predicted to be associated with exposure to pollution would turn out to be severe cases. The incidence of pollution-related chronic bronchitis was based on Abbey et al. (1993), which considered only new cases of the illness.[1] While a new case may not start out being severe, chronic bronchitis is a chronic illness which may progress in severity from onset throughout the rest of the individual's life. It is the chronic illness which is being valued, rather than the illness at onset.

The WTP to avoid a case of pollution-related chronic bronchitis (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 study, and (2) the elasticity of WTP with respect to severity. Because elasticity is a marginal concept and because it is a function of severity (as estimated from Krupnick and Cropper, 1992), WTP adjustments were made incrementally, in one percent steps. At each step, given a WTP to avoid a case of CB of severity level *sev*, the WTP to avoid a case of severity level 0.99**sev* was derived. This procedure was iterated until the desired severity level was reached and the corresponding WTP was derived. Because the elasticity of WTP with respect to severity is a function of severity, this elasticity changes at each iteration. If, for example, it is believed that a pollution-related case of CB is of average severity, that is, a 50 percent reduction in severity from the case described in the Viscusi study, then the iterative procedure would proceed until the severity level was half of what it started out to be.

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[1] It is important that only new chronic bronchitis be considered in this analysis because WTP estimates reflect lifetime expenditures and/or losses associated with this chronic illness, and incidences are predicted separately for each year during the period 1970-1990. If the total prevalence of chronic bronchitis, rather than the incidence of only new chronic bronchitis were predicted each year, valuation estimates reflecting lifetime expenditures could be repeatedly applied to the same individual for many years, resulting in a severe overestimation of the value of avoiding pollution-related chronic bronchitis.

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 study, (2) the severity level of an average pollution-related case of CB (relative to that of the case described by Viscusi), 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 $260,000, 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.

The distribution of WTP to avoid a severe case of CB was based on the distribution of WTP responses in the Viscusi 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 $1,000,000 (1990 dollars)[2]; the median response implied a WTP of about $530,000 (1990 dollars). 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 study could therefore be approximated by a discrete uniform distribution giving a probability of one ninth 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 study was assumed to be the distribution of WTPs to avoid a severe case of CB. The mean of this distribution is about $720,000 (1990 dollars).

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 with 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.

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, 1992).

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 $260,000.

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 $77,000 for a 30 year old, about $58,000 for a 40 year old, about $60,000 for a 50 year old, and about $41,000 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 $260,000 is from 3.4 times the full COI estimate (for 30 year olds) to 6.3 times the full COI estimate (for 60 year olds).

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[2] There is an indication in the Viscusi paper that the dollar values in the paper are in 1987 dollars. Under this assumption, the dollar values were converted to 1990 dollars.

Valuation of Other Morbidity Endpoints Avoided

WTP to avoid a day of specific morbidity endpoints, such as coughing or shortness of breath, has been estimated by only a small number of studies (two or three studies, for some endpoints; only one study for other endpoints). The estimates for health endpoints involving these morbidity endpoints are therefore similarly based on only a few studies. However, it is worth noting that the total benefit associated with any reduction in pollutant concentrations is determined largely by the benefit associated with the corresponding reduction in mortality risk because the dollar value associated with mortality is significantly greater than any other valuation estimate. More detailed explanations for valuation of specific morbidity endpoints is given in Table I-2.

Estimates of WTP may be understated for a couple of reasons. First, if exposure to pollution has any cumulative or lagged effects, then a given reduction in pollution concentrations in one year may confer benefits not only in that year but in future years as well. Benefits achieved in later years are not included. Second, the possible effects of altruism are not considered in any of the economic value derivations. Individuals' WTP for reductions in health risks for others are implicitly assumed to be zero.

Table I-2 summarizes the derivations of the economic values used in the analysis. More detailed descriptions of the derivations of lead-related endpoints (hospital admissions for CHD and stroke, Lost IQ points, IQ below 70, and hypertension) are discussed in Appendix G.

Valuation of Welfare Effects

With the exception of agricultural benefits, economic valuations for welfare effects quantified in the analysis (i.e., household soiling damage, visibility and worker productivity) are documented in Table I-2. For agricultural benefits, estimated changes in crop yields were evaluated with an agricultural sector model, AGSIM. This model incorporates agricultural price, farm policy, and other data for each year. Based on expected yields, the model estimates the production levels for each crop, the economic benefits to consumers, and the economic benefits to producers associated with these production levels. To the extent that alternative exposure-response relationships were available, a range of potential benefits was calculated. Appendix F documents the derivation of the monetary benefits associated with improved agricultural production. The derivation of the residential visibility valuation estimate is discussed further below.

Visibility Valuation

Residential visibility has historically been valued through the use of contingent valuation studies, which employ surveys and questionnaires to determine the economic value respondents place on specified changes in visibility. A number of such studies have been published in the economics literature since the late 1970s, and have reported a wide range of resulting values for visibility, expressed as household willingness to pay (WTP) for a hypothesized improvement in visibility. Those studies were carefully reviewed for their applicability to the retrospective analysis.

One limitation of many existing contingent valuation studies of visibility is that they are local or regional in scope, soliciting values for visibility from residents of only one or two cities in a single region of the country. Studies of visibility values from western cities, the most recent of which was published in 1981, have reported somewhat lower values than those from eastern cities, raising the question of whether eastern and western visibility are different commodities and should be valued differently in this analysis.

While the different visibility values reported in the literature may appear to imply that visibility is not valued equally by survey respondents in the eastern and western U.S., other evidence suggests that eastern and western visibility are not fundamentally different commodities, and that the retrospective benefits calculations should not be based on separate eastern and western visibility values. For example, NAPAP data indicate that California's South Coast Air Basin, which encompasses Los Angeles and extends northward to the vicinity of San Francisco, has median baseline visibility more characteristic of the eastern U.S. than of other areas of the west (NAPAP 1991; IEc 1992, 1993a), reflecting the influence of the higher humidity typical of coastal areas. While inland areas of the west will tend to have lower humidity, and hence greater baseline visibility than either the eastern region or the western coastal zone, such baseline visibility differences are accounted for in the conversion from the visual range metric to DeciView.

Perhaps the most compelling rationale for employing a single nationwide visibility valuation strategy in the retrospective benefits analysis, however, relates to the air quality modeling output used to calculate the control and no-control scenario visibility profiles, and its implications for the valuation of visibility as a commodity. The RADM model and linear scaling technique used for the retrospective analysis model visibility improvements nationwide as changes in regional atmospheric haze. In other words, although the magnitude of visibility effects may vary between regions, the model output does not distinguish between a change in eastern visibility and a change in western visibility as distinct phenomena. Thus, there is no clear reason to value those same visibility changes differently in calculating the benefits of visibility improvements. Consequently, a single, consistent valuation basis has been applied to residential visibility improvements nationwide for this analysis.

In light of advances in the state of the art of contingent valuation over the last decade, the age of many of the existing studies raised questions regarding their suitability to serve as the primary basis for the visibility benefits estimates. A review of the survey and data analysis methods used in the available studies indicated that a study conducted for EPA by McClelland et al. (1991) addressed many of the methodological flaws of earlier studies, employing survey methods and analytical techniques designed to minimize potential biases (IEc 1992). Although this study is unpublished, given its methodological improvements over earlier studies it was chosen as the basis for the central tendency of the visibility benefits estimate, yielding an estimated value of $14 per unit improvement in DeciView as the annual household WTP for visibility improvements (IEc 1997), as specified in Table I-2.

The difficulty of accurately defining the expected statistical distribution of WTP values for visibility improvements on the basis of published studies of uneven reliability, along with the considerable variation in reported visibility values, led to the selection of a hypothesized triangular distribution of values to characterize the uncertainty in the visibility benefits estimate. Reliance on any single study to estimate the uncertainty range would be unlikely to adequately characterize variations in visibility values that might exist across cities, and in any case would fail to capture the full variability of visibility values reported in the literature. Therefore, to ensure that the retrospective study characterizes the full range of uncertainty in visibility values nationwide, the upper and lower bounds of the triangular distribution were derived by combining results from appropriate eastern and western residential visibility valuation studies.

Most of the existing residential visibility valuation studies were found to suffer from part-whole bias, which results from the failure to differentiate values for visibility from those for other air quality amenities, such as reductions in adverse health effects. Of the studies reviewed for this analysis, only the McClelland study and Brookshire et al. (1979) have attempted to obtain bids explicitly for visibility improvements (IEc 1992). Since part-whole bias will tend to produce overstated values for visibility, reported values from all studies that do not correct for part-whole bias were adjusted prior to calculating the lower bound of the uncertainty range. The upper bound of the uncertainty range was calculated using the unadjusted values from all studies, which is equivalent to assuming that the entire value of respondents' stated WTP for improved air quality can be attributed to increased visibility.

The uncertainty range specified in Table I-2 calculated using a consensus function derived from a regression analysis, incorporates a 25 percent adjustment for part-whole bias (i.e., reported values were multiplied by 0.25) in calculating the lower bound. This represents an approximate midpoint of the range defined by the McClelland study's finding that respondents allocated, on average, 18.6 percent of their total WTP to improvements in visibility, and Chestnut and Rowe's (1989) conclusion that visibility improvement accounted for 34 percent of the total WTP reported in the Brookshire et al. study. Similarly, the "Denver Brown Cloud" study results indicate that respondents allocated 27.2 percent of their total WTP to visibility improvements (Irwin et al. 1990). Therefore, the application of a 25 percent adjustment for part-whole bias to all but the McClelland and Brookshire values would appear to be supported by the recent literature, with the resulting consensus value representing a plausible lower bound for the uncertainty range of visibility values. The consensus function approach, incorporating the part-whole bias adjustment, yields estimated upper and lower bound values of $21 and $8, respectively, for annual household WTP per unit improvement in DeciView.

Table I-2. Unit Values for Economically Valuing Health and Welfare Endpoints.

Health or Welfare Endpoint	Estimated Value Per Incidence (1990$)		Derivation of Estimates
	Central Estimate	Uncertainty Distribution	
Mortality	$4.8 million per statistical life	Weibull distribution, mean = $4.8 million std. dev. = $3.24 million	Central Est: $ value is the mean of value-of-statistical-life estimates from 26 studies (5 contingent valuation and 21 labor market studies). Uncertainty: Best-fit distribution to the 26 sample means. The Weibull distribution prevents selection of negative WTP values.
	$293,000 per statistical life-year	Weibull distribution, mean = $293,000 std. dev. = $198,000	Central Est: $ value is the mean of the distribution of the value of a statistical life-year, derived from the distribution of the value of a statistical life (see below). Uncertainty: Assuming the discount rate is five percent, and assuming an expected 35 yrs. remaining to the avg. worker in the wage-risk studies (see above), the value of a statistical life-year is just a constant, 0.061, multiplied by the value of a statistical life. The distribution of the value of a life-year is derived from the distribution of the value of a statistical life. Given that this is a Weibull distribution, as indicated above, the value of a statistical life-year is also a Weibull distribution, with mean equal to 0.061 multiplied by the mean of the original Weibull distribution (0.061x$4.8 million = $293,000) and standard deviation equal to 0.061 multiplied by the standard deviation of the original distribution (0.061 x $3.24 = $198,000). (If the discount rate were considered to also be uncertain, then the distribution of a statistical life-year would depend on this distribution as well and would have to be generated by Monte Carlo methods.)

Health or Welfare Endpoint	Estimated Value Per Incidence (1990$)		Derivation of Estimates
	Central Estimate	Uncertainty Distribution	
Chronic Bronchitis (CB)	$260,000	A Monte Carlo-generated distribution, based on three underlying distributions, as described more fully under "Derivation of Estimates" and in the text.	Central Est: $ value is the mean of a Monte Carlo distribution of WTP to avoid a case of pollution-related CB. WTP to avoid a case of pollution-related CB is derived by adjusting WTP to avoid a severe case of CB (as described in Viscusi et al., 1991) for the difference in severity and taking into account the elasticity of WTP with respect to severity of CB. The mean of the resulting distribution is $260,000. Uncertainty: The distribution of WTP to avoid a case of pollution-related CB was generated by Monte Carlo methods, drawing from each of three distributions: (1) WTP to avoid a severe case of CB is assigned a 1/9 probability of being each of the first nine deciles of the distribution of WTP responses in Viscusi et al., 1991; (2) the severity of a pollution-related case of CB (relative to the case described in the Viscusi study) is assumed to have a triangular distribution, centered at severity level 6.5 with endpoints at 1.0 and 12.0 (see text for further explanation); and (3) the constant in the elasticity of WTP with respect to severity is normally distribution with mean = 0.18 and standard deviation = 0.0669 (from Krupnick and Cropper, 1992). See text for further explanation.
IQ Changes			
1. Lost IQ Points	$3,000 per lost IQ point	none available	Central Est: $ value is the mean of estimates based on results of 2 studies. With an assumed 5% discount rate, the results in Schwartz (1994) yield an estimate of $2,500 per IQ point; the results of Salkever (1995) yield an estimate of $3,400. These estimates include the combined effects on lifetime earnings: (1) *directly* based on IQ decrement, and (2) *indirectly* based on lower educational attainment and reduced labor force participation (subtracting from indirect benefits the costs of additional education and associated opportunity cost).
2. Incidence of IQ < 70	$42,000	none available	Central Est: $ value measures reduction in education costs in terms of special needs for lower IQ students (in mainstream schools).

Health or Welfare Endpoint	Estimated Value Per Incidence (1990$)		Derivation of Estimates
	Central Estimate	Uncertainty Distribution	
Hypertension	$680 per case per year	none available	Central Est: $ value quantifies costs associated with physician care, medications, and hospital charges, in addition to opportunity cost of lost work time due to the disability.
Hospital Admissions			
1. Strokes - initial cerebrovascular accidents (ICD code 436) - initial atherothrombotic brain infarctions (ICD code 434)	$200,000 for males; $150,000 for females	none available	Central Est: $ values for males and females are based on age- and gender-specific estimates of lifetime cost of stroke from Taylor et al., 1996. Estimates include both direct costs (medical expenditures) and indirect costs (reduced productivity) and assume a five percent discount rate. Uncertainty: Although there is uncertainty surrounding the central estimates presented, there is insufficient information to characterize this uncertainty.
2. Coronary Heart Disease (CHD)	$52,000	A Monte Carlo-generated distribution, based on the uncertainty about what proportion of pollution-related CHD events is acute myocardial infarction, what proportion is angina pectoris, and what proportion is unstable angina pectoris (see "Derivation of Estimates").	Central Est: $ value is the mean of the Monte Carlo-generated distribution of WTP to avoid a pollution-related case of CHD, described below. Uncertainty: The distribution was based on the estimates of the total medical costs within 5 years of diagnosis of each of three types of CHD events examined in the Framingham Study, including acute myocardial infarction, angina pectoris, and unstable angina pectoris (Wittels et al., 1990). It is unknown what proportion of pollution-related CHD events are of each type. On each iteration, three proportions were drawn from three continuous uniform distributions, such that the three proportions summed to 1.0. The $ value for an iteration is the weighted average of the $ values for the three types of CHD event (from Wittels et al., 1990), weighted by the corresponding proportions selected.

Health or Welfare Endpoint	Estimated Value Per Incidence (1990$)		Derivation of Estimates
	Central Estimate	Uncertainty Distribution	
3. "Respiratory Illness"	$6,100	Normal distribution, mean = $6,100 std. dev. = $55	Central Est: $ value combines a cost-of-illness estimate, including the hospital charge, based on patients of all ages, and the cost of associated physician care, with the opportunity cost of time spent in the hospital. Source of hospital charge estimate: Elixhauser et al., 1993. Source of physician charge estimates: Abt Associates Inc., 1992. Uncertainty: variation about the central estimate based on the standard error reported for the hospital charge component (greater than the other two components by an order of magnitude).
4. COPD (ICD codes 490-496)	$8,100	Normal distribution, with mean = $8,100 std. dev. = $190	Central Est: $ value combines a cost-of-illness estimate, including the hospital charge, based on patients 65 and older, and the cost of associated physician care, with the opportunity cost of time spent in the hospital. Source of cost-of-illness estimates: Abt Associates Inc., 1992. Uncertainty: variation about the central estimate derived from a standard error estimated for the hospital charge component measured by another study (Elixhauser et al., 1993). The reported standard error for hospital charge was applied to the combined cost-of-illness and opportunity cost estimate by assuming that relative variabilities surrounding the respective means were similar (i.e., coefficients of variation are equal). The hospital charge represents the vast majority of the total value to avoid a hospital admission for COPD.
5. Pneumonia (ICD codes 480-487)	$7,900	Normal distribution, with mean = $7,900 std. dev. = $110	Central Est: $ value combines a cost-of-illness estimate, including the hospital charge, based on patients of all ages, and the cost of associated physician care, with the opportunity cost of time spent in the hospital. Source of hospital charge estimate: Elixhauser et al., 1993. Source of physician charge estimates: Abt Associates Inc., 1992. Uncertainty: Applied the standard error associated with the hospital charge component to the central estimate of $7,900. The hospital charge represents the vast majority of the total value to avoid a hospital admission for pneumonia.

Health or Welfare Endpoint	Estimated Value Per Incidence (1990$)		Derivation of Estimates
	Central Estimate	Uncertainty Distribution	
6. Congestive Heart Failure (ICD code 428)	$8,300	Normal distribution, with mean = $8,300 std. dev. = $120	Central Est: $ value combines a cost-of-illness estimate, including the hospital charge, based on patients of all ages, and the cost of associated physician care, with the opportunity cost of time spent in the hospital. Source of hospital charge estimate: Elixhauser et al., 1993. Source of physician charge estimates: Abt Associates Inc., 1992. Uncertainty: Applied the standard error associated with the hospital charge component to the central estimate of $8,300. The hospital charge represents the vast majority of the total value to avoid a hospital admission for congestive heart failure.
7. Ischemic Heart Disease (ICD codes 410-414)	$10,300	Normal distribution, with mean = $10,300 std. dev. = $88	Central Est: $ value combines a cost-of-illness estimate, including the hospital charge, based on patients of all ages, and the cost of associated physician care, with the opportunity cost of time spent in the hospital. Source of hospital charge estimate: Elixhauser et al., 1993. Source of physician charge estimates: Abt Associates Inc., 1992. Uncertainty: Applied the standard error associated with the hospital charge component to the central estimate of $10,300. The hospital charge represents the vast majority of the total value to avoid a hospital admission for ischemic heart disease.
Respiratory Ailments Not Requiring Hospitalization			
1. Upper Resp. Symptoms (URS) (defined as one or more of the following: runny or stuffy nose, wet cough, burning, aching, or red eyes)	$19	Continuous uniform distribution over the interval [$7, $33]	Central Est: Combinations of the 3 symptoms for which WTP estimates are available that closely match those listed by Pope et al. result in 7 different "symptom clusters," each describing a "type" of URS. A $ value was derived for each type of URS, using IEc mid-range estimates of WTP to avoid each symptom in the cluster and assuming additivity of WTPs. The $ value for URS is the average of the $ values for the 7 different types of URS. Uncertainty: taken to be a continuous uniform distribution across the range of values described by the 7 URS types.

Health or Welfare Endpoint	Estimated Value Per Incidence (1990$)		Derivation of Estimates
	Central Estimate	Uncertainty Distribution	
2. Lower Resp. Symptoms (LRS) (defined in the study as two or more of the following: cough, chest pain, phlegm, and wheeze.)	$12	Continuous uniform distribution over the interval [$5, $19]	Central Est: Combinations of the 4 symptoms for which WTP estimates are available that closely match those listed by Schwartz et al. result in 11 different "symptom clusters," each describing a "type" of LRS. A $ value was derived for each type of LRS, using IEc mid-range estimates of WTP to avoid each symptom in the cluster and assuming additivity of WTPs. The $ value for LRS is the average of the $ values for the 11 different types of LRS. Uncertainty: taken to be a continuous uniform distribution across the range of values described by the 11 LRS types.
3. Acute Bronchitis	$45	Continuous uniform distribution over the interval [$13, $77]	Central Est: Average of low and high values recommended by IEC for use in section 812 analysis (Neumann et al., 1994). Uncertainty: continuous distribution between low and high values (Neumann et al., 1994) assigns equal likelihood of occurrence of any value within the range.
4. Acute Respiratory Symptoms and Illnesses - Presence of any of 19 acute respiratory symptoms - Any Resp. Symptom - Increase in Resp. Illness	$18	1. URS, probability = 40% LRS, probability = 40% URS+LRS, prob. = 20% 2. If URS, use URS $ dist. If LRS, use LRS $ dist. If URS+LRS, randomly select one value each from URS and LRS $ distributions; sum the two	Central Est: Assuming that respiratory illness and symptoms can be characterized as some combination of URS and LRS, namely: URS with 40% probability, LRS with 40% probability, and both URS and LRS with 20% probability. The $ value for these endpoints is the weighted average (using the weights 0.40, 0.40, and 0.20) of the $ values derived for URS, LRS, and URS + LRS. Uncertainty: based on variability assumed for central estimate, and URS and LRS uncertainty distributions presented previously.

Health or Welfare Endpoint	Estimated Value Per Incidence (1990$)		Derivation of Estimates
	Central Estimate	Uncertainty Distribution	
5. Asthma - Acute	$32	Continuous uniform distribution over the interval [$12, $54]	Central Est: Mean of average WTP estimates for the four severity definitions of a "bad asthma day." Source: Rowe and Chestnut (1986), a study which surveyed asthmatics to estimate WTP for avoidance of a "bad asthma day," as defined by the subjects. Uncertainty: based on the range of values estimated for each of the four severity definitions.
6. Shortness of breath	$5.30	Continuous uniform distribution over the interval [$0, $10.60]	Central Est: From Ostro et al., 1995. This is the mean of the median estimates from two studies of WTP to avoid a day of shortness of breath: Dickie et al., 1991 ($0.00), and Loehman et al., 1979 ($10.60). Uncertainty: taken to be a continuous uniform distribution across the range of values obtained from the two studies.
Restricted Activity and Work Loss Days			
1. WLDs	$83	none available	Central Est: Median weekly wage for 1990 divided by 5 (U.S. Department of Commerce, 1992) Uncertainty: Insufficient information to derive an uncertainty estimate.
2. RADs	not monetized[a]	--	--
3. MRADs	$38	triangular distribution centered at $38 on the interval [$16, $61]	Central Est: Median WTP estimate to avoid 1 MRRAD -- minor respiratory restricted activity day -- from Tolley et al. (1986) (recommended by IEc as the mid-range estimate). Uncertainty: range is based on assumption that value should exceed WTP for a single mild symptom (the highest estimate for a single symptom--for eye irritation--is $16.00) and be less than that for a WLD. The triangular distribution acknowledges that the actual value is likely to be closer to the point estimate than either extreme.
4. RRADs	not monetized[a]	--	--

Health or Welfare Endpoint	Estimated Value Per Incidence (1990$)		Derivation of Estimates
	Central Estimate	Uncertainty Distribution	
Welfare Effects			
Household Soiling Damage	\$2.50 per household per $\mu g/m^3$ PM_{10} (annual cost)	Beta distribution with mean=\$2.50, standard deviation=\$1 on the interval [\$1.30, \$10.00]. The shape parameters of this distribution are α=1.2 and β=7.3.	Central Est: Source: ESEERCO (1994). ESEERCO uses \$1.26 as its low estimate of annual cost of soiling and materials damage per household (assuming 2.63 persons per household), taken from Manuel et al. (1982). The Manuel study measured particulate matter as TSP rather than PM-10. Hypothesizing that at least half of the costs of household cleaning are for the time value of do-it-yourselfers, which was not included in the Manuel estimate, ESEERCO multiplied the Manuel estimate by 2 to get a point estimate of about \$2.50, in 1990 \$ (reported by ESEERCO as \$2.70 in 1992 dollars). Uncertainty: The Beta distribution selected is a smooth, continuous function with its probability mass near the mean and it covers the range of reported estimates.
Visibility	Annual household WTP = \$14 per unit decrease in DeciView (decrease in DeciView corresponds to increase in visibility)	Triangular distribution centered at \$14 on the interval [\$8, \$21]	Central Est: Source: IEc 1997. Calculated by dividing the household WTP reported in the McClelland et al. study (1991) by the corresponding hypothesized change in DeciView. Uncertainty: Source: IEc 1997. Calculated by regressing reported household WTP values on the corresponding change in DeciView (converted from reported visual range changes) for all relevant city-scenario combinations posed to respondents in the original studies. The uncertainty range reflects the 25 percent adjustment for part-whole bias applied to reported values prior to calculating the lower bound.
Worker Productivity	change in daily wages: \$1 per worker per 10% change in O_3	none available	Central Est: Based on the elasticity of income with respect to O_3 concentration derived from study of California citrus workers (Crocker and Horst, 1981 and U.S. EPA, 1994). Elasticity applied to the average daily income for workers engaged in strenuous outdoor labor, \$73 (U.S. 1990 Census).

NOTES:

[a] This endpoint was not monetized because including it in the aggregation of economic benefits would result in double-counting (overlap with WLDs and MRADs).

Results of Valuation of Health and Welfare Effects

Table I-3 presents the results of combining the economic valuations described in this Appendix with the health and welfare effects results presented in Appendix D. As noted in Appendix D, there are alternative estimates for some health and welfare impacts, which form the basis of several alternative benefit estimates. Each of the health effects estimates also has quantified statistical uncertainty. The range of estimated health and welfare effects, along with the uncertain economic unit valuations, were combined to estimate a range of possible results. The combining of the health and economic information used the Monte Carlo method presented in Chapter 7. Table I-3 shows the mean estimate results, as well as the measured credible range (upper and lower five percentiles of the results distribution), of economic benefits for each of the quantified health and welfare categories.

The results for aggregate monetized benefits were also calculated using a Monte Carlo method. The results of the Monte Carlo simulations for the economic values for each of the major endpoint categories are presented in Table I-4. Note that for the upper and lower fifth percentiles the sum of the estimated benefits from the individual endpoints does not equal the estimated total. The Monte Carlo method used in the analysis assumes that each health and welfare endpoint is independent of the others. There is a very low probability that the aggregate benefits will equal the sum of the fifth percentile benefits from each of the ten endpoints.

Table I-5 shows the estimated total benefits ranges for the four modeled target years of this study: 1975, 1980, 1985, and 1990. The results of the Monte Carlo simulations of the aggregate economic benefits for these four target years are depicted in Figure I-1.

Table I-6 examines the impact of limiting the scope of the analysis to locations with more certain air quality estimates. The main analysis (as shown in Tables I-3 through I-5) covers almost the entire population of the 48 States.[3] However, the air quality information is less certain for locations far from a monitor. Table I-6 presents the results of limiting the analysis to people living within 50 km of an ozone, NO_2, SO_2, or CO monitor, or in counties with a PM monitor. The availability of monitors changes over time. Hence the proportion of the population included in this analysis changes over time as well. Table I-6 indicates that approximately a quarter of the total benefits estimated in the main analysis comes from areas with less certain air quality information.

The results of the "all U.S. population" analysis provides a more accurate depiction of the *pattern* of economic benefits across years. The accuracy of the *scale* of incidence is less certain. These results provide a better characterization of the total direct benefits of the Clean Air Act in the lower 48 states than do the "monitored area only" results because the latter completely omits historical air quality improvements for about 25 percent of the population. However, the "all U.S. population" results rely on uncertain extrapolations of pollution concentrations, and subsequent exposures, from distant monitoring sites to provide coverage for the 25 percent or so of the population living far from air quality monitors. Thus, the main results presented in Tables I-3 through I-5 include important uncertainties.

Uncertainties

The uncertainty ranges for the results on the present value of the aggregate measured monetary benefits reported in Table I-3 reflect two important sources of measured uncertainty:

- uncertainty about the avoided incidence of health and welfare effects deriving from the concentration-response functions, including both selection of scientific studies and statistical uncertainty from the original studies; and
- uncertainty about the economic value of each quantified health and welfare effect.

These aggregate uncertainty results incorporate many decisions about analytical procedures and specific assumptions discussed in the Appendices to this report.

In order to provide a more complete understanding of the economic benefit results in Table I-3, sensitivity analyses examine several additional important aspects of the main analysis. First, this section ex-

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[3] Except for lead, two to five percent (depending on pollutant) of the population who live in sparsely populated areas are excluded from the main analysis to maximize computer efficiency. All of the population of the 48 states is included in the lead analysis.

Table I-3. Criteria Pollutants Health and Welfare Benefits -- Extrapolated to Entire 48 State Population Present Value (in 1990 using 5% discount rate) of Benefits from 1970 - 1990 (in billions of 1990 dollars).

		Present Value (billions of 1990$)		
Endpoint	**Pollutant(s)**	**5th %ile**	**Mean**	**95th %ile**
Mortality				
Mortality (long-term PM-10 exposure)	PM	$2,369	$16,632	$40,597
Mortality (Lead exposure)	Lead	$121	$1,339	$3,910
Chronic Bronchitis	PM	$409	$3,313	$10,401
Other Lead-induced Ailments				
Lost IQ Points	Lead	$248	$377	$528
IQ < 70	Lead	$15	$22	$29
Hypertension	Lead	$77	$98	$120
Coronary Heart Disease	Lead	$0	$13	$40
Atherothrombotic brain infarction	Lead	$1	$10	$30
Initial cerebrovascular accident	Lead	$2	$16	$45
Hospital Admissions				
*All Respiratory	PM & O3	$8	$9	$11
*COPD + Pneumonia	PM & O3	$8	$9	$10
Ischemic Heart Disease	PM	$1	$4	$6
Congestive Heart Failure	PM & CO	$3	$5	$7
Other Respiratory-Related Ailments				
Children				
Shortness of breath, days	PM	$0	$6	$17
**Acute Bronchitis	PM	$0	$7	$18
**Upper & Lower Respiratory Symptoms	PM	$1	$2	$4
Adults				
Any of 19 Acute Symptoms	PM & O3	$4	$46	$117
All				
Asthma Attacks	PM & O3	$0	$0	$1
Increase in Respiratory Illness	NO2	$1	$2	$4
Any Symptom	SO2	$0	$0	$0
Restricted Activity and Work Loss Days				
MRAD	PM & O3	$50	$85	$123
Work Loss Days (WLD)	PM	$30	$34	$39
Human Welfare				
Household Soiling Damage	PM	$6	$74	$192
Visibility - Eastern U.S.	particulates	$38	$54	$71
Decreased Worker Productivity	O3	$3	$3	$3
Agriculture (Net Surplus)	O3	$11	$23	$35

To avoid double-counting of benefits, the following endpoints were treated as alternatives:

*Hospital admissions for COPD combined with those for pneumonia are treated as an equally-weighted alternative to hospital admissions for all respiratory illnesses.

**The definitions of acute bronchitis and upper and lower respiratory illness overlap; both studies count trouble breathing, dry cough, and wheezing in their estimates. These two studies are treated as alternatives, which reflects the variability of pollution-induced respiratory effects in children.

Table I-4. Present Value of 1970 to 1990 Monetized Benefits by Endpoint Category for 48 State Population (billions of $1990, discounted to 1990 at 5 percent).

		Present Value		
Endpoint	**Pollutant(s)**	**5th %ile**	**Mean**	**95th %ile**
Mortality	PM	$2,369	$16,632	$40,597
Mortality	Pb	$121	$1,339	$3,910
Chronic Obstructive Pulmonary Disease	PM	$409	$3,313	$10,401
IQ (Lost IQ Pts. + Children w/ IQ<70)	Pb	$271	$399	$551
Hypertension	Pb	$77	$98	$120
Hospital Admissions	PM, O3, Pb, & CO	$27	$57	$120
Respiratory-Related Symptoms, Restricted Activity, & Decreased Productivity	PM, O3, NO2, & SO2	$123	$182	$261
Soiling Damage	PM	$6	$74	$192
Visibility	particulates	$38	$54	$71
Agriculture (Net Surplus)	O3	$11	$23	$35

Table I-5. Monte Carlo Simulation Model Results for Target Years, Plus Present Value in 1990 Terms of Total Monetized Benefits for Entire 1970 to 1990 Period (in billions of 1990-value dollars).

Total Benefits By Year ($Billions)	1975	1980	1985	1990	Present Value (5%)
5th percentile	$87	$235	$293	$329	**$5,600**
Mean	$355	$930	$1,155	$1,248	**$22,200**
95th percentile	$799	$2,063	$2,569	$2,762	**$49,400**

Notes:

Present value reflects compounding of benefits from 1971 to 1990.

"Uncertainty Estimates" are results of Monte Carlo analysis combining economic and physical effects uncertainty (i.e., using both between- and within-study variability).

Full uncertainty analysis done only for years shown. Uncertainty estimates for intermediate years computed based on ratios of 5th to 50th percentile and 95th to 50th percentile for years shown. Ratios interpolated between years shown and applied to point estimates for intermediate years.

Figure I-1. Monte Carlo Simulation Model Results for Target Years (in billions of 1990 dollars).

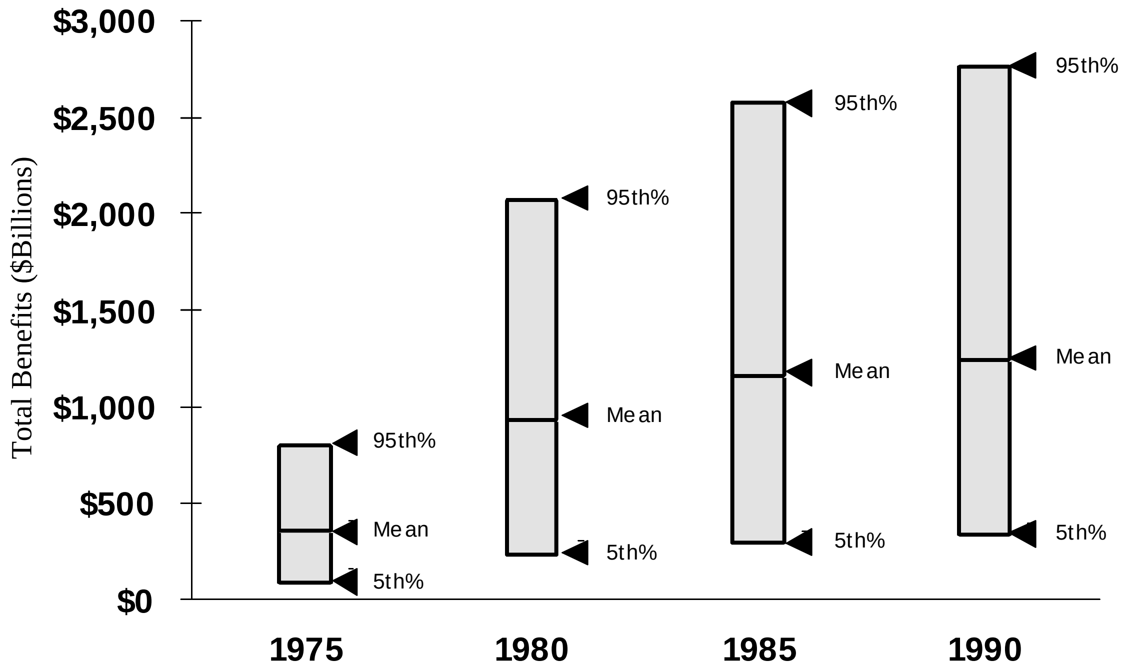

Table I-6. Comparison of 1990 (Single Year) Monetized Benefits by Endpoint for 48 State Population and Monitored Areas (in millions of 1990 dollars).

		Mean Estimate of Monetized Benefits (millions of 1990-dollars)	
Endpoint	**Pollutant(s)**	**48 State Pop.**	**Monitored Areas***
Mortality	PM	$892,390	$580,299
Mortality	Pb	$111,741	$111,741
Chronic Bronchitis	PM	$179,755	$120,053
IQ (Lost IQ Points + Children with IQ < 70)	Pb	$32,381	$32,381
Hypertension	Pb	$8,584	$8,584
Hospital Admissions	PM, O3, Pb, & CO	$4,281	$3,994
Respiratory-Related Symptoms, Restricted Activity, & Decreased Productivity	PM, O3, NO2, & SO2	$10,249	$7,089
Soiling Damage	PM	$3,964	$2,709
Visibility	particulates	$3,382	$3,382
Agriculture (Net Surplus)	O3	$986	$986
	TOTAL ($Millions)	$1,247,713	$871,218

* Monitored areas are those within 50 km of an O3, NO2, SO2, or CO monitor or a PM-monitored county. The "48 State Population" modeling estimate captures benefits for populations in unmonitored areas. Air pollution concentrations in these areas are assigned based on concentrations measured at the closest monitor, for O3, NO2, SO2, and CO. PM concentrations in unmonitored counties are derived by extrapolating those in monitored counties.

plores the effect of selecting alternative discount rates on the aggregate present value benefits estimation. Second, this section examines the sources of the measured aggregate uncertainty, identifying which of the measured uncertainty components of incidence and valuation for individual health effects categories drive the overall uncertainty results. Third, this section examines several issues involving the estimated economic benefits of mortality.

The Effect of Discount Rates

The main analysis reflected in present value results shown in Table I-3 uses a five percent discount rate. The discount rate primarily enters the calculations when compounding the economic benefits estimates from individual years between 1970 and 1990 to estimate the present value of the benefits in 1990. The discount rate also directly enters in the calculations of the economic values of an IQ point and an initial case of coronary heart disease.[4] There is considerable controversy in the economics and policy literature about the appropriate discount rate to use in different settings. Major alternatives recommended by various authors include a discount rate based on the social discount rate (typical estimates are in the 2 to 3 percent range), and a discount rate based on the risk-free rate of return on capital (typically in the 7 to 10 percent range). Table I-7 presents the aggregate uncertainty results using three different discount rates: 3 percent, 5 percent and 7 percent. While the aggregate benefits estimates are sensitive to the discount rate, selecting one of these alternative discount rates affects the aggregate benefits estimates by only about 15 percent.

The Relative Importance of Different Components of Uncertainty

The estimated uncertainty ranges in Table I-3 reflect the measured uncertainty associated with both avoided incidence and economic valuation. A better understanding of the relative influence of individual uncertain variables on the overall uncertainty in the analysis can be gained by isolating the individual effects of important variables on the range of estimated benefits. This can be accomplished by holding all the inputs to the Monte Carlo uncertainty analysis constant (at their mean values), and allowing only one variable -- for example, the economic valuation of mortality -- to vary across the range of that variable's uncertainty. The sensitivity analysis then isolates how this single source of variability contributes to the variation in estimated total benefits. The results are summarized in Figure I-2. The nine individual uncertainty factors that contribute the most to the overall uncertainty are shown in Figure I-2, ordered by the relative significance of their contribution to overall uncertainty. Each of the additional sources of quantified uncertainty in the overall analysis not shown contribute a smaller amount of uncertainty to the estimates of monetized benefits than the sources that are shown.

Table I-7. Effect of Alternative Discount Rates on Present Value of Total Monetized Benefits for 1970 to 1990 (in trillions of 1990 dollars).

Present Value in 1990 of Total Benefits (Trillions of 1990 Dollars)	3%	5%	7%
5th percentile	$4.9	$5.6	$6.5
Mean	$19.2	$22.2	$25.8
95th percentile	$42.7	$49.4	$57.5

Notes:

Present value reflects compounding of benefits from 1971 to 1990.

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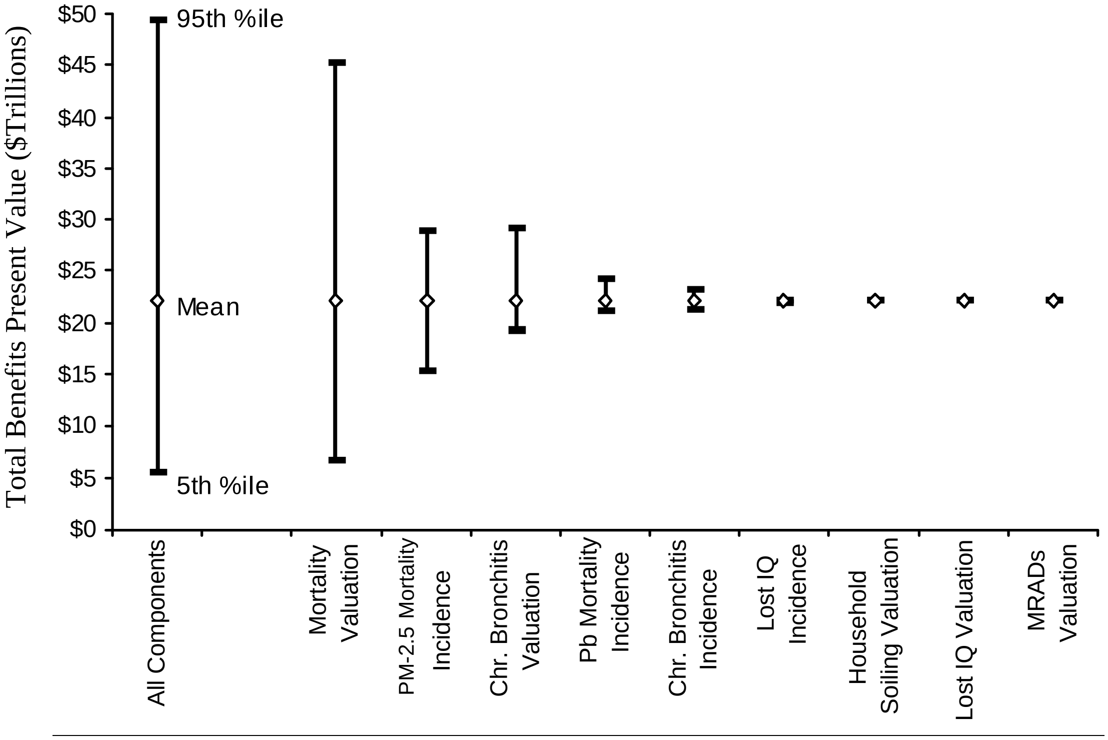
[4] The estimated economic value of lost IQ points due to lead exposure is based on the present value of the impact on lifetime earnings. A discount rate is required to calculate that present value. The impact on income primarily occurs during adulthood, which is 20 to 70 years after the initial lead exposure. This significant lag results in the discount rate having a significant impact on the estimated economic benefits of the IQ loss. Similarly, the cost of illness estimate for an initial case of CHD includes the present value of the annual stream of medical costs incurred after the event, the calculation of which requires an estimate of the discount rate.

Figure I-2. Uncertainty Ranges Deriving From Individual Uncertainty Factors.



Because of the multiple uncertainties in the benefits estimation, the total estimated present value of the monetary benefits of the 1970 to 1990 Clean Air Act range from a low of about $5.6 trillion to a high of about $49.4 trillion (in 1990 dollars, discounted at five percent). Most of the uncertainty in the total estimated benefit levels comes from uncertainty in the estimate of the economic valuation of mortality, followed by the uncertainty in the incidence of mortality from PM (as a surrogate for all non-lead air pollution). The incidence of lead-induced mortality also has a significant influence on the overall uncertainty. The importance of mortality is not surprising, because the benefits associated with reduced mortality are such a large share of overall monetized benefits.

The uncertainty in both the incidence and valuation of chronic bronchitis are the two other significant factors driving the overall uncertainty range. The modeled uncertainty in the other remaining health and welfare endpoints in the analysis contribute relatively small amounts to the overall uncertainty in the estimate of total monetary benefits of the Clean Air Act. Most of these other endpoints account for a relatively small proportion of the overall benefits estimates, making it unlikely that they could contribute significantly to the overall uncertainty. Estimates of either the mean values or standard errors of these variables are generally very small relative to estimated total monetary benefits.

Economic Benefits Associated with Reducing Premature Mortality

Because the economic benefits associated with premature mortality are the largest source of monetized benefits in the analysis, and because the uncertainties in both the incidence and value of premature mortality are the most important sources of uncertainty in the overall analysis, it is useful to examine the mortality benefits estimation in greater detail.

The analytical procedure used in the main analysis to estimate the monetary benefits of avoided premature mortality assumes that the appropriate economic value for each incidence is a value from the currently accepted range of the value of a statistical life. As discussed above, the estimated value per predicted incidence of excess premature mortality is modeled as a Weibull distribution, with a mean value of $4.8 million and a standard deviation of $3.2 million. This estimate is based on 26 studies of the value of mortal risks.

There is considerable uncertainty as to whether the 26 studies on the value of a statistical life provide adequate estimates of the value of a statistical life saved by air pollution reduction. Although there is considerable variation in the analytical designs and data used in the 26 underlying studies, the majority of the studies involve the value of risks to a middle-aged working population. Most of the studies examine differences in wages of risky occupations, using a wage-hedonic approach. Certain characteristics of both the population affected and the mortality risk facing that population are believed to affect the average willingness to pay (WTP) to reduce the risk. The appropriateness of a distribution of WTP estimates from the 26 studies for valuing the mortality-related benefits of reductions in air pollution concentrations therefore depends not only on the quality of the studies (i.e., how well they measure what they are trying to measure), but also on (1) the extent to which the subjects in the studies are similar to the population affected by changes in pollution concentrations, and (2) the extent to which the risks being valued are similar. As discussed below, there are possible sources of both upward and downward bias in the estimates provided by the 26 studies when applied to the population and risk being considered in this analysis.

If the individuals who die prematurely from air pollution are consistently older than the population in the valuation studies, the mortality valuations based on middle-aged people may provide a biased estimate of the willingness to pay of older individuals to reduce mortal risk. There is some evidence to suggest that the people who die prematurely from exposure to ambient particulate matter tend to be older than the populations in the valuation studies. In the general U.S. population far more older people die than younger people; 88 percent of the deaths are among people over 64 years old. It is difficult to establish the proportion of the pollution-related deaths that are among the older population because it is impossible to isolate individual cases where one can say with even reasonable certainty that a specific individual died because of air pollution.

There is considerable uncertainty whether older people will have a greater willingness to pay to avoid risks than younger people. There is 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. More risk averse people would have a greater willingness to pay to avoid risk than less risk averse people. Although the 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 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 pollution reduction relative to that of subjects in wage-risk studies is uncertain. In addition, the workers in the wage-risk studies will have potentially more years remaining in which to acquire streams of income from future earnings.

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

Finally, another possible difference related to the nature of the risk may be that some workplace mortality risks tend to involve sudden, catastrophic events (e.g., workplace accidents), whereas air pollution-related risks tend to involve longer periods of disease and suffering prior to death. Some evidence suggests that WTP to avoid a risk of a protracted death involving prolonged suffering and loss of dignity and personal control is greater than the WTP to avoid a risk (of identical magnitude) of sudden death. Some workplace risks, such as risks from exposure to toxic chemicals, may be more similar to pollution-related risks. It is not clear, however, what proportion of the workplace risks in the wage-risk studies were related to workplace accidents and what proportion were risks from exposure to toxic chemicals. To the extent that the mortality risks addressed in this assessment are associated with longer periods of illness or greater pain and suffering than are the risks addressed in the valuation literature, the WTP measurements employed in the present analysis would reflect a downward bias.

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. (1985), a recent sulfate-related health benefits study conducted for EPA (U.S. EPA, 1995) assumes that the value of a statistical life for those 65 and over is 75 percent of what it is for those under 65.

There is substantial evidence that the income elasticity of WTP for health risk reductions is positive (see, for example, Alberini et al., 1994; Mitchell and Carson, 1986; Loehman and Vo Hu De, 1982; Gerking et al., 1988; and Jones-Lee et al., 1985), 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 PM reductions is likely to be significantly different from that of subjects in wage-risk studies, however, is unclear, as discussed above.

The need to adjust wage-risk-based WTP estimates downward because of the likely upward bias introduced by the age discrepancy has received significant attention (see Chestnut, 1995; IEc, 1992). If the age difference were the only difference between the population affected by pollution changes and the subjects in the wage-risk studies, there might be some justification for trying to adjust the point estimate of $4.8 million downward. Even in this case, however, the degree of the adjustment would be unclear. There is good reason to suspect, however, that there are biases in both directions. Because in each case the extent of the bias is unknown, the overall direction of bias in the mortality values is similarly unknown. Adjusting the estimate upward or downward to compensate for any one source of bias could therefore increase the degree of bias. Therefore, the range of values from the 26 studies is used in the primary analysis without adjustment.

Examining the sensitivity of the overall results to the mortality values can help illuminate the potential impacts of alternative mortality valuations. As mentioned above, a contractor study performed for EPA used one approach to evaluate the economic value of sulfate-related human health improvements resulting from 1990 Clean Air Act Amendments Title IV acid rain controls. That study assumed that 85 percent of the people dying from sulfates (an important component of particulate matter) were over 65, and that people over 65 have a willingness to pay to avoid a mortal risk that is 75 percent of the values that middle-aged people have. Using this approach, the value of an average statistical life (using a weighted average) is reduced to 79 percent of the previous value.

If statistical life-years lost are used as the unit of measure, rather than statistical lives lost, the benefit attributed to avoiding a premature death depends directly on how premature it is. One way to estimate the value of a statistical life-year assumes that the value of a statistical life is directly related to remaining life expectancy and a constant value for each life-year. Such an approach results in smaller values of a statistical life for older people, who have shorter life expectancies, and larger values for younger people. For example, if the $4.8 million mean value of avoiding death for people with a 35 year life expectancy is assumed to be the discounted present value of 35 equal-valued statistical life-years, the implied value of each statistical life-year is $293,000 (using a 5% discount rate). The average number of life-years lost by individuals dying prematurely from exposure to PM is 14 years. This average is obtained by multiplying the predicted number of PM-related premature deaths in each age category by the life expectancy for that age category and dividing by the total number of PM-related premature deaths.) Using $293,000 per life-year, the discounted present value of a statistical life for a person with 14 years of expected life remaining (e.g., a 70 year old) is $2.9 million). If statistical life-years lost are used to value fatal risks, however, other sources of uncertainty are introduced in the valuation process.

If statistical life-years lost is the unit of measure, the value of a statistical life lost depends on (1) how many years of expected life are lost, (2) the individual's discount rate, and (3) whether the value of an undiscounted statistical life-year is the same no matter which life-year it is (e.g., the undiscounted value of the seventy-fifth year of life is the same as the undiscounted value of the fortieth year of life). Each of these is uncertain. The uncertainty surrounding the expected years of life lost by an individual involves the uncertainty about whether individuals who die from exposure to air pollution are average individuals in the demographic (e.g., age-gender-race) classification to which they belong. The uncertainty surrounding individuals' discount rates is well documented. Finally, even if it is assumed that all life-years are valued the same (apart from differences due to discounting), the value of a statistical life-year is derived from the value of a statistical life (of a 40 year old) and the discount rate, each of which is uncertain.

Using life-years lost as the unit of measure means that, rather than estimating a single value of a statistical life lost (applicable to all ages), the analysis would instead estimate age-specific values of statistical lives lost. It is unclear whether the variability of estimates of age-specific values of statistical lives lost (in particular, for ages greater than the average age of workers in the wage-risk studies) would be less than or greater than the variability of the original estimate of the value of a statistical life lost from which they would be derived. If there is an age-related upward bias in the central tendency value of a statistical life that is larger than any downward bias, then valuing life-years rather than lives lost may decrease the bias. Even this, however, is uncertain.

In spite of the substantial uncertainties and paucity of available information, this section presents an example of a preliminary estimate of the present value of avoided premature mortality using the life-years lost approach. The basic approach is to (1) estimate the number of pollution-related premature deaths in each age category, (2) estimate the average number of life-years lost by an individual in a given age category dying prematurely, and (3) using the value of a statistical life-year of $293,000, described above (assuming that the undiscounted value of a life-year is the same no matter when in an individual's life it is) and assuming a five percent discount rate, calculate the value of a statistical life lost in each age category.

To obtain estimates of the number of air pollution-related deaths in each age cohort, it is preferable to have age-specific relative risks. Many of the epidemiological studies, however, do not provide any estimate of such age-specific risks. In this case, the age-specific relative risks must be assumed to be identical.

Some epidemiology studies on PM do provide some estimates of relative risks specific to certain age categories. The limited information that is available suggests that relative risks of mortality associated with exposure to PM are greater for older people. Most of the available information comes from short-term exposure studies. There is considerable uncertainty in applying the evidence from short-term exposure studies to results from long-term (chronic exposure) studies. However, using the available information on the relative magnitudes of the relative risks, it is possible to form a preliminary assessment of the relative risks by different age classes.

The analysis presented below uses two alternative assumptions about age-specific risks: (1) there is a constant relative risk (obtained directly from the health literature) that is applicable to all age cohorts, and (2) the relative risks differ by age, as estimated from the available literature. Estimates of age-specific PM coefficients (and, from these, age-specific relative risks) were derived from the few age-specific PM coefficients reported in the epidemiological literature. These estimates in the literature were used to estimate the ratio of each age-specific coefficient to a coefficient for "all ages" in such a way that consistency among the age-specific coefficients is preserved — that is, that the sum of the health effects incidences in the separate, non-overlapping age categories equals the health effects incidence for "all ages." These ratios were then applied to the coefficient from Pope et al. (1995). Details of this approach are provided in Post and Deck (1996). Because Pope et al. considered only individuals age 30 and older (instead of all ages), the resulting age-specific PM coefficients may be slightly different from what they would have been if the ratios had been applied to an "all ages" coefficient. The differences, however, are likely to be minimal and well within the error bounds of this exercise. The age-specific relative risks used in the example below assume that the relative risks for people under 65 are only 16 percent of the population-wide average relative risk, the risks for people from 65 to 74 are 83 percent of the population-wide risk, and people

75 and older have a relative risk 55 percent greater than the population average. Details of this approach are provided in Post and Deck (1996).

The life-years lost approach also requires an estimate of the number of life-years lost by a person dying prematurely at each given age. The average number of life-years lost will depend not only on whether relative risks are age-specific or uniform across all age groups, but also on the distribution of ages in the population in a location. As noted above, using the same relative risk for all age categories, the average number of life-years lost in PM-related premature deaths in the United States was estimated to be 14 years. Using the age-specific relative risk estimates developed for this analysis, the average number of life-years lost becomes 9.8 years. In a location with a population that is younger than average in the United States, the same age-specific relative risks will produce a larger estimated average number of life-years lost. For example, using the same age-specific relative risks, the average number of life-years lost in PM-related premature deaths in Los Angeles County, which has a younger population, is estimated to be 15.6 years.

The present value benefits estimates for PM-related mortality using the alternative approaches discussed above are shown in Table I-8. Table I-8 is based on a single health study: Pope et al., 1995. Alternative studies, or the uncertainty approach used in the primary analysis, would result in a similar pattern of the relationship between valuation approaches. The pattern of monetized mortality benefits across the different valuation procedures shown in Table I-8 is essentially invariant to the particular relative risk and the particular dollar value used.

As noted above, the life-years lost approach used here assumes that people who die from air pollution are typical of people in their age group. The estimated value of the quantity of life lost assumes that the people who die from exposure to air pollution had an average life expectancy. However, it is possible that the people who die from air pollution are already in ill health, and that their life expectancy is less than a typical person of their age. If this is true, then the number of life years lost per PM-related death would be lower than calculated here, and the economic value would be smaller.

The extent to which adverse effects of particulate matter exposure are differentially imposed on people of advanced age and/or poor health is one of the most important current uncertainties in air pollution-related health studies. There is limited information, primarily from the short-term exposure studies, which suggests that at least some of the estimated premature mortality is imposed disproportionately on people who are elderly and/or of poor health. The Criteria Document for Particulate Matter (U.S. EPA, 1996) identifies only two studies which attempt to evaluate this disproportionality. Spix et al. (1994) suggests that a small portion of the PM-associated mortality occurs in individuals who would have died in a short time anyway. Cifuentes and Lave (1996) found that 37 to 87 percent of the deaths from short-term exposure could have been premature by only a few days, although their evidence is inconclusive.

Table I-8. Alternative Estimates of the Present Value of Mortality Associated With PM (based on Pope et al., 1996, in trillions of 1990 dollars).

Valuation Procedure	Present Value of PM Mortality Benefits
Primary Analysis Method ($4.8 million per statistical life saved)	$16.6
Life Years Lost approaches	
Single relative risk, valuation using 5% discounting	$9.1
Approximate age-specific relative risk, valuation using 5% discounting	$8.3

Notes:

Present value reflects compounding of benefits from 1971 to 1990, using a 5 percent discount rate.

Prematurity of death on the order of only a few days is likely to occur largely among individuals with pre-existing illnesses. Such individuals might be particularly susceptible to a high PM day. To the extent that the pre-existing illness is itself caused by or exacerbated by chronic exposure to elevated levels of PM, however, it would be misleading to define the prematurity of death as only a few days. In the absence of chronic exposure to elevated levels of PM, the illness would either not exist (if it was caused by the chronic exposure to elevated PM) or might be at a less advanced stage of development (if it was not caused by but exacerbated by elevated PM levels). The prematurity of death should be calculated as the difference between when the individual died in the "elevated PM" scenario and when he would have died in the "low PM" scenario. If the pre-existing illness was entirely unconnected with chronic exposure to PM in the "elevated PM" scenario, and if the individual who dies prematurely because of a peak PM day would have lived only a few more days, then the prematurity of that PM-related death is only those few days. If, however, in the absence of chronic exposure to elevated levels of PM, the individual's illness would have progressed more slowly, so that, in the absence of a particular peak PM day the individual would have lived several years longer, the prematurity of that PM-related death would be those several years.

Long-term studies provide evidence that a portion of the loss of life associated with long-term exposure is independent of the death from short-term exposures, and that the loss of life-years measured in the long-term studies could be on the order of years. If much of the premature mortality associated with PM represents short term prematurity of death imposed on people who are elderly and/or of ill health, the estimates of the monetary benefits of avoided mortality may overestimate society's total willingness to pay to avoid particulate matter-related premature mortality. On the other hand, if the premature mortality measured in the chronic exposure studies is detecting excess premature deaths which are largely independent of the deaths predicted from the short term studies, and the disproportionate effect on the elderly and/or sick is modest, the benefits measured in this report could be underestimates of the total value. At this time there is insufficient information from both the medical and economic sciences to satisfactorily resolve these issues from a theoretical/analytical standpoint. Until there is evidence from the physical and social sciences which is sufficiently compelling to encourage broad support of age-specific values for reducing premature mortality, EPA will continue to use for its primary analyses a range of values for mortality risk reduction which assumes society values reductions in pollution-related premature mortality equally regardless of who receives the benefit of such protection.

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Appendix J: Future Directions

Research Implications

In virtually any benefit analysis of environmental issues, the state of scientific information limits the degree of coverage possible and the confidence in benefit estimation. For most benefit categories, further scientific research would allow for a better quantification of benefits. One of the major outcomes of the retrospective analysis is a clear delineation of the major limitations in the scientific and economics literature in carrying out an analysis of this scope. Often, a list of research needs is generated in studies such as this, but there is no clear internal mechanism to address these needs. With this study (and the ongoing section 812 program), a process has been initiated where identified research needs are to be integrated into EPA's overall extramural research grants program, administered by the Office of Research and Development. It is hoped that the research projects that flow from this process will enable future analyses to be less uncertain and more comprehensive.

Certain of the limitations in the retrospective analysis are directly related to the historical nature of the analysis, such as sparse information about air quality in the early 1970's in many areas in the country. Other important limitations are related to the effects of elevated airborne lead concentrations, which has been virtually eliminated by the removal of lead from gasoline. A better understanding of these relationships would improve our understanding of the historical impact of the Clean Air Act, but would only indirectly contribute to developing future air pollution policy. However, most of the research that will reduce the major gaps and uncertainties needed to improve the section 812 analyses will be directly relevant to EPA's primary ongoing mission of developing and implementing sound environmental policies to meet the national goals established in the Clean Air Act and other legislation.

There are a number of biological, physical and economic research areas which the EPA Project Team identified as particularly important for improving future section 812 analyses. These research topics can be divided into two principal categories: (1) those which might reduce uncertainties in cost and benefit estimates with significant potential for influencing estimated net benefits of the Clean Air Act, and (2) those which might improve the comprehensiveness of section 812 assessments by facilitating quantification and/or monetization of currently excluded cost or benefit endpoints. The following subsections provide examples of research topics which, if pursued, might improve the certainty and/or comprehensiveness of future section 812 studies.

Research Topics to Reduce Uncertainty

Scientific information about the effects of long-term exposure to air pollutants is just beginning to emerge, but continues to be the subject of intense scientific inquiry. The relationship between chronic PM exposure and excess premature mortality included in the quantified results of the present analysis is one example of such research. However, many other potential chronic effects that are both biologically plausible and suggested by existing research are not included. Research to identify the relationship linking certain known or hypothesized physical effects (e.g., ozone's effects on lung function or fibrosis) with the development of serious health effects (e.g., cardiopulmonary diseases or premature mortality), and the appropriate economic valuation of the willingness to pay to avoid the risks of such diseases, would reduce the uncertainty caused by a major category of excluded health effects which could have a significant impact on the aggregate benefits estimates.

As described in Chapter 7 and Appendix I, premature mortality is both the largest source of benefits and the major source of quantified uncertainty in the retrospective analysis. In addition to the quantified uncertainty, there is considerable additional unquantified uncertainty about premature mortality associated with air pollution. Much of the information base about these relationships is relatively new, more is coming out virtually daily, and there is substantial disagreement in the scientific community about many of the key issues. EPA's Research Strategy and Research Needs document for particulate matter, currently under development, will address many of these scientific issues as they relate to PM. The following selection of highly uncertain issues could have a significant impact on both the aggregate mortality benefits estimates and the measured uncertainty range:

- the relationship of specific pollutants in the overall premature mortality effect, including the individual or interactive relationships between specific chemicals (e.g., ozone, sulfates, nitrates, and acid aerosols), and particle sizes (i.e., coarse, fine and ultra-fine particles);
- the degree of overlap (if any) between the measured relationships between effects associated with short term exposures and effects from long term exposure;
- the confounding effect of changes in historic air pollution, including changes over time in both pollution levels and the composition of the pollutant mix;
- the extent to which life spans are shortened by exposure to the pollutants, and the distribution of ages at the time of death;
- the willingness to pay to avoid the risks of shortened life spans; and
- the extent to which total $PM_{2.5}$ exposure incrementally augments the variability of outdoor $PM_{2.5}$ and increases the dose that would cause excess morbidity or mortality.

After premature mortality, chronic bronchitis is the next largest health effect benefit category included in the retrospective analysis. There is considerable measured uncertainty about both the incidence estimation and the economic valuation. Additional research could reduce uncertainties about the level of the pollutants associated with the observed effects, the baseline incidence used to model the changes in the number of new cases, and the correspondence between the definition of chronic bronchitis used in the health effects studies and the economic valuation studies.

Another area of potentially useful research would be further examination of the effects of criteria pollutants on cardiovascular disease incidence and mortality. Considering available epidemiological evidence and the potential economic cost of cardiovascular disease, the value of avoiding these outcomes may significantly influence the overall benefit estimates generated in future assessments.

Further research on the willingness to pay to avoid the risk of hospital admissions for specific conditions would reduce a potentially significant source of nonmeasured uncertainty. The Project Team used "avoided costs" for the value of an avoided hospital admission, based on the avoided direct medical cost of hospitalization (including lost wages for the employed portion of the hospitalized population). Avoided costs are likely to be a substantial underestimate of the appropriate willingness to pay, especially for such serious health effects as hospitalization for strokes and congestive heart failure, particularly because they omit the value of avoided pain, suffering, and inconvenience. Furthermore, in addition to hospitalization, there is evidence that some people seek medical assistance as outpatients. It is also likely that there are additional people adversely affected by short-term air pollution levels who seek physician services (but stop short of hospital admissions). Revised estimates of the appropriate economic value of avoided hospitalization and other primary care medical services could increase the total economic benefits of this cluster of health effects sufficiently that it could be a much larger portion of the aggregate benefit total.

Finally, one of the challenges in preparing the retrospective analysis was modeling the integrated relationships between emissions of many different chemicals, the subsequent mixture of pollutants in the ambient air, and the resulting health and welfare effects of simultaneous exposure to multiple pollutants. One element of the uncertainty in the analysis derives from the limited current understanding of any interactive (synergistic or antagonistic) effects of multiple pollutants. The need to better understand these complex issues is not a limited scientific question to improve section 812 analyses, but is the primary focus of EPA's current activities, organized under the Federal Advisory Council Act (FACA) process, to develop an integrated set of attainment policies dealing with ozone, particulate matter, sulfur and nitrogen oxides, and visibility. Further research on multi-pollutant issues may both (a) reduce a source of unmeasured uncertainty in the section 812 analyses and (b) allow for effective apportionment of endpoint reduction benefits to specific pollutants or pollutant mixes.

Research Topics to Improve Comprehensiveness

Even though research efforts falling in this category may not result in significant changes in net monetary benefit estimates, one of the goals of the section 812 studies is to provide comprehensive information about Clean Air Act programs. For example, programs to control hazardous air pollutants (HAPs) tend to impose costs and yield benefits which are relatively small compared to programs of pervasive national applicability such as those aimed at meeting National Ambient Air Quality Standards. Nevertheless, there are significant social, political, financial, individual human health, and specific ecosystem effects associated with emissions of HAPs and the programs implemented to control them. Under these circumstances, continued efforts to understand these consequences and evaluate their significance in relation to other programmatic and research investment opportunities might be considered reasonable, particularly in the context of comprehensive program assessments such as the present study.

Some cost and benefit effects could not be fully assessed and incorporated in the net monetary benefit estimate developed for the present study for a variety of reasons. Various effects were excluded due to (a) inadequate historical data (e.g., lack of data on historical ambient concentrations of HAPs), (b) inadequate scientific knowledge (e.g., lack of concentration-response information for ecological effects of criteria and hazardous air pollutants), or (c) resource-intensity or limited availability of analytical tools needed to assess specific endpoints (e.g., indirect effects resulting from deposition and subsequent exposure to HAPs). Other specific examples of presently omitted or underrepresented effect categories include health effects of hazardous air pollutants, ecosystem effects, any long-term impact of displaced capital on productivity slowdown, and redirected technological innovation.

Although the primary focus of 1970 to 1990 CAA programs was reduction of criteria pollutants to achieve attainment of national ambient air quality standards, emissions of air toxics were also substantially reduced. Some air toxics were deliberately controlled because of their known or suspected carcinogenicity, while other toxic emissions were reduced indirectly due to control procedures aimed at other pollutants, particularly ozone and particulate matter. The current analysis was able to present only limited information on the effects of changes in air toxic emissions. These knowledge gaps may be more serious for future section 812 analyses, however, since the upcoming prospective study will include evaluation of the effects of an expanded air toxic program under the CAA Title III. Existing knowledge gaps that prevented a more complete consideration of toxics in the present study include (a) methods to estimate changes in acute and chronic ambient exposure conditions nationwide, (b) concentration-response relationships linking exposure and health or ecological outcomes, (c) economic valuation methods for a broad array of potential serious health effects such as renal damage, reproductive effects and non-fatal cancers, and (d) potential ecological effects of air toxics.

In addition to research to improve the understanding of the consequences of changes in air pollution on human health and well-being, further research on non-health effects could further improve the comprehensiveness of future assessments. Improvements in air quality have likely resulted in improvements in the health of aquatic and terrestrial ecosystems and the myriad of ecological services they provide, but knowledge gaps prevented them from being included in the current analysis. Additional research in both scientific understanding and appropriate modeling procedures could facilitate inclusion of additional benefits such as improvements in water quality stemming from a reduction in acid deposition-related air pollutants. Water quality improvements would benefit human welfare through enhancements in certain consumptive services such as commercial and recreational fishing, in addition to non-consumptive services such as wildlife viewing, maintenance of biodiversity, and nutrient cycling. Similarly, increased growth, productivity and overall health of U.S. forests could occur from reducing ozone, resulting in benefits from increased timber production, greater opportunities for recreational services such as hunting, camping, wildlife observation, and nonuse benefits such as nutrient cycling, temporary CO_2 sequestration, and existence value. Finally, additional research using a watershed approach to examine the potential for ecological service benefits which emerge only at the watershed scale might be useful and appropriate given the broad geographic scale of the section 812 assessments.

While there are insufficient data and/or analytical resources to adequately model the short-run ecological and ecosystem effects of air pollution reduction, even less is known about the long-run effects of prolonged exposure. Permanent species displacement or altered forest composition are examples of potential ecosystem effects that are not reflected in the current monetized benefit analysis, and could be a source of additional benefits. In addition to these ecological research needs, an equally large, or larger, gap in the benefit-cost analysis is the lack of adequate tools to monetize the benefits of such ecosystem services.

Future Section 812 Analyses

This retrospective study of the benefits and costs of the Clean Air Act was developed pursuant to section 812 of the 1990 Clean Air Act Amendments. Section 812 also requires EPA to generate an ongoing series of prospective studies of the benefits and costs of the Act, to be delivered as Reports to Congress every two years.

Design of the first section 812 prospective study commenced in 1993. The EPA Project Team developed a list of key analytical design issues and a "strawman" analytical design reflecting notional decisions with respect to each of these design issues.[1] The analytical issues list and strawman design were presented to the Science Advisory Board Advisory Council on Clean Air Compliance Analysis (Council), the same SAB review group which has provided review of the retrospective study. Subsequently, the EPA Project Team developed a preliminary design for the first prospective study. Due to resource limitations, however, full-scale efforts to implement the first prospective study did not begin until 1995 when expenditures for retrospective study work began to decline as major components of that study were completed.

As for the retrospective, the first prospective study is designed to contrast two alternative scenarios; however, in the prospective study the comparison will be between a scenario which reflects full implementation of the CAAA90 and a scenario which reflects continued implementation only of those air pollution control programs and standards which were in place as of passage of the CAAA90. This means that the first prospective study will provide an estimate of the incremental benefits and costs of the CAAA90.

The first prospective study is being implemented in two phases. The first phase involves development of a screening study, and the second phase will involve a more detailed and refined analysis which will culminate in the first prospective study Report to Congress. The screening study compiles currently available information on the costs and benefits of the implementation of CAAA90 programs, and is intended to assist the Project Team in the design of the more detailed analysis by providing insights regarding the quality of available data sources and analytical models, and the relative importance of specific program areas; emitting sectors; pollutants; health, welfare, and ecological endpoints; and other important factors and variables.

In developing and implementing the retrospective study, the Project Team developed a number of important modeling systems, analytical resources, and techniques which will be directly applicable and useful for the ongoing series of section 812 Prospective Studies. Principal among these are the Criteria Air Pollutant Modeling System (CAPMS) model developed to translate air quality profile data into quantitative measures of physical outcomes; and the economic valuation models, coefficients, and approaches developed to translate those physical outcomes to economic terms.

The Project Team also learned valuable lessons regarding analytical approaches or methods which were not as productive or useful. In particular, the Project Team plans not to perform macroeconomic modeling as an integral part of the first prospective analysis. In fact, there are currently no plans to conduct a macroeconomic analysis at all. Essentially, the Project Team concluded, with confirmation by the SAB Council, that the substantial investment of time and resources necessary to perform macroeconomic modeling would be better invested in developing high quality data on the likely effects of the CAA on key emitting sectors, such as utilities, on-highway vehicles, refineries, etc. While the intended products of a macroeconomic modeling exercise – such as overall effects on productivity, aggregate employment effects, indirect economic effects– are of theoretical interest, the practical results of such exercises in the context of evaluating environmental programs may be disappointing for several reasons.

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[1] Copies of the prospective study planning briefing materials are available from EPA.

First, the CAA has certainly had a significant effect on several industrial sectors. However, the coarse structure of a model geared toward simulating effects across the entire economy requires crude and potentially inaccurate matching of these polluting sectors to macroeconomic model sectors. For example, the J/W model used for the retrospective study has only 35 sectors, with electric utilities comprising a single sector. In reality, a well-structured analysis of the broader economic effects of the CAA would provide for separate and distinct treatment of coal-fired utility plants, oil-fired plants, and so on. Furthermore, the outputs of the macroeconomic model are too aggregated to provide useful and accurate input information for the sector-specific emission models used to project the emissions consequences of CAA programs. Again, the critical flaw is the inability to project important details about differential effects on utilities burning alternative fuels.

The second critical problem with organizing a comprehensive analysis of the CAA around a macroeconomic modeling approach is that the effect information produced by the macroeconomic model is relatively unimportant with respect to answering the fundamental, target variable: "*How do the overall health, welfare, ecological, and economic benefits of Clean Air Act programs compare to the costs of these programs?"* The Project Team believes that any adverse effect, no matter how small in a global context, should not be deemed "insignificant" if even one individual is seriously harmed. However, the retrospective study results themselves have shown that, when analytical resources are limited, the resources invested in the macroeconomic modeling would have been better spent to provide a more complete and less uncertain assessment of the benefit side of the equation. Even on the cost side of the equation, it is far more important to invest in developing accurate and reliable estimates of sector-specific compliance strategies and the direct cost implications of those strategies. This will be even more true in the prospective study context when the Project Team will be faced with forecasting compliance strategies and costs rather than simply compiling survey data on actual, historical compliance expenditures.

The third and most important limitation of macroeconomic modeling analysis of environmental programs is that, unlike the economic costs of protection programs, the economic benefits are not allowed to propagate through the economy. For example, while productivity losses associated with reduced capital investment due to environmental regulation are counted, the productivity gains resulting from reduced pollution-related illness and absenteeism of workers are not counted. The resulting imbalance in the treatment of regulatory consequences raises serious concerns about the value of the macroeconomic modeling evaluation of environmental programs. In the future, macroeconomic models which address this and other concerns may be developed; however, until such time EPA is likely to have limited confidence in the value of macroeconomic analysis of even broad-scale environmental protection programs.

Based on these findings and other factors, the design of the first prospective study differs in important ways from the retrospective study design. First, rather than relying on broad-scale, hypothetical, macroeconomic model-based scenario development and analysis, the first prospective study will make greater use of existing information from EPA and other analyses which assess compliance strategies and costs, and the emission and air quality effects of those strategies. After developing as comprehensive a data set as possible of regulatory requirements, compliance strategies, compliance costs, and emissions consequences, the data set will be reviewed, refined, and extended as feasible and appropriate. In particular, a number of in-depth sector studies will be conducted to develop up-to-date, detailed projections of the effects of new CAA requirements on key emitting sectors. Candidate sectors for in-depth review include, among others, utilities, refineries, and on-highway vehicles.

The first prospective study will also differ from the retrospective study in that analytical resources will be directed toward development of a more complete assessment of benefits. Efforts will be made to address the deficiencies which prevailed in the retrospective study relating to assessment of the benefits of air toxics control. In addition, the Project Team will endeavor to provide a more complete and effective assessment of the ecological effects of air pollution control.