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# The Benefits and Costs of the Clean Air Act 1990 to 2010

*EPA Report to Congress*

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# Executive Summary

Section 812 of the Clean Air Act Amendments of 1990 requires the Environmental Protection Agency 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. This Report to Congress, the first of a series of prospective studies we plan to produce every two years, presents the results and conclusions of our analysis of the benefits and costs of the Clean Air Act during the period from 1990 to 2010. The main goal of this report is to provide Congress and the public with comprehensive, up-to-date information on the Clean Air Act's social costs and benefits, including improvements in human health, welfare, and ecological resources.

The first report that the EPA created under the section 812 authority, The Benefits and Costs of the Clean Air Act: 1970 to 1990, was published and conveyed to Congress in October 1997. This retrospective analysis comprehensively assessed the benefits and costs of all requirements of the 1970 Clean Air Act and the 1977 Amendments, up to the passage of the Clean Air Act Amendments of 1990. The results of the retrospective analysis showed that the nation's investment in clean air was more than justified by the substantial benefits that were gained in the form of increased health, environmental quality, and productivity.

The Clean Air Act Amendments of 1990 built upon the significant progress made by the original Clean Air Act of 1970 and its 1977 amendments in improving the nation's air quality. The amendments utilized the existing structure of the Clean Air Act, but strengthened those requirements to tighten and clarify implementation goals and timing, increase the stringency of some requirements, revamp the hazardous air pollutant regulatory program, refine and streamline permitting requirements, and introduce new programs for the control of acid rain precursors and stratospheric ozone depleting substances. Because the 1990 Amendments represent an incremental improvement to the nation's clean air program, the analysis summarized in this report was designed to estimate the costs and benefits of the 1990 Amendments incremental to those assessed in the retrospective analysis. Our intent is that this report and its predecessor, the retrospective, together provide a comprehensive assessment of current and expected future clean air regulatory programs and their costs and benefits.

This first prospective analysis consists of a sequence of six steps. These six steps, listed in order of completion, are:

(1) estimate air pollutant emissions in 1990, 2000, and 2010;
(2) estimate the cost of emission reductions arising from the Clean Air Act Amendments;
(3) model air quality based on emissions estimates;
(4) quantify air quality related health and environmental effects;
(5) estimate the economic value of cleaner air; and
(6) aggregate results and characterize uncertainties.

The methodology and results for each step are summarized below and described in detail in the chapters of this report.

## Air Pollutant Emissions

Estimation of reductions in pollutant emissions afforded by the 1990 Clean Air Act Amendments (CAAA) serves as the starting point for this study's subsequent benefit and cost estimates. We focused our emissions analysis on six major pollutants: volatile organic compounds (VOCs), nitrogen oxides

($NO_x$), sulfur dioxide ($SO_2$), carbon monoxide (CO), coarse particulate matter ($PM_{10}$), and fine particulate matter ($PM_{2.5}$). For each of these pollutants we forecast emissions for the years 2000 and 2010 under two different scenarios: a) the Pre-CAAA scenario that assumes no additional control requirements would be implemented beyond those that were in place when the 1990 CAAA were passed; and b) the Post-CAAA scenario that incorporates the effects of controls which, when we formulated the scenario, we expected would be likely to occur as a result of implementing the 1990 Amendments. Emissions estimates for both the Pre-CAAA and Post-CAAA scenarios reflect expected growth in population, transportation, electric power generation, and other economic activity by 2000 and 2010. We compare the emissions estimates under each of these scenarios to estimate the effect of the CAAA requirements on future emissions.

The results of the emissions phase of the assessment indicate that the 1990 Clean Air Act Amendments significantly reduce future emissions of air pollutants. Substantial reductions will be achieved for the two major precursors of ambient ground-level ozone: volatile organic compounds (VOCs) and oxides of nitrogen ($NO_x$). Relative to the Pre-CAAA scenario, estimated VOC emissions under the Post-CAAA case are 35 percent lower by 2010. This change in emissions is due largely to VOC reductions from motor vehicles and area sources (e.g., dry cleaners, commercial bakeries, and other widely dispersed sources).

The $NO_x$ emission reduction under the Post-CAAA scenario represents the greatest proportional emissions change estimated in our analysis. For the year 2010, the Post-CAAA $NO_x$ emissions estimate is 39 percent lower than the Pre-CAAA estimate, representing a decrease in emissions of almost 11 million tons. Nearly half of this reduction is from utilities, largely as a result of the particular $NO_x$ emissions cap and trading program we assumed under the Post-CAAA scenario. The remaining reductions are attributable to cuts in motor vehicle and non-utility point source emissions.

Carbon monoxide (CO) emissions contribute directly to concentrations of carbon monoxide in the environment. The 2010 Post-CAAA estimate for CO emissions is 81.9 million tons, 23 percent lower than the Pre-CAAA projection. The reduction in CO emissions is mostly due to motor vehicle emission controls.

The CAAA also will achieve a substantial reduction in precursors of fine particulate matter ($PM_{2.5}$). Sulfur dioxide ($SO_2$) is an important precursor of PM. By 2010, $SO_2$ emissions are 31 percent lower under the Post-CAAA scenario. Of the 8.2 million ton difference between Pre- and Post-CAAA $SO_2$ estimates, 96 percent is attributable to additional control of utility emissions through a national cap-and-trade program involving marketable $SO_2$ emission allowances. Oxides of nitrogen, discussed above, are also important fine PM precursors.

We project the 1990 Clean Air Act Amendments to have more modest effects on emissions of particulate material which is emitted in solid form (i.e., "primary" or "direct" $PM_{10}$ and $PM_{2.5}$ emissions). Overall, emissions of primary $PM_{10}$ and $PM_{2.5}$ are each approximately four percent lower in 2010 under the Post-CAAA scenario than under the Pre-CAAA scenario. Although the incremental effects of the Clean Air Act Amendments on primary PM emissions will be relatively small, PM in the atmosphere is comprised of both directly emitted primary particles and particles that form in the atmosphere through secondary processes as a result of emissions of $SO_2$, $NO_x$, and organic compounds. These PM species, formed by the conversion of gaseous pollutant emissions, are referred to collectively as "secondary" PM. Because, as noted above, the 1990 Amendments achieve substantial reductions in these gaseous precursor emissions, the Amendments have a much larger effect on $PM_{10}$ and $PM_{2.5}$ levels in the atmosphere than might be apparent if only the changes in directly emitted primary particles are considered.

## Compliance Costs

Our estimate of the costs of the Clean Air Act Amendment provisions is based on an evaluation of the increases in expenditures incurred by various entities to meet the additional control requirements incorporated in the Post-CAAA case. These costs include operation and maintenance (O&M) expenditures —which includes research and development (R&D) and other similarly recurring expenditures— plus amortized capital costs (i.e., depreciation plus

interest costs associated with the existing capital stock). Relative to the Pre-CAAA case, Post-CAAA scenario total annual compliance costs for Titles I through V are approximately $19 billion higher by the year 2000, rising to $27 billion by the year 2010.

Compliance with Title I, Provisions for Attainment and Maintenance of National Ambient Air Quality Standards (NAAQS), accounts for $14.5 billion, or over half, of the estimated increase in year 2010 compliance costs. Compliance with mobile source emissions control provisions under Title II of the Clean Air Act Amendments accounts for an additional 30 percent of the total costs, or $9 billion annually by 2010. Provisions to control acid deposition and emissions of stratospheric ozone depleting substances account for most of the remainder of the costs.

These direct compliance costs provide a good, but incomplete, measure of the total effect of the Clean Air Act Amendments on the U.S. economy. A complete picture of the indirect impacts of these costs would include changes in employment and prices as well as impacts that might be experienced among customers of the firms that must incur these costs. While these indirect effects could be important, we believe the direct cost estimates provide a good initial measure of the effect of the Clean Air Act Amendments on the U.S. economy, as well as an appropriate metric for comparison with the direct benefits reported here.

**Table ES-1**
**Summary Comparison of Benefits and Costs (Estimates in millions 1990$)**

| | Titles I through V | |
|---|---|---|
| | Annual Estimates | |
| | 2000 | 2010 |
| ***Monetized Direct Costs:*** | | |
| Low [a] | | |
| Central | $19,000 | $27,000 |
| High [a] | | |
| ***Monetized Direct Benefits:*** | | |
| Low[b] | $16,000 | $26,000 |
| Central | $71,000 | $110,000 |
| High[b] | $160,000 | $270,000 |
| ***Net Benefits:*** | | |
| Low | ($3,000) | ($1,000) |
| Central | $52,000 | $83,000 |
| High | $140,000 | $240,000 |
| ***Benefit/Cost Ratio:*** | | |
| Low[c] | less than 1/1 | less than 1/1 |
| Central | 4/1 | 4/1 |
| High[c] | more than 8/1 | more than 10/1 |

[a] The cost estimates for this analysis are based on assumptions about future changes in factors such as consumption patterns, input costs, and technological innovation. We recognize that these assumptions introduce significant uncertainty into the cost results; however the degree of uncertainty or bias associated with many of the key factors cannot be reliably quantified. Thus, we are unable to present specific low and high cost estimates.

[b] Low and high benefits estimates are based on primary results and correspond to 5th and 95th percentile results from statistical uncertainty analysis, incorporating uncertainties in physical effects and valuation steps of benefits analysis. Other significant sources of uncertainty not reflected include the value of unquantified or unmonetized benefits that are not captured in the primary estimates and uncertainties in emissions and air quality modeling.

[c] The low benefit/cost ratio reflects the ratio of the low benefits estimate to the central costs estimate, while the high ratio reflects the ratio of the high benefits estimate to the central costs estimate. Because we were unable to reliably quantify the uncertainty in cost estimates, we present the low estimate as "less than X," and the high estimate as "more than Y", where X and Y are the low and high benefit/cost ratios, respectively.

## Human Health and Environmental Benefits

To estimate benefits, the results of the emissions analysis served as the principal input to a linked series of models. We used these models to estimate changes in air quality, human health effects, ecological effects, and, ultimately, the net economic benefits of the Clean Air Act Amendments. The goals of these steps in the analysis were to estimate the implications of changes in emissions resulting from compliance with the Clean Air Act Amendments on criteria pollutant air quality throughout the lower 48 states, and the impacts on human health and the environment that result from these changes.

We focused our air quality modeling efforts on estimating the impact of Pre- and Post-CAAA emissions on ambient concentrations of ozone, $PM_{10}$, $PM_{2.5}$, $SO_2$, $NO_x$, and CO and on acid deposition and visibility in future years. We found that the majority of the total monetized benefits, however, is attributable to changes in particulate matter concentrations and, more specifically, to the effect of these ambient air quality changes on avoidance of premature mortality. We estimate that 2010 Post-CAAA $PM_{10}$ and $PM_{2.5}$ concentrations in the eastern U.S. will average about 5 to 10 percent lower than 2010 Pre-CAAA concentrations, with some areas of the eastern U.S. experiencing much greater reductions of up to 30 percent. The air quality modeling also indicates a substantial overall reduction in future-year $PM_{10}$ and $PM_{2.5}$ concentrations throughout the western U.S., including most population centers, following implementation of the Clean Air Act Amendments.

The direct benefits of the air quality improvements we estimated under the Post-CAAA scenario include reduced incidence of a number of adverse human health effects, improvements in visibility, and avoided damage to agricultural crops. The estimated annual economic value of these benefits in the year 2010 ranges from $26 to $270 billion, in 1990 dollars, with a central estimate, or mean, of $110 billion. These estimates do not include a number of other potentially important effects which could not be readily quantified and monetized (i.e., converted to dollar terms). These excluded effects include a wide range of ecosystem changes, air toxics-related human health effects, and a number of additional health effects associated with criteria pollutants.

In addition, these results reflect the particular choices we made with respect to interpretations of the available scientific and economic literature and adoption of paradigms for representing health and environmental changes in economic terms. We refer to these results, then, as our "primary" estimates; however, in the text of this report we also present some alternative results which reflect other available choices for models or assumptions.

One particularly important assumption of our primary analysis is 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. Future research may lead to revisions in this assumption as well as other key assumptions, data, and models we use to estimate the benefits and costs of the Clean Air Act. Such revisions may in turn imply significant changes in the estimates of Clean Air Act costs and benefits presented here and in past and future assessments. In our judgment, however, the primary results reflect the best currently available science and the most up-to-date tools and data we had at our disposal — and the most reasonable assumptions we could adopt— as each step of the analysis was implemented.

Cleaner air also yields benefits to ecological systems. This first section 812 prospective analysis devotes a great deal of effort to characterizing and, where possible, quantifying and monetizing the impacts of air pollutants on natural systems. Our increased effort is in part a result of the findings of the retrospective analysis, where we identified a better understanding of ecological effects as an important research direction for the first prospective and subsequent analyses. Quantified benefits of CAAA programs reflected in the overall monetized benefits include: increased agricultural and timber yields; reduced effects of acid rain on aquatic ecosystems; and reduced effects of nitrogen deposited to coastal estuaries. Many ecological benefits, however, remain difficult or impossible to quantify, or can only be quantified for a limited geographic area. The magnitude of quantified benefits and the wide range of unquantified benefits nonetheless suggest that as we learn more about ecological systems and can conduct more comprehensive ecological benefits assessments, estimates of these benefits could be substantially greater.

We developed separate estimates for the Title VI provisions of the CAAA designed to protect stratospheric ozone. Stratospheric ozone is the layer of the atmosphere that protects the planet from the harmful effects of ultraviolet radiation (UV-b). Our primary estimate of the cumulative benefits of Title VI is $530 billion. Using the same uncertainty estimation procedure as for other parts of the analysis, we estimate Primary Low and Primary High estimates of $100 billion to $900 billion, respectively. These estimates partially reflect potential averting behaviors, such as remaining indoors or increasing use of sunscreens or hats, which may mitigate the effects of the UV-b exposure increases estimated in the Pre-CAAA case.

## Comparing Costs to Benefits

Based on the specific tools and techniques we employed, our primary estimate of the net benefit (benefits minus costs) over the entire 1990 to 2010 period of the additional criteria pollutant control programs incorporated in the Post-CAAA case is $510 billion. Our results imply that the monetizable benefits alone exceeded the direct compliance costs by four to one. For many of the factors contributing to this net benefit estimate (especially physical effects and economic valuation estimates), we were able to generate quantitative estimates of uncertainty. By statistically combining these uncertain estimates, we were able to develop a range of net benefit estimates which provide a partial indication of the overall uncertainty surrounding the central estimate of net benefits. This range, reflecting a 90 percent probability range around the mean, or central estimate, is negative $20 billion (implying a small probability that costs could exceed monetized benefits) to positive $1.4 trillion.

The estimates for Title VI also indicate that cumulative benefits ($500 billion) well exceed cumulative costs ($27 billion). The time period of our Title VI analysis (175 years) suggests that these estimates are very uncertain. Nonetheless, the conclusion that benefits well exceed costs holds even at our Primary Low estimate of benefits (the low end of the 90 percent probability range, or $100 billion), and regardless of discount rate used to generate the cumulative estimates from the perspective of the present.

The assumptions necessitated by data limitations, by the current state of the art in each phase of the analytical approach, by the need to predict future conditions, and by the state of current research on air pollution's effects imply that both the mean estimate and the 90 percent probability range around the central estimate are uncertain. While alternative choices for data, models, modeling assumptions, and valuation paradigms may yield results outside the range projected in our primary analysis, we believe based on the magnitude of the difference between the estimated benefits and costs that it is unlikely that eliminating uncertainties or adopting reasonable alternative assumptions would change the fundamental conclusion of this study: the Clean Air Act Amendments' total benefits to society exceed its costs.

The uncertainties in the primary estimates and the controversies which persist regarding model choices and valuation paradigms nonetheless highlight the need for a variety of new and continued research efforts. Based on the findings of this study, the highest priority research needs are:

- Improved emissions inventories and inventory management systems
- A more geographically comprehensive air quality monitoring network, particularly for fine particles and hazardous air pollutants
- Use of integrated air quality modeling tools based on an open, consistent model architecture
- Development of tools and data to assess the significance of wetland, aquatic, and terrestrial ecosystem changes associated with air pollution
- Increased basic and targeted research on the health effects of air pollution, especially particulate matter
- Continued development of economic valuation methods and data, particularly valuation of changes in risks of premature mortality associated with air pollution

Properly directed and funded, such research would improve the results of future analyses of the benefits and costs of the Clean Air Act.

## Review Process

The CAA requires EPA to consult with an outside panel of experts during the development and interpretation of the 812 studies. This panel of ex-

perts was organized in 1991 under the auspices of EPA's Science Advisory Board (SAB) as the Advisory Council on Clean Air Act Compliance Analysis (hereafter, the Council). Organizing the review committee under the SAB ensured that highly qualified experts would review the section 812 studies in an objective, rigorous, and publicly open manner consistent with the requirements and procedures of the Federal Advisory Committee Act (FACA). Council review of the present study began in 1993 with a review of the analytical design plan. Since the initial June 1993 meeting, the Council has met many times to review proposed data, proposed 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 were referred to subcommittees comprised of both Council members and members of other SAB committees. The Council's Health and Ecological Effects Subcommittee (HEES) met several times and provided its own review findings to the full Council. Similarly, the Council's Air Quality Modeling Subcommittee (AQMS) held in-person and teleconference meetings to review methodology proposals and modeling results and conveyed its review recommendations to the parent committee.

An interagency review was conducted, during which a number of analytical issues were discussed. Conducting a benefit/cost analysis of a major statute such as the Clean Air Act requires scores of methodological decisions. Many of these issues are the subject of continuing discussion within the economic and policy analysis communities and within the Administration. Key issues include the treatment of uncertainty in the relationship between particulate matter exposure and mortality; the valuation of premature mortality; the treatment of tax interaction effects; the assessment of stratospheric ozone recovery; and the treatment of ecological and welfare effects. These issues could not be resolved within the constraints of this review. Thus, this report reflects the findings of the EPA and not necessarily other agencies of the Administration.

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# Acronyms and Abbreviations

| | |
|---|---|
| μEq | microequivalents |
| μg | microgram |
| ACT | average cost per ton |
| AGSIM | AGricultural SImulation Model |
| AIC | Akaike information criterion |
| AIRS | Aerometric Information Retrieval System |
| ANC | acid neutralizing capacity |
| ANOVA | analysis of variance |
| AOD | airway obstructive disease |
| AP-42 | EPA's Compilation of Air Pollution Emission Factors |
| ATDM | aerosol and toxics deposition module |
| AQM | air quality modeling |
| AQMS | Air Quality Modeling Subcommittee |
| ATLAS | Aggregate Timber Land Assessment System |
| $b_{ext}$ | light extinction coefficient |
| BAAQMD | Bay Area Air Quality Management District |
| BACT | best available control technology |
| BAF | bioaccumulation factor |
| BARCT | best available retrofit control technology |
| BCF | bioconcentration factor |
| BEA | Bureau of Economic Analysis |
| BID | background information document |
| BIES | Biogenic Emissions Inventory System |
| BLS | Bureau of Labor Statistics |
| BMP | best management practice |
| BNR | biological nutrient removal |
| BS | black smoke |
| C-R | concentration-response |
| CAA | Clean Air Act |
| CAAA | Clean Air Act Amendments |
| CAPI | Clean Air Power Initiative |
| CAPMS | Criteria Air Pollutant Modeling System |
| CARB | California Air Resources Board |
| CASAC | Clean Air Science Advisory Board |
| CASTNet | Clean Air Act Status and Trends Network |
| CB | chronic bronchitis |
| CEM | continuous emissions monitoring |
| CES | constant elasticity of substitution |
| CFC | chlorofluorocarbon |
| CFFP | Clean Fuel Fleet Program |
| CGE | computable general equilibrium |
| CI | compression ignition |
| CO | carbon monoxide |
| COH | coefficient of haze |
| COI | cost of illness |
| COPD | chronic obstructive pulmonary disease |
| CRC | capital recovery cost |
| CRF | capital recovery factor |
| CTG | control technique guideline |
| CV | contingent valuation |
| dbh | diameter at breast height |
| DDT | dichlorodiphenyl-trichloroethane |
| DOE | Department of Energy |
| dV | deciview |
| E-GAS | Economic Growth Analysis System |
| EC | elemental carbon |
| EGU | electrical generating unit |
| EMFAC | emission factors model |
| ER | emergency room |
| EPA | Environmental Protection Agency |
| EPS | Emissions Processing System |
| ERCAM | Emission Reduction and Cost Analysis Model |
| ERL | Environmental Research Laboratory |
| FACA | Federal Advisory Committee Act |
| FAPRI | Food and Agricultural Policy Research Institute |
| FCM | Fuel Consumption Model |
| FDA | Food and Drug Administration |
| $FEV_1$ | forced expiratory volume in one second |

| | |
|---|---|
| FGD | flue gas desulfurization |
| FHWA | Federal Highway Administration |
| FMVCP | Federal Motor Vehicle Control Program |
| FORCARB | forest carbon model |
| FORTRAN | formula translation |
| FR | Federal Register |
| GCVTC | Grand Canyon Visibility Transport Commission |
| GDP | gross domestic product |
| GIRAS | Geographic Information Retrieval Analysis System |
| GIS | geographic information system |
| GNP | gross national product |
| GSP | gross state product |
| H + | hydrogen ions |
| ha | hectare |
| HAP | hazardous air pollutant |
| HARVCARB | harvested carbon model |
| HBFC | hydrobromofluorocarbons |
| HC | hydrocarbon |
| HCFC | hydrochlorofluorocarbon |
| HDDV | heavy-duty diesel vehicle |
| HDGV | heavy-duty gasoline vehicle |
| HDV | heavy-duty vehicle |
| HEES | Health and Ecological Effects Subcommittee |
| Hg | mercury |
| HIV-1 | human immunodeficiency virus type one |
| $HNO_3$ | nitric acid |
| HPMS | Highway Performance Monitoring System |
| $HS_2O_4$ | sulfuric acid |
| I/M | inspection and maintenance |
| ICI | industrial/commercial/institutional |
| ICD | International Classification of Disease |
| ID | identification code |
| IMPROVE | Interagency Monitoring of PROtected Environments |
| IPM | Integrated Planning Model |
| kg | kilogram |
| km | kilometer |
| kWh | kilowatt hour |
| LAER | lowest achievable emission rate |
| lb | pound |
| LDAR | leak detection and repair |
| LDDT | light-duty diesel truck |
| LDDV | light-duty diesel vehicle |
| LDGT | light-duty gasoline truck |
| LDGV | light-duty gasoline vehicle |
| LEV | low emission vehicle |
| LRS | lower respiratory symptom |
| LTO | landing and takeoff operations |
| m | meter |
| $m^3$ | cubic meter |
| MACT | maximum achievable control technology |
| MAG | Maricopa Association of Governments |
| MAGIC | Model of Acidification of Groundwater in Catchments |
| MC | motorcycle |
| MCF | methyl chloroform |
| MDL | method detection limit |
| MM4 | mesoscale model four |
| MMBtu | million British thermal units |
| MRAD | minor restricted activity day |
| Models-3 | Third Generation Air Pollution Modeling System |
| MOU | memorandum of understanding |
| MOBILE | mobile source emission factor model |
| MPO | metropolitan planning organization |
| MWC | municipal waste combustor |
| MWI | medical waste incinerator |
| N | nitrogen |
| NAA | nonattainment area |
| NAAQS | National Ambient Air Quality Standards |
| NAPAP | National Acid Precipitation Assessment Program |
| NASA | National Aeronautics and Space Administration |
| NCAR | National Center for Atmospheric Research |
| NCLAN | National Crop Loss Assessment Network |
| NE | northeast |
| NEMS | National Energy Modeling System |
| NERC | North American Electric Reliability Council |
| NESHAP | National Emission Standards for Hazardous Air Pollutants |

| | |
|---|---|
| NET | National Emission Trend |
| $NH_3$ | ammonia |
| NHANES | National Health and Nutrition Examination |
| NIH | National Institutes of Health |
| NMOC | non-methane organic compound |
| NO | nitrogen oxide |
| $NO_2$ | nitrogen dioxide |
| $NO_x$ | nitrogen oxides |
| NP | national park |
| NPI | National Particulates Inventory |
| NPP | net primary productivity |
| NPV | net present value |
| NSPS | new source performance standard |
| NSR | new source review |
| NSWS | National Surface Waters Survey |
| NYSDEC | New York Department of Environmental Conservation |
| $O_3$ | ozone |
| O&M | operation and maintenance |
| OBD | onboard diagnostic |
| OC | organic carbon |
| ODS | ozone-depleting substance |
| OMB | Office of Management and Budget |
| OMS | Office of Mobile Sources |
| OPPE | Office of Policy, Planning and Evaluation |
| ORIS | Office of the Regulatory Information System |
| OSD | ozone season daily |
| OTAG | Ozone Transport Assessment Group |
| OTC | Ozone Transport Commission |
| OTR | Ozone Transport Region |
| P-i-G | plume-in-grid |
| PAN | peroxyacetyl nitrate |
| Pb | lead |
| PCB | polychlorinated biphenyl |
| PCDD | polychlorinated dibenzo-p-dioxin |
| PCDF | polychlorinated dibenzofurans |
| PCE | perchloroethylene |
| pH | logarithm of the reciprocal of hydrogen ion concentration, a measure of acidity |
| PM | particulate matter (both $PM_{10}$ and $PM_{2.5}$) |
| $PM_{10}$ | particulate matter less than or equal to 10 microns in diameter |
| $PM_{2.5}$ | particulate matter less than or equal to 2.5 microns in diameter |
| PnET | Net Photosynthesis and Evapo-Transpiration model |
| POC | parameter occurrence code |
| POTW | publically owned treatment works |
| ppb | parts per billion |
| ppm | parts per million |
| PRYL | percentage relative yield loss |
| PRZM | Pesticide Root Zone Model |
| PSU | Pennsylvania State University |
| QALY | quality adjusted life years |
| R&D | research and development |
| RACT | reasonable available control technology |
| RAD | restricted activity day |
| RADM | Regional Acid Deposition Model |
| RELMAP | Regional Lagrangian Model of Air Pollution |
| REMSAD | Regulatory Modeling System for Aerosols and Acid Deposition |
| RE | rule effectiveness |
| RFG | reformulated gasoline |
| RHC | reactive hydrocarbon |
| RIA | regulatory impact analysis |
| RFP | reasonable further progress |
| $RO_2$ | peroxy radical |
| ROP | rate of progress |
| RPM | Regional Particulate Model |
| RUM | Random Utility Model |
| RVP | Reid vapor pressure |
| S | sulfur |
| SAB | Science Advisory Board |
| SAS | Statistical Analysis Software |
| SAV | submerged aquatic vegetation |
| SCAQMD | South Coast Air Quality Management District |
| SCAQS | South Coast Air Quality Study |
| SCC | Source Classification Code |
| SCR | selective catalytic reduction |
| SEDS | State Energy Data Systems |
| SI | spark ignition |
| SIC | Standard Industrial Classification |
| SIP | State Implementation Plan |
| $SO_2$ | sulfur dioxide |

| | |
|---|---|
| SOA | secondary organic aerosol |
| SoCAB | South Coast Air Basin |
| SOCMI | synthetic organic chemical manufacturing industry |
| SUM06 | sum of hourly ozone concentrations at or above 0.06 ppm |
| TAC | total annualized costs |
| TAF | temporal allocation factors |
| TAMM | Timber Assessment Market Model |
| TBRP | Tampa Bay Estuary Program |
| TCDD | tetrachlorodibenzo-p-dioxin |
| TEQ | toxic equivalency |
| TLEV | transitional low emission vehicle |
| tpd | tons per day |
| TREGRO | tree growth model |
| TSDF | treatment, storage, and disposal facility |
| TSP | total suspended particulates |
| UAM | Urban Airshed Model |
| URS | upper respiratory symptoms |
| USDA | United States Department of Agriculture |
| ULEV | ultra-low emission vehicle |
| USGS | United States Geological Survey |
| UV | ultraviolet |
| VMT | vehicle miles traveled |
| VNA | Voronoi neighbor averaging |
| VOC | volatile organic compound |
| VR | visual range |
| VSL | value of statistical life |
| VSLY | value of statistical life year |
| WEFA | Wharton Economic Forecasting Associates |
| WHO | World Health Organization |
| WLD | work-loss days |
| WTA | willingness-to-accept |
| WTP | willingness-to-pay |
| $XO_2$ | halogenated peroxy radical |
| yr | year |
| ZEV | zero emission vehicle |

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# Introduction

Chapter 1

## Background and Purpose

Section 812 of the 1990 Clean Air Act Amendments requires the EPA to develop periodic Reports to Congress that estimate the benefits and costs of the Clean Air Act (CAA). The first report EPA created under this authority, *The Benefits and Costs of the Clean Air Act: 1970 to 1990*, was published and conveyed to Congress in October 1997. This retrospective analysis comprehensively assessed benefits and costs of requirements of the 1970 Clean Air Act and the 1977 Amendments, up to the passage of the Clean Air Act Amendments of 1990. The results of the retrospective analysis showed that the nation's investment in clean air was more than justified by the substantial benefits that were gained in the form of increased health, environmental quality, and productivity. The aggregate benefits of the CAA during the 1970 to 1990 period exceeded costs by a factor of 10 to 100 times.

Before the retrospective analysis was complete, we began the process of assessing the prospective benefits and costs of the Clean Air Act Amendments (CAAA), covering the period 1990 to 2010. This report, the first of a series that we plan to produce every two years, is the result of our prospective analysis of the 1990 Amendments.

Similar to the retrospective analysis, this document has one primary and several secondary objectives. The main goal is to provide Congress and the public with comprehensive, up-to-date information on the CAAA's social costs and benefits, including health, welfare, and ecological benefits. Data and methods derived from the retrospective analysis have already been used to assist policy-makers in refining clean air regulations over the last two years, and we hope the information continues to prove useful to Congress during future Clean Air Act reauthorizations. Beyond the statutory goals of section 812, EPA also intends to use the results of this study to help support decisions on future investments in air pollution research. In addition, lessons learned in conducting this first prospective will help better target efforts to improve the accuracy and usefulness of future prospective analyses.

## Relationship of This Report to Other Regulatory Analyses

The Clean Air Act Amendments of 1990 augment the significant progress made in improving the nation's air quality through the original Clean Air Act of 1970 and its 1977 amendments. The amendments built off the existing structure of the original Clean Air Act, but went beyond those requirements to tighten and clarify implementation goals and timing, increase the stringency of some federal requirements, revamp the hazardous air pollutant regulatory program, refine and streamline permitting requirements, and introduce new programs for the control of acid rain and stratospheric ozone depleters. Because the 1990 Amendments represent an additional improvement to the nation's existing clean air program, the analysis summarized in this report was designed to estimate the costs and benefits of the 1990 CAAA incremental to those costs and benefits assessed in the retrospective analysis. In economic terminology, this report addresses the marginal costs and benefits of the 1990 CAAA. Our intent is that this report and its predecessor, the retrospective analysis, together provide a comprehensive assessment of current and expected future clean air regulatory programs and their costs and benefits.

Because of the time and resources necessary to conduct this type of comprehensive prospective assessment, however, and the ongoing refinements in Clean Air Act regulatory programs, the estimates presented in this report do not reflect some recent

major developments in EPA's clean air program. The prospective analysis, for example, does not capture the benefits and costs of EPA's recent revision of the particulate matter and ozone National Ambient Air Quality Standards (NAAQS), the recently proposed Tier II tailpipe standards, or the recently finalized regional haze standards. Neither costs nor benefits of those actions are reflected in the estimates presented here. In most cases, Regulatory Impact Analyses (RIAs) for those actions did incorporate the section 812 prospective Post-CAAA scenario as their starting point, or baseline, from which the actions were assessed, and in most respects the RIAs used a methodology consistent with that used here.[1] As a result, cost and benefit estimates presented in those RIAs can be considered incremental to the primary estimates presented in this document.

In addition to omitting these actions from the assessment, this first prospective analysis required locking in a set of emissions reductions to be used in subsequent analyses at a relatively early date (late 1996), and as a result we were compelled to forecast the implementation outcome of several pending programs. The most important of these was the then-ongoing Ozone Transport Assessment Group (OTAG) recommendations for achieving regional-scale reductions of emissions of ground-level ozone precursors. The NOx control program incorporated in the Post-CAAA scenario may not reflect the NOx controls that are actually implemented in a regional ozone transport rule. We acknowledge and discuss these types of discrepancies and their impact on the outcome of our analysis in the document.

Finally, despite our efforts to comprehensively evaluate the costs and benefits of all provisions of the Clean Air Act and its Amendments, there remain a few categories of effects that are not addressed by either the retrospective or prospective analyses. For example, this first prospective analysis does not assess the effect of CAAA provisions on lead exposures, primarily because the 1990 Amendments do not include major new provisions for the control of lead emissions. The vast majority of lead emissions sources present in 1970 were addressed by programs initiated under the original Clean Air Act and the 1977 Amendments; evaluation of the costs and health benefits of these programs were important elements of the retrospective analysis. In the retrospective, however, we were unable to quantify the potentially substantial ecological benefits of controls on lead emissions. While this first prospective analysis reflects a significantly greater investment in quantifying ecological effects, for the reason stated above we did not assess the ecological effects of lead in this analysis either. As a result, the ecological effects of this persistent pollutant, past emissions of which may continue to be released from soils for many years, are not captured by either the retrospective or prospective analyses. In addition, lead previously deposited to soils may be re-entrained in the air as road dust, dust plumes from construction excavations, and other particulate matter emission processes subject to 1990 CAAA controls. Reductions in this re-entrainment of, and potential exposure to, pre-1990 emitted lead due to post-1990 control programs, however, are not reflected in either the section 812 retrospective (1970 to 1990) or prospective (1990 to 2010) benefit analyses.

## Requirements of the 1990 Clean Air Act Amendments

The first prospective analysis, despite the limitations discussed above, presents a comprehensive estimate of costs and benefits of all titles of the 1990 Clean Air Act Amendments. The 1990 Amendments consist of the following eleven titles:

- **Title I.** Establishes a detailed and graduated program for the attainment and maintenance of the National Ambient Air Quality Standards.

- **Title II.** Regulates mobile sources and establishes requirements for reformulated gasoline and clean fuel vehicles.

- **Title III.** Expands and modifies regulations of hazardous air pollutant emissions; and establishes a list of 189 hazardous air pollutants to be regulated.

[1] There are minor differences in the assumptions used to construct the Post- CAAA scenario for this analysis and the baseline used in the PM and ozone NAAQS RIA. For example, the RIA baseline incorporates the effects of 7- and 10-year MACT rules that are not reflected here, because of the timing of the two analyses, and the RIA used a 95 percent rule-effectiveness assumption. In most respects, however, the analyses are compatible.

- **Title IV.** Establishes control programs for reducing acid rain precursors.

- **Title V.** Requires a new permitting system for primary sources of air pollution.

- **Title VI.** Limits emissions of chemicals that deplete stratospheric ozone.

- **Title VII.** Presents new provisions for enforcement.

- **Titles VIII through XI.** Establishes miscellaneous provisions for issues such as disadvantaged business concerns, research, training, new regulation of outer continental shelf sources, and assistance for people who lose their jobs as a result of the Clean Air Act Amendments.

As part of the requirements under Title VIII, section 812 of the Clean Air Act Amendments of 1990 requires the EPA to analyze the costs and benefits to human health and the environment that are attributable to the Clean Air Act. In addition, section 812 directs EPA to measure the effects of this statute on economic growth, employment, productivity, cost of living, and the overall economy of the United States.

## Analytical Design and Review

### *Target Variable*

The prospective analysis compares the overall health, welfare, ecological and economic benefits of the 1990 Clean Air Act Amendment programs 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. Within this analysis, costs can be reliably attributed to individual programs, but the broad-scale approach adopted in the prospective study precludes reliable re-estimation of the benefits of individual standards or programs. Similar to the retrospective benefits analysis, this study calculates the change in incidences of adverse effects implied by changes in ambient concentrations of air pollutants. However, pollutant emissions reductions achieved contribute to changes in ambient concentrations of those, or secondarily formed, pollutants in ways that are highly complex, interactive, and often nonlinear. Therefore, benefits cannot be reliably matched to provision-specific changes in emissions or costs.

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 "Pre-CAAA" and a "Post-CAAA" scenario. The Pre-CAAA scenario essentially freezes federal, state, and local air pollution controls at the levels of stringency and effectiveness which prevailed in 1990. The Post-CAAA scenario assumes that all federal, state, and local rules promulgated pursuant to, or in support of, the 1990 CAAA 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 Chapter 2 and Appendix A of this document.

### *Key Assumptions*

Similar to the retrospective analysis, we made two key assumptions during the scenario design process to avoid miring the analytical process in endless speculation. First, as stated above, we froze air pollution controls at 1990 levels throughout the Pre-CAAA scenario. Second, we assumed that the geographic distributions of population and economic activity remain the same between the two scenarios, although these distributions do change over time under both scenarios to reflect expected patterns of high and low population and economic growth across the country.

The first assumption is an obvious simplification. In the absence of the 1990 CAAA, one would expect to see some air pollution abatement activity, either voluntary or due to state or local regulation. It is conceivable that state and local regulation would have required air pollution abatement equal to –or even greater than– that required by the 1990 CAAA;

particularly since some states, most notably California, have in the past done so. If one were to assume that state and local regulations would have been equivalent to 1990 CAAA standards, then a cost-benefit analysis of the 1990 CAAA would be a meaningless exercise since both costs and benefits would equal zero. Any attempt to predict how states' and localities' regulations would have differed from the 1990 CAAA would be too speculative to support the credibility of the ensuing analysis. Instead, the Pre-CAAA scenario has been structured to reflect the assumption that states and localities would not have invested further in air pollution control programs after 1990 in the absence of the federal CAAA. Thus, this analysis accounts for all costs and benefits of air pollution control from 1990 to 2010 and does not speculate about the fraction of costs and benefits attributable exclusively to the federal CAAA. 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. In the end, the benefits of air pollution controls result from 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 Pre-CAAA scenario, air quality is worse than the actual 1990 conditions and the projected air quality in the Post-CAAA scenario. It is possible that under the Pre-CAAA scenario more people, relative to the Post-CAAA 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 differences between the two scenarios with respect to the spatial pattern of economic activity.

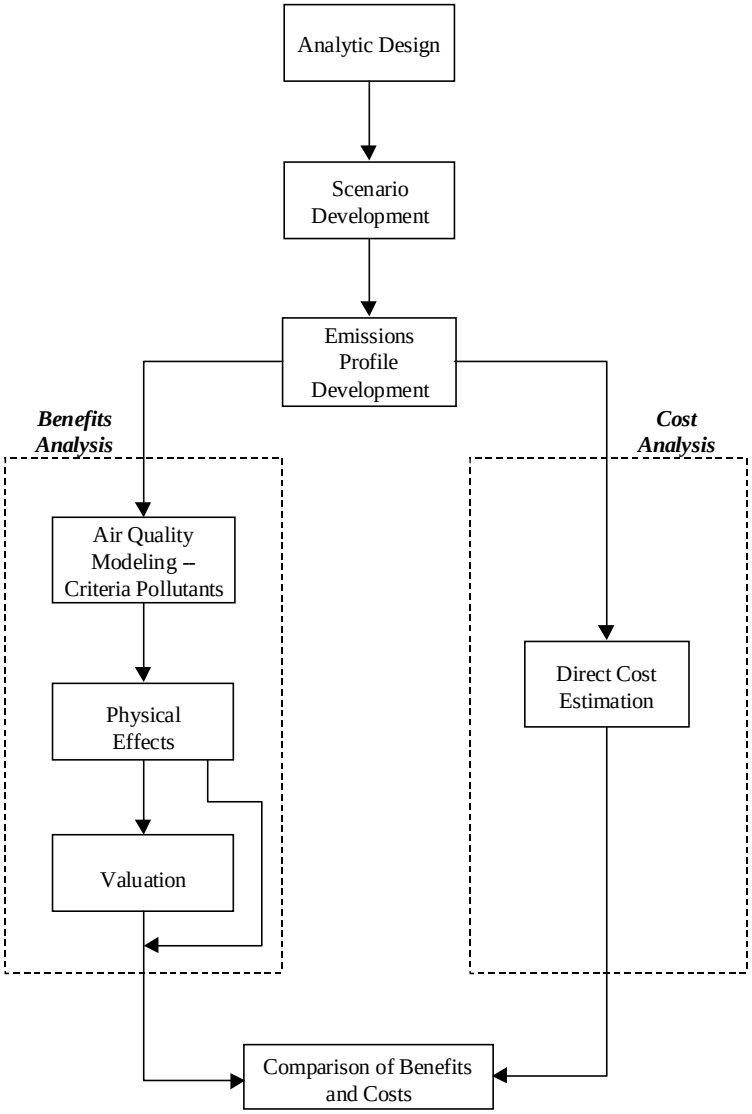
## Analytic Sequence

The analysis comprises a sequence of six basic steps, summarized below and described in detail later in this report. These six steps, listed in order of completion, are:

(1) emissions modeling
(2) direct cost estimation
(3) air quality modeling
(4) health and environmental effects estimation
(5) economic valuation
(6) results aggregation and uncertainty characterization

Figure 1-1 summarizes the analytical sequence used to develop the prospective results; we describe the analytic process in greater detail below.

**Figure 1-1**
**Analytic Sequence for**
**First Section 812 Prospective Analysis**



The first step of the analysis is the estimation of the effect of the 1990 CAAA on emissions sources. We generated emissions estimates through a three step process: (1) construction of an emissions inventory for the base year (1990); (2) projection of emissions for the Pre-CAAA case for two target years, 2000 and 2010, assuming a freeze on emissions control regulation at 1990 levels and continued economic progress, consistent with sector-specific Bureau of Economic Analysis economic activity projections; and (3) construction of Post-CAAA estimates for the same two target years, using the same set of economic activity projections used in the Pre-CAAA case but with regulatory stringency, scope, and timing consistent with EPA's CAAA implementation plan (as of late 1996). The analysis reflects application of utility and other sector-specific emissions models developed and used in various offices of EPA's Office of Air and Radiation. These emissions models provide estimates of emissions of six criteria air pollutants[2] from each of several key emitting sectors. We provide more details in Chapter 2 and Appendix A.

The emissions modeling step is a critical component of the analysis, because it establishes consistency between the subsequent cost and benefit estimates that we develop. Estimates of direct compliance costs to achieve the emissions reductions estimated in the first step are generated as either an integral or subsequent output from the emissions estimation models, depending on the model used. For example, the Integrated Planning Model used to estimate emissions and compliance costs for the utility sector develops an optimal allocation of reductions of sulfur and nitrogen oxides taking into account the regulatory flexibility inherent in the Title IV trading schemes for emissions allocations. In a few cases, for example the Title V permitting requirements, we estimate public and private costs incurred to implement the regulatory requirements through analysis of the relevant RIAs conducted to support promulgation of the rules.

Emissions estimates also form the first step in estimating benefits. After the emissions inventories are developed, they are translated 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 project's resource constraints, the EPA Project Team adopts simplified, linear scaling approaches for some gaseous pollutants. However, for particulate matter, ozone, and other air quality conditions that involve substantial non-linear formation processes and/or long-range atmospheric transport and transformation, the EPA Project Team invests the time and resources needed to use more sophisticated modeling systems. For example, we exercise EPA's Regional Acid Deposition Model/Regional Particulate Model (RADM/RPM) to estimate secondarily formed particulate matter in the eastern U.S.

Up to this point of the analysis, modeled conditions and outcomes establish the Pre-CAAA and Post-CAAA scenarios. However, at the air quality modeling step, the analysis returns to a foundation based on actual historical conditions and data. Specifically, actual 1990 historical air quality monitoring data are used to define the baseline conditions from which the Pre-CAAA and Post-CAAA scenario air quality projections are constructed. We derive air quality conditions under the Pre-CAAA scenario by scaling the historical data adopted for the base-year (1990) by the ratio of the modeled Pre-CAAA and base-year air quality. We use the same approach to estimate future-year air quality for the Post-CAAA scenario. This method takes advantage of the richness of the monitoring data on air quality, provides a realistic grounding for the benefit measures, and yet retains analytical consistency by using the same modeling process for both scenarios. The outputs of this step of the analysis are profiles for each pollutant characterizing air quality conditions at each monitoring site in the lower 48 states.

The Pre-CAAA and Post-CAAA scenario air quality profiles serve as inputs to a modeling system that translates air quality to physical outcomes (e.g., mortality, emergency room visits, or crop yield

[2] The six pollutants are particulate matter (separate estimates for each of $PM_{10}$ and $PM_{2.5}$), sulfur dioxide ($SO_2$), nitrogen oxides ($NO_x$), carbon monoxide (CO), volatile organic compounds (VOCs), and ammonia ($NH_3$). One of the CAA criteria pollutants, ozone ($O_3$), is formed in the atmosphere through the interaction of sunlight and ozone precursor pollutants such as $NO_x$ and VOCs. Ammonia is not a criteria pollutant, but is an important input to the air quality modeling step because it affects secondary particulate formation. The sixth criteria pollutant, lead (Pb), is not included in this analysis since airborne emissions of lead were virtually eliminated by pre-1990 Clean Air Act programs.

losses) through the use of concentration-response functions. Scientific literature on the health and ecological effects of air pollutants provides the source of these concentration-response functions. At this point, we derive estimates 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 geographic area.

In the next step, we use economic valuation models or coefficients to estimate the economic value of the reduction in incidence of those adverse effects amenable to monetization. For example, a distribution of unit values derived from the economic literature provides estimates of the value of reductions in mortality risk. In addition, we compile and present benefits that cannot be expressed in economic terms. In some cases, we calculate quantitative estimates of scenario differences in the incidence of a nonmonetized effect. In many cases, available data and techniques are insufficient to support anything more than a qualitative characterization of the change in effects.

Next, we compare costs and monetized benefits to provide our primary estimate of the net economic benefits of the 1990 CAAA and associated programs, and a range of estimates around that primary estimate reflecting quantified uncertainties associated with the physical effects and economic valuation steps. The monetized benefits used in the net benefit calculations reflect only a portion of the total benefits due to limitations in analytical resources, available data and models, and the state of the science. For example, in many cases we are unable to quantify or monetize the potentially large benefits of air pollution controls that result from protection of the health, structure, and function of ecosystems. In addition, although available scientific studies demonstrate clear links between air quality changes and changes in many human health effects, the available studies do not always provide the data needed to quantify and/or monetize some of these effects.

Finally, we present a limited set of alternative benefit estimates which reflect methods, models, or assumptions that differ from those we used to derive the primary net benefit estimate. We also quantify some of the uncertainties surrounding these alternative estimates. In addition, beyond those variables for which alternative results are estimated, we conduct sensitivity analyses for a number of variables that may influence the primary net benefit estimate. The primary estimate and the range around this estimate, however, reflect our current interpretation of the available literature; our judgments regarding the best available data, models, and modeling methodologies; and the assumptions we consider most appropriate to adopt in the face of important uncertainties.

In addition, throughout the report at the end of the chapter we summarize the major sources of uncertainty for each analytic step. Although the impact of many of these uncertainties cannot be quantified, we qualitatively characterize the magnitude of effect on our net benefit results by assigning one of two classifications to each source of uncertainty: *potentially major* factors could, in our estimation, have effects of greater than five percent of the total net benefits; and *probably minor* factors likely have effects less than five percent of total net benefits.

## Review Process

The CAA requires EPA to consult with an outside panel of experts during the development and interpretation of the 812 studies. This panel of experts was organized in 1991 under the auspices of EPA's Science Advisory Board (SAB) as the Advisory Council on Clean Air Act Compliance Analysis (hereafter, the Council). Organizing the review committee under the SAB ensured that highly qualified experts would review the section 812 studies in an objective, rigorous, and publicly open manner consistent with the requirements and procedures of the Federal Advisory Committee Act (FACA). Council review of the present study began in 1993 with a review of the analytical design plan. Since the initial June 1993 meeting, the Council has met many times to review proposed data, proposed 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 were referred to subcommittees comprised of both Council members and members of other SAB committees. The Council's Health and Ecological Effects Subcommittee (HEES) met several times and provided

its own review findings to the full Council. Similarly, the Council's Air Quality Modeling Subcommittee (AQMS) held in-person and teleconference meetings to review methodology proposals and modeling results and conveyed its review recommendations to the parent committee.

An interagency review was conducted, during which a number of analytical issues were discussed. Conducting a benefit/cost analysis of a major statute such as the Clean Air Act requires scores of methodological decisions. Many of these issues are the subject of continuing discussion within the economic and policy analysis communities and within the Administration. Key issues include the treatment of uncertainty in the relationship between particulate matter exposure and mortality; the valuation of premature mortality; the treatment of tax interaction effects; the assessment of stratospheric ozone recovery; and the treatment of ecological and welfare effects. These issues could not be resolved within the constraints of this review. Thus, this report reflects the findings of the EPA and not necessarily other agencies of the Administration.

## Report Organization

The remainder of the main text of this report summarizes the key methodologies and findings our prospective study.

- Chapter 2 summarizes emissions modeling and key elements of the regulatory scenarios.
- Chapter 3 discusses the direct cost estimation.
- Chapter 4 presents the air quality modeling methodology and sample results.
- Chapter 5 describes the approaches used and principal results obtained through the human health effects estimation process.
- Chapter 6 describes the human health effects economic valuation methodology and results.
- Chapter 7 summarizes the ecological and other welfare effects analyses, including assessments of commercial timber, agriculture, visibility, and other categories of effects.
- Chapter 8 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 provides additional detail on the sector-specific emissions modeling effort.
- Appendix B covers the direct costs.
- Appendix C provides details of the air quality models used and results obtained.
- Appendix D presents the human health effects estimation methodology and results.
- Appendix E describes the ecological benefits estimation methods and results.
- Appendix F presents the agricultural benefits estimation methodology and results.
- Appendix G provides details of the stratospheric ozone analysis.
- Appendix H describes the methods and assumptions used to value quantified effects of the CAA in economic terms.
- Appendix I describes areas of research which may increase comprehensiveness and/or reduce uncertainties in effect estimates for future assessments.

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# Emissions

Chapter 2

Estimation of pollutant emissions, a key component of this prospective analysis, serves as the starting point for subsequent benefit and cost estimates. We focused the emissions analysis on six major pollutants: volatile organic compounds (VOCs), nitrogen oxides ($NO_x$), sulfur dioxide ($SO_2$), carbon monoxide (CO), particulate matter with an aerodynamic diameter of 10 microns or less ($PM_{10}$), and fine particulate matter ($PM_{2.5}$).[1] For each of these pollutants we projected 1990 emissions to the years 2000 and 2010 under two different scenarios: a) the *Pre-CAAA* scenario which assumes no additional control requirements would be implemented beyond those in place when the 1990 Amendments were passed; and b) the *Post-CAAA* scenario which incorporates the effects of controls authorized by the 1990 Amendments. We compare the emissions estimates under each of these scenarios to forecast the effect of the CAAA requirements on future emissions.

This chapter consists of four sections. The first section provides an overview of our approach for developing the Pre- and Post-CAAA control scenarios and projecting emissions from 1990 levels to 2000 and 2010. The second section summarizes our emissions projections for the years 2000 and 2010 and presents our estimates of changes in future emissions resulting from the implementation of the 1990 Amendments. The third section compares these results with other estimates that are based upon more recent emissions data. Finally, we conclude this chapter with a summary of the key uncertainties associated with estimating emissions.

## Overview Of Approach

We projected emissions for five major source categories: industrial point sources, utilities, nonroad engines/vehicles, motor vehicles, and area sources (see Table 2-1).[2] The basic method involves estimating emissions in the 1990 base-year, adjusting the base-year emissions to reflect projected growth in the level of pollution-generating activity by 2000 and 2010 in the absence of additional CAAA requirements, and modifying these projections to reflect future-year control assumptions. The resulting estimates depend largely upon three factors: the method for selecting the base-year inventory, the indicators used to forecast growth and the effectiveness of future controls, and the specific regulatory programs incorporated in the Pre- and Post-CAAA scenarios.

We constructed the base-year inventory using 1990 emissions levels. For all of the air pollutants examined in this analysis except particulate matter, we selected emissions levels from Version 3 of the National Particulates Inventory (NPI) to serve as the baseline. This inventory consists of emissions data compiled primarily by the National Acid Precipitation Assessment Program (NAPAP), EPA's Office of Mobile Sources (OMS), and the Federal Highway Administration (FHWA). For both $PM_{2.5}$ and $PM_{10}$, however, we updated NPI estimates to incorporate changes in the methodology used to calculate fugitive dust emissions. Adoption of this new technique, also used to develop EPA's National Emission Trend

[1] We also estimated ammonia ($NH_3$) emissions. $NH_3$ influences the formation of secondary PM (PM formed as a result of atmospheric chemical processes). We used $NH_3$ emissions estimates as an input during the air quality modeling phase of the prospective analysis when estimating future-year ambient PM concentrations. However, we did not examine the human health and environmental effects of exposure to $NH_3$. In addition to $NH_3$, we also estimated mercury (Hg) emissions. We qualitatively evaluated the effects of Hg emissions on ecological systems, but we did not examine the impact of Hg on human health. We did not estimate the effect of the CAAA on lead (Pb) emissions. By 1990 most major airborne Pb emission sources were already controlled and the CAAA has minimal additional impact on Pb emissions.

[2] We estimated utility and industrial point source emissions at the plant/facility level. We estimated nonroad engine/vehicle, motor vehicle, and area source emissions at the county level.

**Table 2-1**
**Major Emissions Source Categories**

| Source Category | Examples |
|---|---|
| Industrial Point Sources | boilers, cement kilns, process heaters, turbines |
| Utilities | electricity producing utilities |
| Nonroad Engines/Vehicles | aircraft, construction equipment, lawn and garden equipment, locomotives, marine engines |
| Motor Vehicles | buses, cars, trucks (sources that usually operate on roads and highways) |
| Area Sources | agricultural tilling, dry cleaners, open burning, wildfires |

(NET) $PM_{2.5}$ and $PM_{10}$ inventory, leads to lower estimates of fugitive dust emissions and therefore of overall primary PM.[3]

Once we established the base-year inventory, we projected emissions to the years 2000 and 2010, accounting for the influences expected to cause future emissions to differ from 1990 levels. For all but utility sources, we rely on an emissions analysis using the Emissions Reduction and Cost Analysis Model (ERCAM) which incorporates the effects of the level of pollution-generating activity and the stringency and success of regulations designed to protect air quality. In this analysis, we view changes in economic growth as an important indicator of future activity levels and thus, future emissions. We used 1995 Bureau of Economic Analysis (BEA) Gross State Product (GSP) projections to forecast the growth of emissions from industrial point sources. We relied on BEA GSP projections as well as data on BEA predicted changes in population to estimate future emissions from nonroad and area sources.[4] We used BEA population growth as an indicator of the increase in nonroad emissions from recreational marine vessels, recreational vehicles, and lawn/garden equipment as well as an indicator of the increase in area source solvent emissions (e.g., VOC emissions from dry cleaners). For motor vehicle sources, we estimated the growth in activity based primarily on the projected increase in vehicle miles traveled (VMT). We develop future VMT estimates using the EPA MOBILE fuel consumption model.

We estimated the impact of CAAA regulations on industrial point source, nonroad, motor vehicle, and area source emissions based on expected control efficiency and rule effectiveness. Control efficiency represents the percentage reduction in emissions anticipated as a result of the implementation of the CAAA, assuming full compliance and successful operation of all control mechanisms. The rule effectiveness factor accounts for equipment malfunction, non-compliance, and other circumstances that influence the overall effectiveness of air pollution regulations. We selected a rule effectiveness of 80 percent as the standard for this analysis which we applied to stationary source $NO_x$ and VOC controls.[5] Rule effectiveness was not calculated for mobile source controls as an adjustment factor separate from the emissions rates estimated for the various vehicle classes.

To estimate future utility source emissions, we relied on the Integrated Planning Model (IPM). This optimization model forecasts, for the 48 contiguous states and the District of Columbia, emissions from all existing utility power generation units, as well as from independent power producers and other cogeneration facilities that sell wholesale power and are included in the North American Electric Reliability Council (NERC) data base for reliability planning. The model considers future capacity additions by both utilities and independent power producers which might cause an increase in emissions. In addition, the model is capable of producing baseline air

[3] Primary PM consists of directly emitted particles such as wood smoke and road dust. Secondary PM forms in the atmosphere as a result of atmospheric chemical reactions.

[4] The growth forecast for area source agricultural tilling is based on projections of acres planted, not BEA GSP and population projections.

[5] At the time we selected the general rule effectiveness for use in this analysis, 80 percent was the standard factor applied in air pollution modeling. More recent analyses have used higher rule effectiveness values. If a higher rule effectiveness value had been used in this analysis, emissions reduction estimates would be larger and the estimated benefits associated with air quality improvements would be greater.

emissions forecasts and estimates of air emissions levels under various control options at the national and NERC regional and subregional level. We used IPM to estimate base-year (1990) utility source emissions and to project future-year (2000 and 2010) emissions under both the Pre- and Post-CAAA scenarios.

Using emissions analysis or IPM, we estimated future emissions for each of the five major source categories under both the Pre- and Post-CAAA scenarios. While the selection of the base-year inventory, emission growth factors, and rate of regulatory effectiveness all influence the emissions projections, the difference between Pre- and Post-CAAA estimates is primarily determined by the difference in control assumptions incorporated in the two projection scenarios.

## Scenario Development

We developed two contrasting emissions control scenarios, the Pre-CAAA scenario and the Post-CAAA scenario. The Pre-CAAA scenario maintains the air pollution regulatory requirements which existed in 1990 through the 2000 and 2010 analytical period and serves as a baseline against which we measure the changes in emissions projected under the Post-CAAA scenario.[6] This latter scenario assumes the implementation of the 1990 Clean Air Act Amendments and incorporates the influences of the following provisions:

- Title I VOC and $NO_x$ reasonably available control technology (RACT) and reasonable further progress (RFP) requirements for ozone nonattainment areas;
- Title II motor vehicle and nonroad engine/vehicle provisions;
- Title III 2- and 4-year maximum achievable control technology (MACT) standards;
- Title IV $SO_2$ and $NO_x$ emissions programs for utilities;
- Title V permitting system for primary sources of air pollution; and
- Title VI emissions limits for chemicals that deplete stratospheric ozone.[7]

The Post-CAAA scenario also assumes the implementation of region-wide $NO_x$ controls and a cap-and-trade system designed to reduce emissions during the summer months from large utility and industrial sources in the 37 easternmost states that comprise the Ozone Transport Assessment Group (OTAG) domain.[8] In addition, the Post-CAAA scenario incorporates the effects of a similarly designed trading program for the 11 northeast states that comprise the Ozone Transport Region (OTR). This trading program is consistent with Phase II of the Ozone Transport Commission (OTC) Memorandum of Understanding (MOU).[9] We provide more detailed discussion of both Pre- and Post-CAAA scenario development in Appendix A.

## Emissions Estimation Results

The results of the Pre- and Post-CAAA projections indicate that the 1990 Clean Air Act Amendments will likely have a significant effect on future emissions of air pollutants. Table 2-2 displays both base-year (1990) and future-year (2000 and 2010) emissions estimates for the modeled scenarios along with the percent change from Pre- to Post-CAAA VOC, $NO_x$, $SO_2$, CO, $PM_{10}$, and $PM_{2.5}$ projections. A more detailed breakout of 2010 Pre- and Post-CAAA emissions estimates, displaying emissions for each major source category, is contained in Table 2-3. Figures 2-1 through 2-6 show the emissions projections for each of the pollutants examined in this analysis.

Emissions projections for VOC, $NO_x$, $SO_2$, and CO, displayed in Figures 2-1 through 2-4, follow

[6] We also attempted to incorporate in the Pre-CAAA (baseline) scenario the non-CAAA regulations and policies we expect will have a significant effect on emissions between 1990 and 2010. For example, the IPM, which we used to estimate utility emissions, incorporates the effect of the deregulation of railroad rates on $SO_2$ emissions. IPM accounts for the influence of the future cost of low-sulfur coal prices expected to occur as a result of lower railroad rates. The impact of prescribed burning policies for private and federally owned lands on PM emissions is also incorporated in the Pre-CAAA scenario.

[7] For a more detailed discussion of the CAAA provisions incorporated in the Post-CAAA scenario, see Appendix A.

[8] The $NO_x$ control program incorporated in the Post-CAAA scenario may not reflect the $NO_x$ controls that are actually implemented in a regional ozone transport rule.

[9] The Post-CAAA scenario does not incorporate any influences of the recently revised PM and ozone NAAQS regulations or any impact of the recently proposed Tier II tailpipe standards.

**Table 2-2**
**Summary of National Annual Emissions Projections**
**(thousand tons)**

| Pollutant | 1990 Base-Year | 2000 Pre-CAAA | 2000 Post-CAAA | 2000 % Change | 2010 Pre-CAAA | 2010 Post-CAAA | 2010 % Change |
|---|---|---|---|---|---|---|---|
| VOC | 22,715 | 24,410 | 17,874 | -27% | 27,559 | 17,877 | -35% |
| $NO_x$ | 22,747 | 25,021 | 18,414 | -26% | 28,172 | 17,290 | -39% |
| $SO_2$ | 22,361 | 24,008 | 18,013 | -25% | 26,216 | 18,020 | -31% |
| CO | 94,385 | 95,572 | 80,919 | -15% | 107,034 | 81,943 | -23% |
| Primary $PM_{10}$ | 28,289 | 28,768 | 28,082 | -2% | 28,993 | 28,035 | -3% |
| Primary $PM_{2.5}$ | 7,091 | 7,353 | 7,216 | -2% | 7,742 | 7,447 | -4% |

Notes: Totals reflect emissions for the 48 contiguous States, excluding Alaska and Hawaii.
Percent change between Pre-CAAA and Post-CAAA scenarios.

similar patterns. Pre-CAAA estimates indicate emissions of these pollutants would increase, on average, by almost 20 percent from 1990 to 2010. These increases reflect the expectation that anticipated growth in activity levels in the relevant emitting sectors will more than offset reductions achieved by pre-1990 control programs. While we predict relatively steady growth in emissions in the absence of the 1990 Amendments, projections show emissions of these four pollutants would increase at a slightly faster rate over the last ten years of the 20 year projection period.

Post-CAAA estimates of VOC, $NO_x$, $SO_2$, and CO emissions for the modeled regulatory scenarios decrease significantly from 1990 to 2000 and then plateau, remaining relatively constant from 2000 to 2010. The initial decrease is triggered by the implementation of the CAAA and the associated controls. After cleaner means of production are adopted, better emissions control technologies are implemented, and other required changes and improvements are made, emissions reduction slows and in some instances stops all together; emissions may even begin to increase. Although the Post-CAAA estimates for each of the above mentioned pollutants show little or no change in the level of emissions from 2000 to 2010, an overall comparison of our Pre- and Post-CAAA projections indicates that during this time period the 1990 Amendments continue to have an increasingly beneficial effect on emission levels.

Comparison of Pre- and Post- CAAA emissions estimates reveals that by 2010, estimated VOC emissions will be 35 percent lower as a result of the implementation of the CAAA than they would have been if no new control requirements, beyond those in place in 1990, were mandated. This sizeable change in emissions attributable to the Amendments is due largely to estimated VOC reductions from motor vehicle and area sources. The 2010 Post-CAAA estimate for these two source categories combined is 8.2 million tons lower than the Pre-CAAA projection, a total which accounts for 84 percent of the predicted difference in VOC emissions estimated under the two scenarios.

Based on the regulatory programs incorporated in the Post-CAAA scenario, we project that $NO_x$ emissions will be reduced by the greatest percentage. Comparison of projections for the year 2010 indicates the Post-CAAA $NO_x$ estimate is 39 percent lower than the Pre-CAAA estimate, representing a decrease in emissions of 10.8 million tons. We project nearly half of this reduction will come from utilities, while the remaining portions will come from cuts in motor vehicle and non-utility point source emissions.

Figure 2-3 shows that by 2010 we anticipate $SO_2$ levels will be 31 percent lower than they would have been under the Pre-CAAA scenario. We project 96 percent of the 8.2 million ton difference between Pre- and Post-CAAA estimates will result from regulation of utilities, while the remaining reduction comes from motor vehicles.

We estimate 2010 Post-CAAA CO emissions will be 81.9 million tons, 23 percent lower than the Pre-CAAA projection. Much of this reduction we project will be achieved as a result of nonattainment (Title I) and motor vehicle provisions (Title II) of the 1990 Amendments. The more influential programs (in order of importance) are expected to be enhanced vehicle emission inspections, wintertime oxygenated fuel use, and LEV program adoption.

Figures 2-5 and 2-6 indicate that the 1990 Clean Air Act Amendments have more modest effects on primary $PM_{10}$ and $PM_{2.5}$ emissions.[10] For both of these pollutants, Pre-CAAA projections increase at a slow rate from 1990 to 2010. Post-CAAA emissions estimates for primary $PM_{10}$ and $PM_{2.5}$, however, follow different paths. While we estimate implementation of the CAAA will cause primary $PM_{10}$ levels to slowly decrease from 1990 to 2010, Post-CAAA projections indicate primary $PM_{2.5}$ emissions will actually rise despite the influence of the CAAA. Overall, however, emissions of primary $PM_{10}$ and $PM_{2.5}$ both will be approximately four percent lower in 2010 than they would have been without the CAAA.[11]

The significant influence of area source emissions on primary PM emissions levels, combined with the limited regulation of this major source category, explains the limited effect of the CAAA on primary particulate matter emissions. According to data used in this analysis, area sources account for over 90 percent of primary $PM_{10}$ emissions and over 80 percent of primary $PM_{2.5}$ emissions.[12] As a result, even the successful reduction of motor vehicle and nonroad emissions have only a slight impact on overall primary $PM_{10}$ and $PM_{2.5}$ estimates developed for this study.[13] Furthermore, the CAAA's most significant primary PM area source controls target emissions in counties not in compliance with the National Ambient Air Quality Standards (NAAQS).[14] Currently, however, there are fewer than 85 counties in the country that are not in attainment with the national standards. Emissions changes in these areas are capable of having only a minor influence on the overall primary PM level in the United States. Even minor changes in primary PM emissions leading to minor changes in the concentrations of this pollutant, however, are significant. In the subsequent portions of this analysis, sizable benefits are estimated to result from small reductions in PM concentrations in the atmosphere.

The seemingly small impact on direct PM emissions resulting from implementation of the CAAA depicted in Figures 2-5 and 2-6 can be misleading. While these figures illustrate the impact of the 1990 CAAA on primary PM emissions, it is important to remember that ambient PM concentrations are influenced by the presence of both primary and secondary PM. VOCs, $NO_x$, and $SO_2$, all pollutants regulated by the CAA, are secondary PM precursors. The reduction in the emissions of these three pollutants also leads to lower overall PM concentrations in the atmosphere. The complete impact of the CAAA on PM thus is not fully captured by Figures 2-5 and 2-6. Additional discussion of the influence of the CAAA on PM and ambient air quality is provided in Chapter 4 and Appendix C.

As part of this prospective analysis we also estimated future-year $NH_3$ emissions. The 1990 Amendments, however, do not include provisions designed

[10] EPA projected $PM_{10}$ and $PM_{2.5}$ levels holding natural source emissions of particulate matter constant at 1990 levels. The estimates presented in Figures 2-5 and 2-6 have been adjusted; these estimates represent total PM emissions minus natural source emissions (wind erosion).

[11] Directly emitted PM, such as fugitive dust, is referred to as primary PM. Secondary PM is not directly emitted, but rather forms in the atmosphere. $NO_s$ and $SO_2$ are two examples of secondary PM precursors.

[12] As discussed on pages 18 and 20 and in Table 2-5, however, some recent data indicate that the composition data used in this analysis may underestimate the contribution from motor vehicle carbonaceous emissions.

[13] The difference between 2010 Pre- and Post-CAAA estimates for $PM_{10}$ and $PM_{2.5}$ motor vehicle emissions is 31 percent and 39 percent respectively. The difference between 2010 Pre- and Post CAAA estimates for $PM_{10}$ and $PM_{2.5}$ nonroad emissions is 19 percent and 20 percent respectively.

[14] The PM NAAQS referred to here is the 50 $ug/m^3$ (annual mean) 150 $ug/m^3$ (daily mean) standard.

**Table 2-3**
**Summary by Source Category of National Annual Emission Projections to 2010 (thousand tons)**

| Pollutant | Source Category | 1990 | 2010 Pre-CAAA | 2010 Post-CAAA | % Change |
|---|---|---|---|---|---|
| VOC | Utility | 37 | 49 | 50 | 2% |
| | Point | 3,500 | 4,200 | 3,500 | -19% |
| | Area | 10,000 | 13,000 | 8,500 | -36% |
| | Nonroad | 2,100 | 2,600 | 1,900 | -28% |
| | Motor Vehicle | 6,800 | 7,300 | 3,900 | -46% |
| | TOTAL | 23,000 | 28,000 | 18,000 | -35% |
| $NO_x$ | Utility | 7,400 | 9,100 | 3,800 | -58% |
| | Point | 2,900 | 3,600 | 2,200 | -39% |
| | Area | 2,200 | 3,000 | 3,000 | -1% |
| | Nonroad | 2,800 | 3,400 | 2,700 | -20% |
| | Motor Vehicle | 7,400 | 9,100 | 5,600 | -39% |
| | TOTAL | 23,000 | 28,000 | 17,000 | -39% |
| CO | Utility | 330 | 450 | 460 | 2% |
| | Point | 6,000 | 7,400 | 7,400 | 0% |
| | Area | 12,000 | 14,000 | 14,000 | 0% |
| | Nonroad | 14,000 | 19,000 | 18,000 | -4% |
| | Motor Vehicle | 62,000 | 66,000 | 42,000 | -37% |
| | TOTAL | 94,000 | 107,000 | 82,000 | -23% |
| $SO_2$ | Utility | 16,000 | 18,000 | 9,900 | -44% |
| | Point | 4,600 | 6,000 | 6,000 | 0% |
| | Area | 1,000 | 1,500 | 1,500 | 0% |
| | Nonroad | 240 | 240 | 240 | 0% |
| | Motor Vehicle | 570 | 770 | 410 | -47% |
| | TOTAL | 22,000 | 26,000 | 18,000 | -31% |
| Primary $PM_{10}$ | Utility | 280 | 310 | 280 | -9% |
| | Point | 930 | 1,200 | 1,200 | 0% |
| | Area | 26,000 | 27,000 | 26,000 | -3% |
| | Nonroad | 340 | 410 | 340 | -19% |
| | Motor Vehicle | 360 | 300 | 210 | -31% |
| | TOTAL | 28,000 | 29,000 | 28,000 | -3% |
| Primary $PM_{2.5}$ | Utility | 110 | 120 | 110 | -8% |
| | Point | 590 | 750 | 750 | 0% |
| | Area | 5,800 | 6,300 | 6,100 | -2% |
| | Nonroad | 290 | 360 | 290 | -20% |
| | Motor Vehicle | 290 | 230 | 140 | -39% |
| | TOTAL | 7,100 | 7,700 | 7,400 | -4% |

NOTES: Table may not sum due to rounding. Percentage change was calculated prior to rounding.

to regulate $NH_3$. As a result, the Pre- and Post-CAAA estimates follow a similar upward trend. We estimate $NH_3$ emissions will increase roughly 55 percent from 1990 to 2010. Although we do not estimate the costs and benefits associated with $NH_3$ controls and changes in $NH_3$ ambient concentrations as part of this analysis, estimation of $NH_3$ emissions is an important part of the prospective study. $NH_3$ is a secondary PM precursor, and we relied on future-year $NH_3$ emissions estimates as model input to help us estimate PM concentrations.

We also estimated the effect of CAAA provisions on mercury (Hg) emissions for five separate Hg emissions sources: medical waste incinerators (MWI), municipal waste combustors (MWCs), electric utility plants, hazardous waste combustors, and chlor-alkali plants.[15] Together, these sources account for 75 to 80 percent of national anthropogenic airborne Hg emissions. In this analysis we qualitatively examine the effects of mercury emissions reductions on ecological systems (see Chapter 7 and Appendix E). We do not, however, evaluate the impact of Hg on human health.

Table 2-4 displays, for each emission category, base-year (1990) and future-year (2000 and 2010) Pre- and Post-CAAA emissions estimates. The table also shows the difference between Pre- and Post-CAAA estimates for each projection year. Overall, the results of this analysis indicate that the 1990 Amendments will provide a reduction in Hg emissions of 44.2 tons per year (tpy) in the year 2000 and a reduction of 56.2 tpy in 2010. These changes represent a 35 percent reduction in airborne mercury emissions for the year 2000 and a 42 percent reduction for 2010. We estimate that most of the reduction will be the result of New Source Performance Standards for MWI and MWCs.

**Table 2-4**
**Airborne Mercury Emission Estimates**

| | | 2000 Emissions (tons) | | | 2010 Emissions (tons) | | |
|---|---|---|---|---|---|---|---|
| **Source Category** | **1990 Emissions (tons)** | **Pre-CAAA** | **Post-CAAA** | **Diff.** | **Pre-CAAA** | **Post-CAAA** | **Diff.** |
| Medical Waste Incin. | 50 | 17.9 | 1.3 | 16.6 | 22.6 | 1.6 | 21.0 |
| Municipal Waste Comb. | 54 | 31.2 | 5.5 | 25.7 | 33.8 | 6.0 | 27.8 |
| Electric Utility Generation | 51.3 | 63.0 | 61.1 | 1.9 | 68.5 | 65.4 | 3.1 |
| Hazardous Waste Comb. | 6.6 | 6.6 | 6.6 | 0 | 6.6 | 3.0 | 3.6 |
| Chlor-Alkali Plants | 9.8 | 6.0 | 6.0 | 0 | 2.0 | 1.3 | 0.7 |
| **Total CAAA Benefits (Reductions)** | | | | **44.2** | | | **56.2** |

[15] With the exception of electric utility plant Hg emissions that were estimated using IPM, we relied on previously generated estimates (typically from recently conducted RIAs) to evaluate the impact of the CAAA on Hg emissions. For a more complete discussion of the methodology, see Appendix A.

**Figure 2-1**
**Pre- and Post-CAAA Scenario VOC Emissions Estimates**

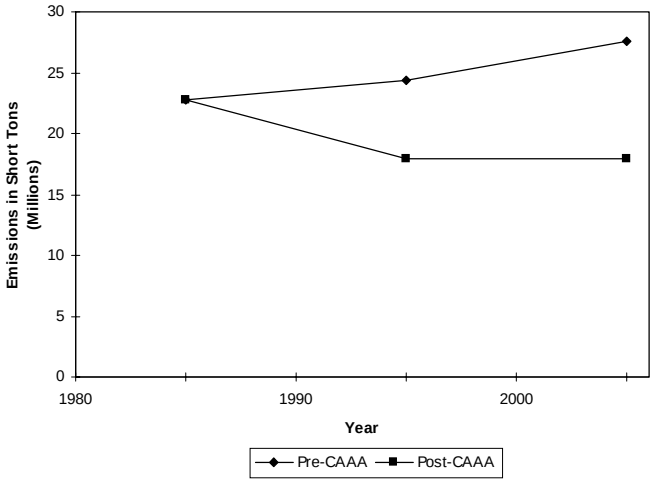



**Figure 2-2**
**Pre- and Post-CAAA Scenario $NO_X$ Emissions Estimates**

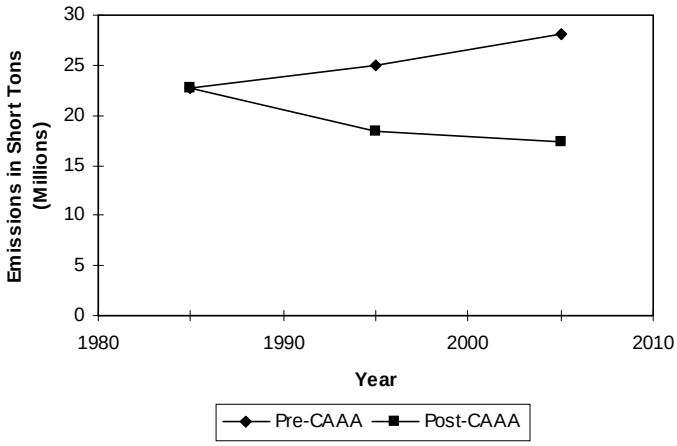



**Figure 2-3**
**Pre- and Post-CAAA Scenario $SO_2$ Emissions Estimates**

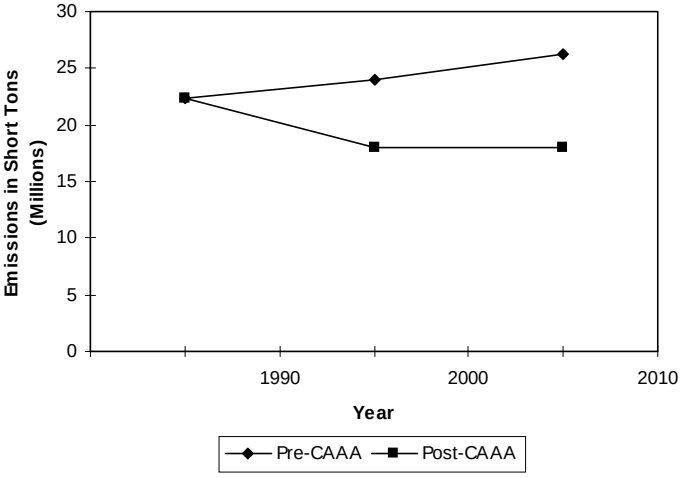


**Figure 2-4**
**Pre- and Post-CAAA Scenario CO Emissions Estimates**

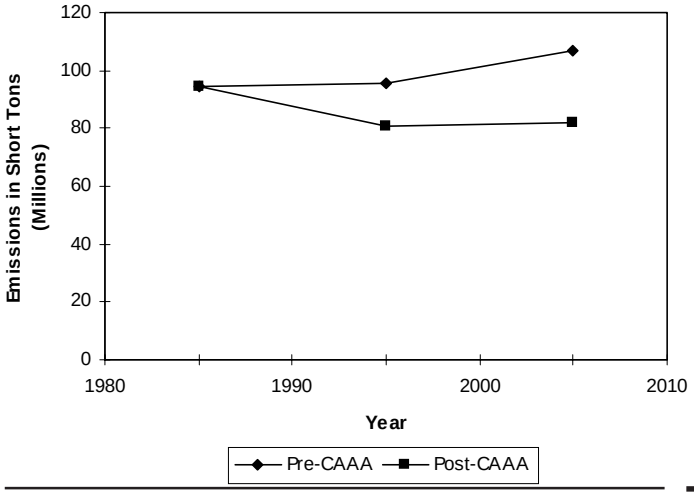



**Figure 2-5**
**Pre- and Post-CAAA Scenario Primary $PM_{10}$ Emissions Estimates**

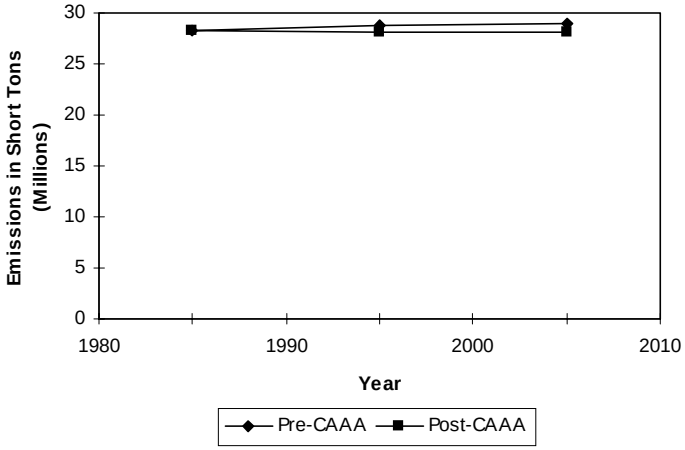
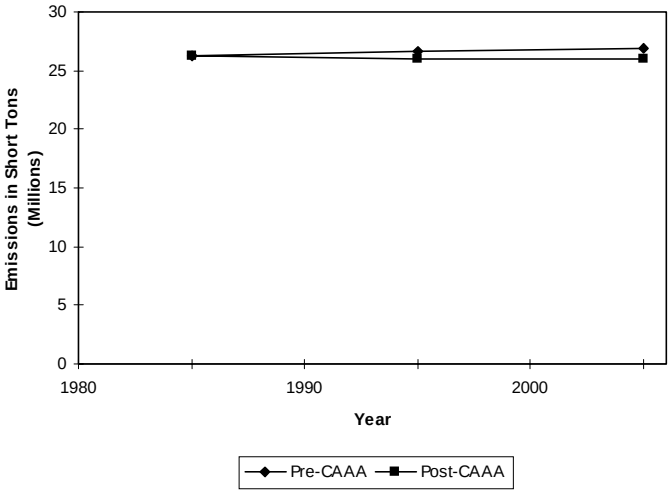



**Figure 2-6**
**Pre- and Post-CAAA Scenario Primary $PM_{2.5}$ Emissions Estimates**



## Comparison of Emissions Estimates With Other Existing Data

Comparison of the emissions projections generated by the prospective analysis to historical emissions estimates drawn from the National Air Pollutant and Emissions Trends reports (*Trends*) provides a check on the reasonableness of our emissions inventories. In addition, comparison of emissions projections from the prospective analysis with those of the Grand Canyon Visibility Transport Commission (GCVTC) study of western regional haze provides an initial test of the sensitivity of emissions projections to base-year inventories and growth assumptions. Analysis of PM emissions and comparison of estimated and observed PM data also help us evaluate the prospective study's emissions estimation methods.

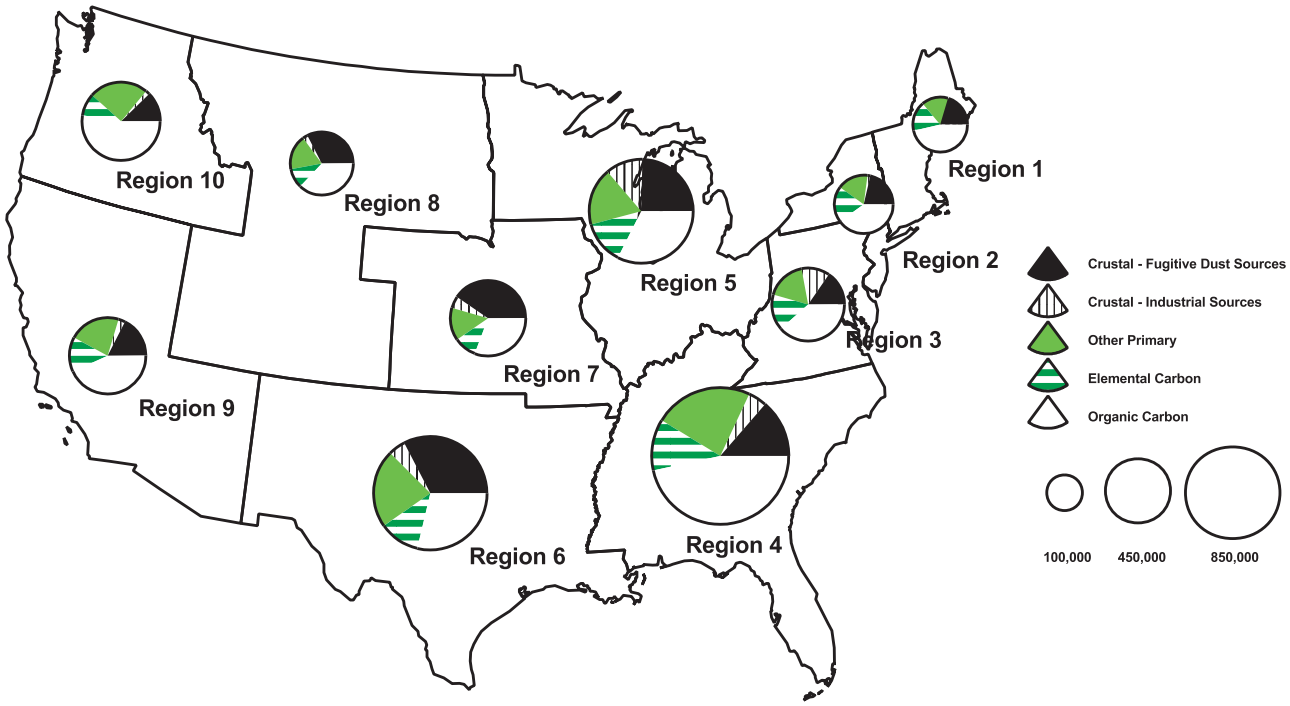
*Trends* reports contain historical estimates of annual VOC, $NO_x$, $SO_2$, CO, and $PM_{10}$, emissions. While the most recent report only provides emissions data through the first half of the 1990s, comparison of these estimates from 1990 to 1996 with emissions trends projected under the Post-CAAA scenarios reveals that emissions figures from both are similar. The disparity that does exist between the two sets of estimates largely stems from the fact that the Post-CAAA scenario trend lines running from 1990 to 2000 consist of only two data points. As a result, Post-CAAA trend lines cannot capture yearly fluctuations in emissions and the exact timing of emissions cuts. Only for $NO_x$ are the *Trends* and Post-CAAA estimates significantly different; this is because the *Trends* report is still in the process of incorporating the State's periodic emission inventory into the NET database. As a result, *Trends* values do not capture all the $NO_x$ emission reductions that have occurred since 1990. For example, significant reductions attributable to reasonable available control technology (RACT) requirements for major stationary source $NO_x$ emitters areas are not reflected in the *Trends* figures.

The Grand Canyon Visibility Transport Commission conducted an air pollution analysis for Western States that projected emissions for selected pollutants, including $NO_x$, $SO_2$, and $PM_{2.5}$, from 1990 base-year levels for the year 2000 and every tenth subsequent year up to 2040. GCVTC estimates of future-year emissions levels differ from Post-CAAA projections. This disparity results from the use of different base-year inventories in the two studies and from specific regional reductions not incorporated in the prospective analysis scenarios. Despite the difference in GCVTC and Post-CAAA estimates, the change in the level of emissions from 1990 to 2010 predicted by the two studies is similar. Comparison of both sets of projections illustrates the sensitivity of future-year emissions estimates to the base-year inventory.

The 1997 National Air Quality and Emissions Trends Report provides a summary of $PM_{2.5}$ concentration speciation data. This report shows the relative contribution of the major PM emissions source components (crustal material, carbonaceous particles, nitrate, and sulfate) to ambient $PM_{2.5}$ concentrations in urban and nonurban areas throughout the U.S.[16] Comparison of primary $PM_{2.5}$ emissions estimates generated for this analysis with the observed concentration data presented in the 1997 report indicates that the ratio in the prospective study of crustal material to primary carbonaceous particles is high. At least part of this apparent overestimation of crustal material and underestimation of carbonaceous particulates, however, is due to the fact that much of the emitted crustal material quickly settles and does not have a quantifiable impact on ambient air quality. In this analysis, we apply a factor of 0.2 to crustal emissions to estimate the fraction of crustal $PM_{2.5}$ that makes its way into the "mixed layer" of the atmosphere and influences pollutant concentrations. Figure 2-7 displays the breakout of primary $PM_{2.5}$ into its adjusted crustal and carbonaceous (elemental carbon and organic carbon) components. The figure divides crustal material into two subcategories, fugitive dust or industrial sources, based on the source of the material and also shows the fraction of primary $PM_{2.5}$ that is

[16] Crustal material is directly emitted from fugitive dust sources such as agricultural operations, construction, paved and unpaved roads, and wind erosion as well as from some industrial sources such as metals processing. Carbonaceous particles, as defined in the 1997 National Air Quality and Emissions Trends Report," are emitted directly and as condensed liquid droplets from fuel combustion, burning of forests, rangelands, and fields; off highway and highway mobile sources (gas and diesel); and certain industrial processes".

**Figure 2-7**
**1990 Primary $PM_{2.5}$ Emissions by EPA Region (tons/year)**




neither crustal nor carbonaceous. The ratios of adjusted crustal material to primary carbonaceous particles presented in Figure 2-7 are in line with the observed $PM_{2.5}$ concentration data presented in the 1997 report.

## Uncertainty In Emission Estimates

Table 2-5 provides a list of sources of uncertainty associated with estimating base-year emissions, the expected direction of bias introduced by each uncertainty (if known), and the relative significance of each uncertainty in the overall 812 benefits analysis. The emissions estimates presented in the prospective analysis are characterized by three major sources of uncertainty: estimation of the base-year inventory, prediction of the growth in pollution-generating activity, and assumptions about future-year controls.

Base-year emissions were estimated using emissions factors that express the relationship between a particular human/industrial activity and the level of emissions. The accuracy of base-year emissions estimates varies from pollutant to pollutant, depending largely on how directly the selected activity and emissions correlate. We likely estimated 1990 $SO_2$ emissions with the greatest precision. Sulfur dioxide emissions are generated during combustion of sulfur-containing fuel and are directly related to fuel sulfur content. In addition, we were able to verify these estimates through comparison with Continuous Emission Monitoring (CEM) data. As a result, we were able to accurately estimate $SO_2$ emissions using emissions factors based on data on fuel usage and fuel sulfur content. Nitrogen oxides are also a product of fuel combustion, allowing us to estimate emissions of this pollutant using the same general technique used to estimate $SO_2$ emissions. However, the processes involved in the formation of $NO_x$ during combustion are more complicated than those involved in the formation of $SO_2$; thus, our $NO_x$ emissions estimates are more variable and less certain than $SO_2$ estimates.

Volatile organic compounds, like $SO_2$ and $NO_x$, are products of fuel combustion; however, these compounds are also a product of evaporation. To estimate evaporative emissions of this pollutant we

used emissions factors that relate changes in emissions to changes in temperature. Because future meteorological conditions are difficult to predict, the uncertainty associated with forecasting temperature influences the uncertainty in our VOC emissions estimates. The likely significance of this uncertainty, in terms of its impact on the overall monetary benefit present in this analysis, is probably minor.

In this analysis we estimated primary $PM_{2.5}$ emissions based on unit emissions that may not accurately reflect the composition and mobility of particles. The ratio of crustal to carbonaceous particulate material, for example, likely is high as a result of overestimation of the fraction of crustal material, primarily composed of fugitive dust, and underestimation of the fraction of carbonaceous material. Because the CAAA has a greater impact on emissions sources that generate carbonaceous particles (mobile sources) than on sources that mainly emit crustal material (area sources), we likely underestimate the impact of the CAAA on reducing $PM_{2.5}$, thereby reducing monetary benefits estimates. The uncertainty associated with estimating the partition of $PM_{2.5}$ emissions components could conceivably have a major impact on the net benefit estimate; compared to secondary $PM_{2.5}$ precursor emissions, however, changes in primary $PM_{2.5}$ emissions have a relatively small impact on $PM_{2.5}$ related benefits.

We estimated future-year emissions levels based on expected growth in pollution-generating activities. Inherent uncertainties and data inadequacies/limitations exist in forecasting growth for any future period. Also, the growth indicators we used in this analysis may not directly correlate with changes in the factors that influence emissions. Both of these factors contribute to the uncertainty associated with this study's emissions results. For example, the best indicator of pollution-generating activity is fuel use or some other measure of input/output that most directly relates to emissions. The key BEA indicator used in this analysis, GSP, is closely correlated with the pollution-generating activity associated with many manufacturing industry processes (iron and steel, petroleum refining, etc.). However, a good portion of industrial sector emissions are from boilers and furnaces, whose activity is related to production, but not as closely as to product output. Activities such as fuel switching may produce different emission patterns than those reflected in the results of this study.

Our future-year control assumptions are also a source of uncertainty. Despite our efforts to minimize this uncertainty, whether each of the Post-CAAA controls will be adopted, whether Post-CAAA control programs will be more or less effective than estimated, and whether unanticipated technological shifts will reduce future-year emissions are all unknown. For example, the Post-CAAA scenario includes implementation of a region-wide $NO_x$ control strategy designed to regulate the regional transport of ozone. However, the control program assumed under the Post-CAAA scenario may not reflect the $NO_x$ controls that are actually implemented in a regional ozone transport rule.

**Table 2-5**
**Key Uncertainties Associated with Emissions Estimation**

| Potential Source of Error | Direction of Potential Bias for Net Benefits Estimate | Likely Significance Relative to Key Uncertainties in Net Benefit Estimate* |
|---|---|---|
| $PM_{2.5}$ emissions are largely based on scaling of $PM_{10}$ emissions. | Overall, unable to determine based on current information, but current emission factors are likely to underestimate $PM_{2.5}$ emissions from combustion sources, implying a potential underestimation of benefits. | Potentially major. Source-specific scaling factors reflect the most careful estimation currently possible, using current emissions monitoring data. However, health benefit estimates related to changes in $PM_{2.5}$ constitute a large portion of overall CAAA-related benefits. |
| Primary $PM_{2.5}$ emissions estimates are based on unit emissions that may not accurately reflect composition and mobility of the particles. For example, the ratio of crustal to primary carbonaceous particulate material likely is high. | Underestimate. The effect of overestimating crustal emissions and underestimating carbonaceous when applied in later stages of the analysis, is to reduce the net impact of the CAAA on primary $PM_{2.5}$ emissions by underestimating $PM_{2.5}$ emissions reductions associated with mobile source tailpipe controls. | Potentially major. Mobile source primary carbonaceous particles are a significant contributor to public exposure to $PM_{2.5.}$ Overall, however, compared to secondary $PM_{2.5}$ precursor emissions, changes in primary $PM_{2.5}$ emissions have only a small impact on $PM_{2.5}$ related benefits. |
| The Post-CAAA scenario includes implementation of a region-wide $NO_x$ emissions reduction strategy to control regional transport of ozone that may not reflect the $NO_x$ controls that are actually implemented in a regional ozone transport rule. | Unable to determine based on current information. | Probably minor. Overall, magnitude of estimated emissions reductions is comparable to that in expected future regional transport rule. In some areas of the 37 state region, emissions reductions are expected to be overestimated, bur in other areas, $NO_x$ inhibition of ozone leads to underestimates of ozone benefits (e.g., some eastern urban centers). |
| VOC emissions are dependent on evaporation, and future patterns of temperature are difficult to predict. | Unable to determine based on current information. | Probably minor. We assume future temperature patterns are well characterized by historic patterns, but an acceleration of climate change (warming) could increase emissions. |
| Use of average temperatures (i.e., daily minimum and maximum) in estimating motor-vehicle emissions artificially reduces variability in VOC emissions. | Unable to determine based on current information. | Probably minor. Use of averages will overestimate emissions on some days and underestimate on other days. Effect is mitigated in Post-CAAA scenarios because of more stringent evaporative controls that are in place by 2000 and 2010. |

**Table 2-5 (continued)**
**Key Uncertainties Associated with Emissions Estimation**

| Potential Source of Error | Direction of Potential Bias for Net Benefits Estimate | Likely Significance Relative to Key Uncertainties in Net Benefit Estimate* |
|---|---|---|
| Economic growth factors used to project emissions are an indicator of future economic activity. They reflect uncertainty in economic forecasting as well as uncertainty in the link to emissions. | Unable to determine based on current information. | Probably minor. The same set of growth factors are used to project emissions under both the Pre-CAAA and Post-CAAA scenarios, mitigating to some extent the potential for significant errors in estimating differences in emissions. |
| Uncertainties in the stringency, scope, timing, and effectiveness of Post-CAAA controls included in projection scenarios. | Unable to determine based on current information. | Probably minor. Future controls could be more or less stringent, wide-reaching (e.g., $NO_x$ reductions in OTAG region - see above), or effective (e.g., uncertainty in realizing all Reasonable Further Progress requirements) than projected. Timing of emissions reductions may also be affected (e.g., sulfur emissions reductions from utility sources have occurred more rapidly than projected for this analysis). |

* The classification of each potential source of error reflects the best judgement of the section 812 Project Team. The Project Team assigns a classification of "potentially major" if a plausible alternative assumption or approach could influence the overall monetary benefit estimate by approximately five percent or more; if an alternative assumption or approach is likely to change the total benefit estimate by less than five percent, the Project Team assigns a classification of "probably minor."

# Direct Costs

Chapter 3

The costs of complying with the requirements of the Clean Air Act Amendments (CAAA) of 1990 will affect all levels of the U.S. economy. The impact, initially experienced through the direct costs imposed by regulations promulgated under the amendments, will also be seen in patterns of industrial production, research and development, capital investment, productivity, employment, and consumption. The purpose of the analysis summarized in this chapter is to estimate the incremental change in annual compliance costs from 1990 to 2010 that are directly attributable to the 1990 Clean Air Act Amendments.

This chapter consists of four sections. The first section summarizes our approach to estimating direct compliance costs. In the second section we present the results of the cost analysis. We first report the total costs of Titles I through V and then present estimates for major individual provisions. We also briefly discuss our derivation of Title VI costs. In the third section, we provide a qualitative discussion of the potential magnitude of social costs and other impacts associated with the Amendments to characterize the potential welfare loss not captured in the direct cost approach. We conclude the chapter with a discussion of the major analytic uncertainties and include the results of quantitative sensitivity tests of key data and assumptions.

## Approach to Estimating Direct Compliance Costs

As discussed in the previous chapter, the first step of the prospective analysis required the development of emission estimates for the base-year, 1990, and for the two target years in our analytic time period, 2000 to 2010. We developed two scenarios, Pre-CAAA and Post-CAAA, that reflect three key parameters: (i) base-year inventory selection, (ii) indicators of forecasted economic growth, and (iii) effects of future year controls and selected CAAA provisions. The Pre-CAAA scenario applies the stringency and scope of air pollution regulations as they existed in 1990 and projects emissions and costs to 2000 and 2010. This scenario establishes a baseline that represents projected emission levels and control costs in the absence of the 1990 Amendments. Under the Post-CAAA scenario, costs are based on compliance with selected CAAA provisions. Together these two scenarios form the foundation upon which the incremental costs and benefits of complying with the 1990 Amendments are estimated. For more information on the development of these scenarios, see Chapter 2.

We closely integrate the modeling of direct compliance costs with emissions projections by maintaining consistency among control assumptions (i.e. emissions scenarios) used as inputs in the cost estimation modeling and in the analysis of emissions projections and benefits. We use two models to estimate costs, Emission Reduction and Cost Analysis Model (ERCAM) and Integrated Planning Model (IPM). These models generate cost estimates for the Post-CAAA scenarios in two projection years, 2000 and 2010. The estimates are calculated relative to costs under the same year Pre-CAAA scenario, so estimates represent incremental costs of compliance with the 1990 Amendments.

We use ERCAM to estimate costs associated with regulating particulate matter (PM), volatile organic compounds (VOCs), and non-utility source oxides of nitrogen ($NO_x$).[1] The model is essentially a cost-accounting tool that provides a structure for modifying and updating changes in inputs while main-

[1] This model was developed by E. H. Pechan & Associates, Inc. to facilitate EPA's analysis of emissions control.

taining consistency with the emission and cost analyses. Cost scenarios and assumptions are developed for each non-utility source category (e.g., point, area, nonroad, and motor vehicle sources) and in response to specific provisions and emission targets. The model estimates costs based on inputs such as cost per ton, source-specific cost equations, incremental production, and operating cost estimates. For this analysis, we collected data and inputs from information presented in regulatory impact assessments (RIAs), background information documents (BIDs), regulatory support documents, and Federal Register notices.

To estimate the costs of reducing utility $NO_x$ and sulfur dioxide ($SO_2$) emissions, we use the Integrated Planning Model (IPM). IPM allows us to estimate the control costs of several pollutants while maintaining consistent control scenarios and economic forecasts of the electric power industry. It assesses the optimal mix of pollution control strategies subject to a series of specified constraints. Key inputs and constraints in the model include targeted emissions reductions (on a seasonal or annual basis), costs and constraints of control technology, and economic parameters (e.g., forecasted demand for electricity, power plant availability/capacity, costs of fuel, etc.)

To assess the costs of reducing emission of pollutants or sectors not covered by our two models, we estimate costs using the best available cost equations or other types of analyses. For example, we estimate non-utility $SO_2$ emission control costs for point sources by applying source-specific cost equations for flue gas desulfurization (FGD)/scrubber technology to affected sources in 2000 and 2010. While we do not explicitly model CO attainment costs, we include in the analysis the costs of programs designed to reduce CO emissions, such as oxygenated fuels and a cold temperature CO motor vehicle emission standard. Finally, to estimate costs of the rate of progress/reasonable further progress (ROP/RFP) provisions, requirements under Title I that require ozone nonattainment areas to make steady progress toward attainment, we first estimate the emissions reduction shortfall that must be achieved in each target year in each nonattainment area, and then apply a cost per ton estimate from a schedule of measures that could be applied locally to meet the necessary ROP/RFP requirement. For more detail on the specific methods used to estimate compliance costs for each pollutant and source category, see Appendix B.

The cost estimates in this chapter are the incremental costs of the 1990 Amendments (i.e. the difference between pre- and Post-CAAA cost estimates). We present the results as total annualized costs (TAC) in 2000 and 2010. Annualized costs include both capital costs, such as costs of control equipment, and operation and maintenance (O&M) costs.[2] They do not represent actual cash flow in a given year, but are rather an estimate of average annual burden over the period during which firms will incur costs. In annualizing costs, we convert total capital investment to a uniform series of total per-year equivalent payments over a given time period using an assumed real cost-of-capital at five percent. We then add O&M and other reoccurring costs to the annualized capital cost to arrive at TAC. The discounted sum of these annual expenditures is equal to the net present value of total costs incurred over the time period of this analysis.[3]

## Direct Compliance Cost Results

Total annual compliance costs for Titles I through V of the 1990 Amendments in the year 2000 will be approximately $19.4 billion; the estimate increases to $26.8 billion in the year 2010. These costs reflect "annualized" operation and maintenance (O&M) expenditures (which includes research and development (R&D) and other similarly recurring expenditures) plus amortized capital costs (i.e., depreciation plus interest costs associated with the ex-

[2] For a few VOC source categories, we estimate that capital investment will not be necessary; for these sources, compliance costs reflect O&M costs only.

[3] We recalculate the control cost estimates from regulatory documents that use a seven or ten percent discount rate so that the costs will be consistent with the five percent discount rate assumption used in this analysis. We also calculate cost using three percent and seven percent discount rates, as sensitivity tests; for detail see the discussion of uncertainty later in this chapter, in Chapter 8, and in Appendix B.

isting capital stock) for the particular year.[4] We present cost estimates by title and emissions source category (point sources, area sources, utilities, nonroad engines and vehicles, and motor vehicles) in Table 3-1.

In some cases, assigning costs to a single CAAA title is complicated by the fact that there are rules issued pursuant to more than one title.[5] In addition, with the passage of the 1990 Amendments, the States were given greater discretion in developing CAAA compliance strategies. For example, the States can determine how best to meet progress requirements and are responsible for creating permit programs (under Title V). As a result, a significant portion of the costs also represent State-level strategies and decisions for reducing emissions.

Title I, Provisions for Attainment and Maintenance of National Ambient Air Quality Standards (NAAQS), represents pollution controls (of VOC, $NO_x$, and PM emissions) implemented primarily by point and area sources. Title I provisions also account for State programs designed to meet progress requirements. By 2010, we project the costs of Title I provisions will account for over half of total CAAA direct compliance costs ($14.5 billion). An additional 34 percent of estimated total costs ($9 billion) is attributed to regulating mobile source emissions under Title II. Collectively, the combined direct compliance costs of these two titles is $16 billion in 2000 and $23 billion by 2010.

The remaining three titles account for less than 20 percent of total CAAA direct costs. We estimate that Title III provisions, which target hazardous air pollutant (HAP) emissions, will cost $840 million by the year 2010. This estimate represents total annualized capital costs (TACs) for individual two- and four-year MACT standards. While the majority of this estimated cost reflects reducing VOC emissions (since HAP emissions were not included as part of the Section 812 base- year inventory), Title III costs do include some costs of final MACT rules that regulate non-VOC HAP emissions.

In order to estimate the costs associated with Title IV, we considered the implications of pollution abatement controls (for $SO_2$ and $NO_x$) on the electric power industry's operation of generation units and how, over time, this would affect the demand for electricity. The annual compliance estimate for Title IV costs is $2.3 billion in 2000. This estimate decreases to $2.0 billion by 2010. This decrease reflects, in part, the future compliance cost savings resulting from the $SO_2$ allowance trading program.

Title V costs are associated with new operating permit programs. The estimate accounts for approximately one percent of total costs projected under the Post-CAAA 2010 scenario. States are expected to implement Title V permit programs by 2005. The estimate reflects the costs of State-developed programs during the first five-year implementation period. These costs include incremental administrative costs incurred by the permitted sources, State and local permitting agencies, and EPA. The estimate excludes federally-implemented State programs and state programs which were already established in the baseline.

Our presentation of cost estimates for the stratospheric ozone protection provisions of Title VI is, by necessity, different from other titles. Ideally, one should compare the costs of actions taken in a given year to the benefits attributable to these actions. For Title VI, a cost-benefit comparison of any given year requires assumptions that result in potentially misleading figures. The difficulty is due to the differing time horizons and the complexity of the process by which ozone-depleting substances (ODSs) cause adverse effects on human health and the environment. Title VI provisions incur costs over significantly varying time horizons; for example, the cost analysis of Sections 604 and 606 provisions spans 85 years (from 1990 to 2075). At the same time, the analysis of Section 611 extends from 1994 to 2015. In response to this analytic difficulty, we base our comparison of Title VI costs to Title VI benefits on net present values.

[4] Capital expenditures are investments, generating a stream of benefits and opportunity costs over an investment's lifetime. In a cost-benefit analysis, the appropriate accounting technique is to annualize capital expenditures. This technique involves spreading the costs of capital equipment uniformly over the useful life of the equipment, by using a discount rate to account for the time value of money. In this analysis, all capital expenditures were annualized using a real five percent interest rate.

[5] In those cases, we generally assigned costs to a single title based upon implementation dates and the year by which emission reductions are expected.

**Table 3-1**
**Summary of Direct Costs for Titles I to V of CAAA, By Title and Selected Provisions**
(Annual Costs in million 1990$)

| Title/Provision | Primary Cost Estimate 2000 | Percentage of Total Costs | Primary Cost Estimate 2010 | Percentage of Total Costs |
|---|---|---|---|---|
| ***Title I- Provisions for Attainment and Maintenance of NAAQS*** | | | | |
| Stationary $NO_x$ Controls, Utility Industry | $ 790 | 4% | $ 2,500 | 9% |
| Progress Requirements | 1,200 | 6% | 2,500 | 9% |
| PM NAAQS Controls | 1,900 | 10% | 2,200 | 8% |
| California LEV | 320 | 2% | 1,100 | 4% |
| National LEV | 180 | 1% | 1,100 | 4% |
| High Enhanced I/M | 1,100 | 6% | 1,400 | 5% |
| Other Title I Programs | 3,100 | 16% | 3,700 | 14% |
| Title I: Total Costs | $ 8,600 | 44% | $ 14,500 | 54% |
| ***Title II- Provisions Relating to Mobile Sources*** | | | | |
| California Reformulated Gasoline | $ 2,000 | 10% | $ 2,400 | 9% |
| $NO_x$ Tailpipe/Extended Useful Life Standard | 1,500 | 8% | 1,700 | 6% |
| Other Title II Programs | 3,900 | 20% | 4,900 | 18% |
| Title II: Total Costs | $ 7,400 | 38% | $ 9,050 | 34% |
| ***Title III- Hazardous Air Pollutants*** | | | | |
| Title III: Total Costs | $ 780 | 4% | $ 840 | 3% |
| ***Title IV- Acid Deposition Control*** | | | | |
| Title IV: Total Costs | $ 2,300 | 12% | $ 2,040 | 8% |
| ***Title V- Permits*** | | | | |
| Title V: Total Costs | $ 300 | 2% | $ 300 | 1% |
| **Total Annual Cost** | $ 19,400 | 100% | $ 26,800 | 100% |

Note: Totals may not sum due to rounding. Only major provisions are listed under each title - other, less costly provisions not listed here are nonetheless included in the totals by title and the overall total.

The net present value of Title VI program costs reflect selected actions and their associated costs from Sections 604, 606, 608, 609, and 611. Examples of these actions include: replacement of ozone-depleting chemicals with alternative technologies and materials; recycling and storage of unused chlorofluorocarbons; labeling; training; and administration. Using a discount rate of five percent and a 85-year time horizon (from 1990 to 2075), we estimate the net present value of Title VI costs is $27 billion. For illustrative purposes, we calculated an annualized estimate of Title VI costs. It is, however, important to recognize that these estimates may overestimate actual compliance costs in those years, especially in the year 2000, because of the phased nature of implementation— see Appendix G for more details. Our annualized estimate of total Title VI costs is $1.4 billion. This value reflects an annualized equivalent value of costs incurred over 85 years (from 1990 to 2075) using a five percent discount rate.

## Selected Provisions

Our analysis indicates eight provisions will account for approximately 54 percent of the total direct compliance costs estimate for 2010. Six are Title I provisions that affect stationary sources and vehicle

emissions. The remaining two provisions target mobile sources under Title II. These provisions are:

- PM NAAQS controls[6],
- Electric power industry compliance (stationary $NO_x$ control),
- Progress Requirements,
- California Low Emission Vehicle (LEV)program,
- National Low Emission Vehicle (LEV) program,
- High Enhanced Inspection and Maintenance (I/M) program,
- California Reformulated Gasoline, and
- $NO_x$ Tailpipe/Extended Useful Life Standard.

The 1990 CAAA regulates stationary source emissions primarily under Title I. Among the relevant provisions, PM NAAQS, utility industry compliance with $NO_x$ standards, and progress requirements are the main sources of Title I costs. From 2000 to 2010, we estimate the control costs of all three provisions will increase by at least a factor of two. Under the Post-CAAA scenario developed for the emissions analysis, the utility industry's compliance with $NO_x$ emission standards affects all electric generation units using fossil fuels. Existing oil and gas units face Reasonable Available Control Technology (RACT) requirements and all new units must comply with more stringent New Source Performance Standards (NSPS) and New Source Review (NSR) requirements. By 2010, estimated costs for stationary $NO_x$ controls more than triple ($790 million to $2,500 million). The cost estimate indicates that the provision will be the single largest source of CAAA direct costs. The second largest component of total costs in 2010 is attributed to progress requirements. Annual compliance costs with progress requirements double from 2000 to 2010 ($1.2 billion and $2.5 billion, respectively). Among the three provisions, the annual costs associated with PM NAAQS compliance exhibits the least amount of growth. We estimate annual costs for PM NAAQS compliance will grow from $1.9 billion in 2000 to $2.2 billion in 2010.

[6] We estimate the PM NAAQS provision costs based on compliance with standards that were in effect prior to 1997 revisions (62 Fed. Reg. 38,652, 1997).

Among the provisions regulating vehicle emissions, only the national and California LEV programs exhibit a trend of increasing direct costs of the same magnitude as seen with the costs of regulating stationary sources under Title I. The combined cost of national and California LEV programs is $2.2 billion in 2010. For the California LEV program, the increase in cost is largely a function of higher per vehicle cost estimates (e.g., zero emission vehicles (ZEV) are mandated in the year 2003). Our cost analysis of the national LEV program assumes that only the Northeast Ozone Transport Region (OTR) states will incur costs in the year 2000. By 2010, however, we expect that the program will affect areas outside of the OTR. As a result, 2010 national LEV costs increase with the expected expansion and increased volume of vehicle sales. Unlike many of the other provisions, high enhanced I/M costs do not exhibit significant growth from 2000 to 2010. We estimate this provision accounts for approximately six percent of total costs in 2000 and five percent in 2010. These costs, however, are uncertain pending State decisions regarding the design of their programs.

Among the analyzed Title II provisions, we attribute nearly 15 percent of total annual direct costs to the California reformulated gasoline (RFG) program and $NO_x$ Tailpipe/Extended Useful Life Standard. Although the reformulated gasoline program affects only California, the state accounts for nearly ten percent of annual gasoline sales in the United States. We estimate compliance costs of $1.9 billion in the year 2000. As the program enters Phase 2, estimated costs grow to $2.4 billion. The trend in cost associated with $NO_x$ Tailpipe/Extended Useful Life Standard is very different. While costs increase slightly between the years 2000 and 2010, the provision's share of total cost slightly decreases.

## Characterization of Other Economic Impacts

In an ideal setting, a cost-benefit analysis would not only identify, but also quantify and monetize, an exhaustive list of social costs associated with a regulatory action. This would include assessing how regulatory actions targeting a specific industry or set of facilities can alter the level of production and consumption in the directly affected market and related

markets. For example, regulation of emissions from the electric utility industry that results in higher electricity rates would have both supply-side and demand-side responses. In secondary markets, the increased electricity rates affect production costs for various industries and initiate behavioral changes (e.g., using alternative fuels as a substitute for electric power). With each affected market, there are also associated externalities that should be included in estimating social costs. Returning to the utilities example, the externalities associated with electric power generation versus nuclear power generation can be very different. The mix of externalities could change as consumers substitute nuclear power for electric power. It is frequently difficult to accurately characterize one or all of these dimensions of market responses and estimate the resulting social costs.

There are three generally practiced approaches to calculating costs associated with regulation: (i) direct compliance cost, (ii) partial equilibrium modeling, and (iii) general equilibrium modeling. Direct compliance cost estimates are calculated differently than the economic welfare impact estimates that result from partial or general equilibrium modeling; a direct cost estimate is often the most straightforward of the three approaches. This method estimates compliance expenditures or, in economic terms, how an industry's or firm's marginal cost curve shifts due to increased production costs associated with regulatory compliance. As a result, this method does not account for firm responses and market responses, such as adjustment of production levels and product prices. The other two methods measure changes in producer and consumer welfare, and incorporate these types of adjustments.

The direct cost approach likely overstates actual compliance expenditures, but may have an ambiguous relationship to total social costs. There are two primary reasons for the overstatement of compliance expenditures. First, the direct cost approach does not account for market responses. As a result, total direct cost estimates reflect the incremental cost per unit of output multiplied by the generally higher, pre-regulation quantity produced. Second, a direct cost approach tends to make the simplifying assumption that firms rely on static pollution abatement technology, when in fact the presence of compliance costs provides an incentive to innovate. Several *ex post* cost analyses suggest that the marginal cost curve may not necessarily shift by the full amount of the pollution abatement. For example, firms may respond by altering production processes to more efficiently reduce emissions.[7] Social cost estimates, however, may include other costs not reflected in direct cost estimates (discussed below), thereby offsetting the tendency for direct cost estimates to overstate expenditures.

Measuring net welfare changes due to regulatory action requires either partial or general equilibrium modeling. These more complicated approaches estimate social costs by accounting for a wider range of market consequences associated with compliance with pollution abatement requirements. The partial equilibrium approach is particularly appropriate when social costs are predominantly incurred in directly affected markets. It requires modeling both supply and demand functions in the affected economic sector. Therefore, measures of social cost reflect behavioral responses by both producers and consumers in a specific market and do not necessarily reflect how those changes affect related markets.

In cases where the regulatory action is known to have an impact on many sectors of the US economy, the general equilibrium model is a more appropriate approach to estimating social costs. Like the partial equilibrium model, the general equilibrium model estimates social costs by accounting for direct compliance costs and producer and consumer market behavior. The general equilibrium model can capture first-order effects that occur in multiple sectors of the economy, and may also provide insight into unanticipated indirect effects in sectors that might not have been included in the scope of a partial equilibrium analysis.

The relationship of general equilibrium estimates to estimates from the other two cost approaches is not always clear. General equilibrium estimates have a broader basis from which to estimate social costs and can reflect the net welfare changes across the full range of economic sectors in the U.S. Partial equilibrium modeling tends to understate full social costs because of its restricted scope (i.e., generally limited to one industry). Total direct cost estimates are likely to overstate costs in the primary market because they do not reflect consumer and producer responses. This is demonstrated in comparisons of

[7]Morgenstern *et al.* (1998) estimate the ratio of incurred abatement expenditures to estimated direct costs can be as low as 0.8.

estimates generated using a direct cost approach and a partial equilibrium approach. The extent to which a direct cost estimate will overstate or understate a social cost estimate from a general equilibrium model depends on the magnitude of the "ripple effects" in economic sectors not targeted by a regulation.[8]

In the 812 retrospective analysis (EPA, 1997), we recognized that the Clean Air Act has a pervasive impact on the US economy and opted for the general equilibrium approach. The retrospective nature of the analysis, however, provided us with fairly well-developed historical data sets of goods and service flows throughout the economy. These data sets facilitated the development of detailed, year-by-year expenditures in all sectors of the economy, from which we modeled producer and consumer behavior and estimated net social costs. In the retrospective, our central estimate of total annualized direct costs, from 1970 to 1990, was $523 billion. In comparison, we estimated the aggregate welfare effects to be between $493 and $621 billion.[9]

For the prospective analysis, however, we adopt a direct compliance cost approach. Although the general equilibrium approach may represent a more theoretically preferable method for measuring social costs, we use the simpler direct cost modeling method for three reasons:

- First, we believe that the direct cost approach provides a good first approximation of the CAAA's economic impacts on various sectors the U.S. economy. Comparison of the direct cost approach to the partial equilibrium modeling suggests that the direct cost approach likely overstates costs to the entity that incurs the pollution control cost expenditure. As discussed earlier, the direct cost approach does not reflect adjustments to prices and quantities that might mitigate the effects of regulation. Recent research analyzing ex ante and ex post cost estimates of regulations suggests that *ex ante* analyses are far more likely to overstate than understate costs.[10] However, direct cost estimates may also understate the effects of long-term changes in productivity and the ripple effects of regulation on other economic sectors that are captured by a general equilibrium approach. The magnitude of those other effects, including potential magnification of social costs by existing tax distortions, may be substantial.

- Second, we believe that the closer approximation of social costs that might be gained through a general equilibrium approach could be compromised by the difficulty and uncertainty associated with projecting future economic and technological changes. The general equilibrium approach could provide many insights that the direct cost approach cannot, but also introduces a significant level of additional uncertainty.

- Third, the focus of the present analysis is a comparison of direct costs and direct benefits. To provide a balanced treatment of costs and benefits in a general equilibrium framework, the social cost model must be designed and configured to reflect the indirect economic consequences of both costly and beneficial economic effects. None of the general equilibrium models available in the timeframe of this study could be configured to support effective analysis of the full range of specific direct costs and, especially, direct benefits of the 1990 Clean Air Act Amendments.

[8] Current regulatory analyses that apply partial equilibrium modeling or general equilibrium modeling tend to measure costs with the assumption that markets are currently operating under optimally efficient conditions. Emerging literature suggests that a full accounting of the social costs and efficiency impacts of environmental regulations could also include an assessment of the incremental costs that reflect existing market distortions, such as those imposed by the current tax code. The distortions introduced by existing taxes, in combination with new regulatory requirements, are collectively referred to as the tax-interaction effect. One of the major conclusions of this emerging literature is that the social cost of environmental policy changes can be substantially higher when pre-existing tax distortions are taken into account. Our direct cost estimates do not reflect quantification of this effect, in part because of the emerging nature of this literature and in part because existing estimates of the magnitude of the tax-interaction effect are calculated as increments to social costs and are not necessarily applicable adjustments to direct cost estimates.

[9] Estimates are in 1990 dollars. The retrospective states, "In general the estimated second order macroeconomic effects were small relative to the size of the U.S. economy." The rate of long term GNP growth between the control and no-control scenarios amounted to roughly one-twentieth of one percent less growth.

[10] See, for example, Harrington *et al* (1999), referenced in Appendix B, for a comparative analysis of ex ante and ex post regulatory cost estimates.

- Fourth, undertaking a general equilibrium modeling exercise remains a very resource-intensive task. For the purposes of comparing costs to benefits we concluded that more detailed modeling would not be the most cost-effective use of the project resources.

## Uncertainty in the Cost Estimates

### Overview

As we note at the beginning of this chapter, explicit and implicit assumptions regarding changes in consumption patterns, input costs, and technological innovation are crucial to framing the question of the CAAA's cost impact. Given the nature of this prospective study, there is no way to verify the accuracy of the assumptions applied to future scenarios. We can envision other plausible analyses with estimates that differ from results in this chapter. Moreover, for many of the factors contributing to uncertainty, the degree or even direction of the bias is unknown or cannot be determined. Nevertheless, uncertainties and/or sensitivities can be identified and in many cases the potential measurement errors can be quantitatively characterized. In this section of the chapter, we first discuss several quantitative sensitivity analyses undertaken to characterize the impact of key assumptions on the ultimate cost analysis. We conclude the chapter with a qualitative discussion of the impact of both quantified and unquantified sources of uncertainty.

### Quantitative Sensitivity Tests

In order to characterize the uncertainty in the cost estimates, we conducted sensitivity analyses on the key parameters and analytic assumptions of six major provisions. The provisions are the following:

- Progress Requirements,
- California Reformulated Gasoline,
- PM NAAQS Controls,
- LEV program (the National and California programs combined),
- Non-utility Stationary Source $NO_x$ Controls, and
- $NO_x$ Tailpipe/Extended Useful Life Standard.

We selected these provisions because they are among the most significant sources of CAAA costs, yet cost estimates for each of the provisions incorporate significant uncertainties. Collectively, these provisions account for nearly 50 percent of total direct compliance cost estimates for 2010. Table 3-2 summarizes the methods we used to conduct the cost sensitivity analyses and the results.

For each test, we developed three estimates for one or more components of costs affecting the total cost estimate for a given provision: (1) a central estimate, equal to the 2010 primary cost estimate reported in this chapter[11], (2) a low estimate; and (3) a high estimate. The low and high estimates assess the potential magnitude of the effect of the component(s) on the provision's costs and consequently, total CAAA costs, using reasonable alternative assumptions for each cost component. For progress requirements, PM NAAQS controls, and stationary source $NO_x$ controls, the cost projections are based on models of future emissions controls. Accurately identifying the set of adopted controls is a key source of uncertainty. For example, cost-effective control measures for complying with progress requirements have not yet been identified and the sensitivity test suggests the potential for substantial variability in progress requirement compliance costs. In the case of motor vehicle provisions, there are two significant sources of uncertainty, projecting future car sales and forecasting accurate per vehicle costs.

The results indicate that the sensitivity of our primary cost estimates (central estimates) is not uniform across provisions. In addition, low and high estimates may vary by as much as a factor of two. These sensitivity analyses demonstrate the potential effect of altering selected assumptions and data. We do not assign probabilities to the likelihood of the alternative. In other words, it would be inappropriate simply to add up the array of low and high estimates to arrive at an overall range of uncertainty around the central estimates, because it is unlikely that a plausible scenario could be constructed where all the estimates are concurrently either at the high

[11] The one exception is the central estimate of progress requirements. Our sensitivity analysis which is based on more recent cost information indicates that our primary estimate is more reflective of a high estimate. See Appendix B for more details.

or low end of their individual plausible ranges. A better interpretation of these results is that uncertainty in key input parameters can have a significant effect on the overall uncertainty of our estimates of direct compliance costs and ultimately the net benefits calculation.

In addition to examining specific provisions, we conducted a sensitivity analysis of the cost of capital used throughout the analysis. Cost estimates presented earlier in this chapter reflect application of a cost of capital (for the purposes of annualizing total capital costs) of five percent. We also examined the effect on cost estimates for those provisions which involve significant capital expenditures and where we could recalculate annualized costs from the available information. These provisions include non-utility and area source estimates for VOC, $NO_x$, and PM control. The alternative estimates use three and seven percent for the cost of capital. Results indicate that cost estimates are only moderately sensitive to the discount rate. The provisions evaluated have a total annualized capital cost of approximately $3 billion in 2010. Varying the cost of capital generated alternative estimates of $2.8 billion (three percent) and $3.1 billion (seven percent).[12]

## Qualitative Analysis of Key Factors Contributing to Uncertainty

There are a wide range of other factors which contribute to uncertainty in the overall cost estimates. In most cases, the effect of these other factors cannot be quantitified, though some may have significant influences on our overall net benefits estimate. We present a summary of these factors in Table 3-3 below, and provide a characterization of the potential effect of each uncertainty on the primary estimate of the net benefits (i.e., if costs are overestimated, net benefits are underestimated). The two most important factors are the potential impact of innovation on the ultimate control costs incurred and the conservative assumptions we made to estimate RFP costs.

The regulatory documents which provide cost inputs to ERCAM and the IPM contain the most recent data available, given existing technological development. Between 2000 and 2010, however, advancements in control technologies will allow sources to comply with CAAA requirements at lower costs. For example, we anticipate technological improvements for complying with the multiple tiers of proposed emission standards during the phase-in of nonroad engine controls will likely lead to reduced costs. In addition, the costs for certain control equipment may decrease over time as demand increases and technology innovation and competition exert downward pressure on equipment prices. For instance, selective catalytic reduction (SCR ) costs have decreased over the past three years as more facilities begin to apply the technology. We also believe that even in the absence of new emission standards, manufacturers will eventually upgrade engines to improve performance or to control emissions more cost-effectively; firms will institute technologies such as turbocharging, aftercooling, and variable-valve timing, all of which improve engine performance.

There is considerable uncertainty surrounding the development of States' control plans for meeting ozone NAAQS attainment requirements. We base the RFP cost estimate on the assumption that ozone nonattainment areas (NAAs) will take credit for $NO_x$ reductions for meeting progress requirements. Additional area-specific analysis would be necessary to determine the extent to which areas find $NO_x$ reductions beneficial in meeting attainment and progress requirement targets. Trading of $NO_x$ for VOC to meet RFP requirements may result in distributions of VOC and $NO_x$ emission reductions which differ from those used in this analysis. In response to these uncertainties, we adopted a conservative strategy for estimating the costs of RFP reductions in the primary analysis. We use a relatively high cost per ton reduced estimate of $10,000 for all required reductions. Since the time we conducted our primary cost analysis more information has emerged suggesting controls could cost much less, perhaps as little as $3,500 (see Table 3-2 and Appendix B for more details). In our sensitivity analysis of this variable, we incorporate the more recent cost per ton estimates. The analysis suggests that the $10,000 per ton reduced may in fact be more repre-

[12] Note that these calculations reflect the use of alternative discount rates to estimate annual costs. The use of alternative rates to calculate the total net present value of costs incurred through the full 1990 to 2010 study period is examined separately in Chapter 8, where we compare total costs to total benefits.

**Table 3-2**
**Results of Quantitative Sensitivity Tests**

| Provision | Primary Cost Estimate in 2010[1] (billions 1990 $) | Strategy for Sensitivity Analysis | Range of Estimates from Sensitivity Test (billions 1990 $) |
|---|---|---|---|
| Progress Requirements | $2.46 | Vary unit costs for unidentified measures | $1.07 - $2.46 (central, $1.15) |
| California Reformulated Gasoline | $2.45 | Vary incremental fuel costs and gasoline sales estimates | $1.4 - $3.5 |
| PM NAAQS Controls | $2.22 | Vary model attainment plan assumptions and cost per ton estimates | $0.09 to $3.35 |
| LEV costs (California and National Combined) | $2.16 | Vary per vehicle costs and projections of vehicle sales | $1.08 - $2.48 |
| Non-Utility Stationary Source $NO_x$ Costs | $2.15 | Vary unit-level cost per ton | $1.1 - $3.2 |
| $NO_x$ Tailpipe/Useful Life Standards | $1.65 | Vary per vehicle costs and vehicle sales data | $0.83 - $2.48 |

Note:
[1] In all cases, except progress requirements, the Post-CAAA 2010 primary cost estimates is equal to the central estimate in the sensitivity analysis. For more details on the sensitivity analysis of progress requirements and other provisions, see Appendix B.

sentative of an upper bound cost estimate, rather than a central estimate as our primary cost analysis reflects. The result of our conservative approach indicates that we may overstate RFP costs by a factor of two in 2010.

One other factor is also worth noting, although its impact is likely to be less important than the previous two factors. Under the 1990 CAAA, EPA created economic incentive provisions in several rules to provide flexibility for affected facilities that comply with the rules. These provisions include banking, trading, and emissions-averaging provisions. Flexible compliance provisions tend to lower the cost of compliance. For example, the emissions-averaging program grants flexibility to facilities affected by the marine vessels rule, the petroleum refinery National Emission Standard for Hazardous Air Pollutants (NESHAP), and the gasoline distribution NESHAP. These facilities can choose which sources to control, as long as they achieve the required overall emissions reduction. In many of the cost analyses, EPA does not attempt to quantify the effect that economic incentive provisions will have on the overall costs of a particular rule. In these cases, to the extent that affected sources use economic incentive provisions to minimize compliance costs, costs may be overstated. The major trading programs authorized under the Amendments, however, governing sulfur and nitrogen oxide emissions reductions from utilities and major non-utility point sources, are reflected in the cost estimates presented here.

**Table 3-3**
**Key Uncertainties Associated with Cost Estimation**

| Potential Source of Error | Direction of Potential Bias for Net Benefits | Likely Significance Relative to Key Uncertainties on Net Benefits Estimate[1] |
|---|---|---|
| Costs are based on today's technologies. Innovations in future emission control technology and competition among equipment suppliers tend to reduce costs over time. | Underestimate | Probably minor. Available evidence suggests that estimates of pollution control costs based on current engineering can substantially overestimate the ultimate cost incurred, resulting in understating net benefits.[2] |
| Uncertainty of final State strategies for meeting Reasonable Further Progress (RFP) requirements. | Underestimate | Probably minor. We apply a conservative estimate for costs of RFP measures. Available evidence for identified RFP measures suggests costs could be as much as 70 percent lower than this value. The bias most likely results in significantly understating net benefits. |
| Errors in emission projections that form the basis of selecting control strategies and costs in both the IPM and ERCAM models. | Unable to determine based on current information | Probably minor. In many cases, emissions reductions are specified in the regulations, suggesting that errors in the estimation of absolute levels of emissions under Pre- and Post-CAAA scenarios may have only a small impact on cost estimates. The effect on net benefits is unknown. |
| Exclusion of the impact of economic incentive provisions, including banking, trading, and emissions averaging provisions. | Underestimate | Probably minor. Economic incentive provisions can substantially reduce costs, but the major economic programs for trading of sulfur and nitrogen dioxide emissions are reflected in the analysis. |
| Incomplete characterization of certain indirect costs, including vehicle owner opportunity costs associated with Inspection and Maintenance Programs and performance degradation issues associated with the incorporation of emission control technology. | Overestimate | Probably minor. Preliminary evidence suggests that the opportunity costs of vehicle owners is most likely small relative to other cost inputs.[3] In addition, it is will vary from State to State and is subject to a variety of influencing factors. The potential magnitude of indirect costs associated with performance degradation is more uncertain, because few data currently exist to quantify this effect. |

**Table 3-3 (continued)**
**Key Uncertainties Associated with Cost Estimation**

| Potential Source of Error | Direction of Potential Bias for Net Benefits | Likely Significance Relative to Key Uncertainties on Net Benefits Estimate[1] |
|---|---|---|
| Choice to model direct costs rather than social costs | Unable to determine based on current information | Probably minor. The relationship of social cost to direct cost estimates is influenced by multiple factors that operate in opposite directions, suggesting the magnitude of the net effect is reduced. Social cost estimates can reflect the net welfare changes across the full range of economic sectors in the U.S, and so may yield higher estimates of costs than a direct cost approach. In addition, social cost estimates can be constructed to reflect the potentially substantial cost-magnifying effect of existing tax distortions. Direct cost estimates, however, are likely to overstate costs in the primary market because they do not reflect consumer and producer responses. The extent to which a direct cost estimate will overstate or understate a social cost estimate depends on the magnitude of the "ripple effects" in economic sectors not targeted by a regulation. In addition, assessment of the effect on net benefit estimates must also account for any economy-wide effects of direct benefits (e.g., the broader implications of improving health status, and improving environmental quality). |
| Use of costs for rules that are currently in draft form (i.e., not yet finalized). | Unable to determine based on current information | Probably minor. Rules that are most important to the overall cost estimate are largely finalized. For example, there is some uncertainty as to how the cap-and-trade program through the SIP process will lower NOx emissions in an efficient manner. The expected effect on net benefits is minimal. |
| Exclusion of costs of 7-year and 10-year MACT standards and the residential risk standards for the 2- and 4-year MACT standards. | Unable to determine based on current information | Probably minor. Costs for the 7- and 10-year MACT standards are likely to be less than for the 2- and 4-year standards included in the analysis and the need for, and potential scope and stringency of, future Title III residual risk standards remain highly uncertain. For consistency, benefits of the 7- and 10-year standards and the residual risk standards are also excluded. |

Note:
[1] The classification of each potential source of error reflects the best judgement of the section 812 Project Team. The Project Team assigns a classification of "potentially major" if a plausible alternative assumption or approach could influence the overall monetary benefit estimate by approximately five percent or more; if an alternative assumption or approach is likely to change the total benefit estimate by less than five percent, the Project Team assigns a classification of "probably minor."
[2] For more detail, see Harrington et al (1999), referenced in Appendix B.
[3] Preliminary evidence based on Arizona's Enhanced I/M program indicates that major components of the programs costs are associated with test and repair costs rather than the costs of waiting and travel for vehicle owners. (Harrington and McConnell, 1999.) To date, Enhanced I/M programs have been implemented in only four States.

# Air Quality Modeling

Chapter 4

Air quality modeling links changes in emissions to changes in the atmospheric concentrations of pollutants that may affect human health and the environment. A crucial analytical step, air quality modeling is one of the more complex and resource-intensive components of the prospective analysis. This chapter outlines how we estimated future-year pollutant concentrations under both the Pre- and Post-CAAA scenarios using air quality modeling results and ambient monitor data. The first section of the chapter begins with a discussion of some of the challenges faced by air quality modelers and a brief description of the models we used in this analysis. The following section provides an overview of the general methodology we used to estimate future-year ambient concentrations. This methodology section includes a description of how we used modeling results to adjust monitor concentration data and estimate ambient concentrations for the years 2000 and 2010. The third section of this chapter summarizes the results of the air quality modeling and presents the expected effects of the CAAA on future-year pollutant concentrations. A discussion of the key uncertainties associated with air quality modeling concludes the chapter.

## Overview of Air Quality Models

Air quality modelers face two key challenges in attempting to translate emission inventories into pollutant concentrations. First, they must model the dispersion and transport of pollutants through the atmosphere. Second, they must model pertinent atmospheric chemistry and other pollutant transformation processes. These challenges are particularly acute for those pollutants that are not emitted directly, but instead form through secondary processes. Ozone is the best example; it forms in the atmosphere through a series of complex, non-linear chemical interactions of precursor pollutants, particularly certain classes of volatile organic compounds (VOCs) and nitrogen oxides ($NO_x$). We faced similar challenges when estimating PM concentrations. Atmospheric 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 recognizing 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.

Air quality modelers and researchers have responded to the need for scientifically valid and reliable estimates of air quality changes by developing a number of sophisticated atmospheric dispersion and transformation models. Some of these models have been employed in support of the development of federal clean air programs, national assessment studies, State Implementation Plans (SIPs), and individual air toxic source risk assessments. In this analysis, we used several of these well-established models to develop a picture of future changes in air quality resulting from the implementation of the 1990 CAAA.

We focused our air quality modeling efforts on estimating the impact of Pre- and Post-CAAA emissions on future-year ambient concentrations of ozone, $PM_{10}$, $PM_{2.5}$, $SO_2$, $NO_x$, and CO and on future-year acid deposition and visibility. The ideal model for this analysis would be a single integrated air quality model capable of estimating ambient concentrations for all criteria pollutants throughout the U.S. Although EPA is working to develop such a model, at the time of this analysis the model was not sufficiently developed and tested. In the absence of a single integrated model, we employed the Urban Airshed Model (UAM) in our analysis of ozone and used both the Regional Acid Deposition Model/Regional Particulate Model (RADM/RPM) and the Regulatory Modeling System for Aerosols and Acid

Deposition (REMSAD) model to assess $PM_{10}$, $PM_{2.5}$, acid deposition and visibility. All three of these models are three-dimensional grid models which require emissions and meteorological data as input. Each of these models calculate pollutant concentrations by simulating the physical and chemical pollution formation processes that occur in the atmosphere.

We conducted separate UAM, RADM/RPM, and REMSAD model runs for the 1990 base-year and each future-year projection scenario. The primary model input used for each run consisted of emissions estimates corresponding to the year and scenario being modeled (as described in Chapter 2 and Appendix A) and historical meteorological data corresponding to a past time period, referred to as a simulation period. We selected previous ozone episodes, i.e., multi-day events characterized by weather conditions conducive to ozone formation and transport (and as a result, characterized by multi-day spans with higher than average ozone concentrations), to serve as the simulation periods for UAM model runs. Although ozone concentrations during these simulation periods exceed the seasonal average, because the simulation periods for both the eastern and western U.S. cover roughly a two week span, ozone concentration peaks are largely offset by the surrounding lows. Overall, the selected simulation periods reasonably represent summertime ozone forming meteorological conditions and ozone concentrations. RADM/RPM simulation periods used to model PM, acid deposition, and visibility were chosen using a random selection process, while separate simulation periods at the beginning of each of the four seasons were chosen for REMSAD.

Table 4-1 provides an overview of the air quality models used in this analysis. We modeled concentrations of all pollutants across the 48 contiguous states; however due to the lack of an integrated model, separate air quality models were used to estimate ozone and PM for the eastern and western U.S. Table 4-1 shows the domain for each model and the simulation periods selected for use with each model and provides an overview of the spatial resolution of the models used as part of this analysis. The finer the resolution (i.e., the smaller the grid cells) the better the model can capture the effects of localized changes in emissions and weather conditions on ambient air quality. Recognizing the relationship between grid cell resolution and the certainty of results, we endeavored to estimate pollutant concentrations in more populated areas using higher resolution models. For this reason, we used the fine grid UAM-IV, an urban-scale model, to estimate ambient ozone levels in selected western cities. Similarly, we used an intermediate resolution grid (12 km x 12 km) to model ozone in "inner OTAG" states where population density is high and ozone transport is a major problem.[1]

Using the three-dimensional grid cell models, UAM, RADM/RPM, and REMSAD, we estimated grid-cell specific, hourly ozone and daily $PM_{10}$, and $PM_{2.5}$ concentrations for each day of the relevant simulation periods. We conducted separate model runs for the 1990 base-year and 2000 and 2010 future-year Pre- and Post-CAAA scenarios. Using these results, we ultimately projected the impact of the CAAA on ozone and PM ambient levels.

We relied on the same models used to predict PM concentrations to estimate changes in future-year acid deposition and visibility. For each model grid-cell we predicted daily acid deposition levels and visibility. Estimates for each day of the simulation period were generated for the base-year and both projection years under the Pre- and Post-CAAA scenarios.

We estimated future-year Pre- and Post-CAAA ambient $SO_2$, NO, $NO_2$, and CO concentrations by adjusting 1990 concentrations using future-year to base-year emissions ratios. This technique assumes a linear relationship between changes in emissions in an area and changes in that area's ambient concentration of the emitted pollutant.[2] Although this technique does not take into account pollutant transport or atmospheric chemistry, we believe linear scaling generates reasonable approximations of ambient concentrations of gaseous pollutants such as $SO_2$, $NO_x$, and CO.

[1] The Ozone Transport Assessment Group (OTAG) consists of the 37 easternmost states and the District of Columbia. The "inner OTAG" region is comprised of the more eastern (and more populated) states within the OTAG domain.

[2] 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.

**Table 4-1**
**Overview of Air Quality Models**

| Air Quality Measure | Region | Model | Spatial Resolution | Simulation Period |
|---|---|---|---|---|
| Ozone | Eastern U.S. | UAM-V | a) 12 km x 12 km grid for "Inner OTAG Region"<br>b) 36 km x 36 km grid for remainder of 37-state OTAG region | July 20-30, 1993 and July 7-18, 1995 |
| Ozone | Western U.S. | UAM-V | 56 km x 56 km grid (regional scale) covering the 11 westernmost states (states west of North and South Dakota, including western Texas) | July 1-10, 1990 |
| Ozone | San Francisco Bay Area | UAM-IV | 4 km x 4 km (urban scale) grid covering the San Francisco Bay Area, the Monterrey Bay Area, Sacramento, and a portion of the San Joaquin Valley | Aug. 3-6, 1990 |
| Ozone | Los Angeles Area | UAM-IV | 5 km x 5 km grid covering the South Coast Air Basin from Los Angeles to beyond Riverside and including part of the Mojave Desert | June 23-25, 1987 and Aug. 26-28, 1987 |
| Ozone | Maricopa County (Phoenix) Area | UAM-IV | 4 km x 4 km grid covering urbanized portion of Maricopa County | Aug. 9-10, 1992 and June 13-14, 1993 |
| Particulate Matter | Eastern U.S. | RADM/RPM | 80 km x 80 km grid (coarse resolution) covering eastern North America from the Rocky Mountains eastward to Newfoundland, Canada and the Florida Keys (see Fig. C-14 in Appendix C) | 30 randomly selected 5-day periods spanning a four-year period |
| Particulate Matter | Western U.S. | REMSAD | 56 km x 56 km grid covering the 11 westernmost states | ten-day period for each of four seasons:<br>May 1-10,<br>July 1-10,<br>Oct. 1-10, and<br>Dec. 1-10 |
| Visibility | Eastern U.S. | RADM/RPM | (same as PM) | (same as PM) |
| Visibility | Western U.S. | REMSAD | (same as PM) | (same as PM) |
| Acid Deposition | Eastern U.S. | RADM | (same as RADM/RPM) | (same as RADM/RPM) |
| Sulfur Dioxide | U.S. | linear scaling | 56 km x 56 km REMSAD grid covering 48 contiguous states | not applicable |
| Oxides of Nitrogen | U.S. | linear scaling | 56 km x 56 km REMSAD grid covering 48 contiguous states | not applicable |
| Carbon Monoxide | U.S. | linear scaling | 56 km x 56 km REMSAD grid covering 48 contiguous states | not applicable |

## General Methodology

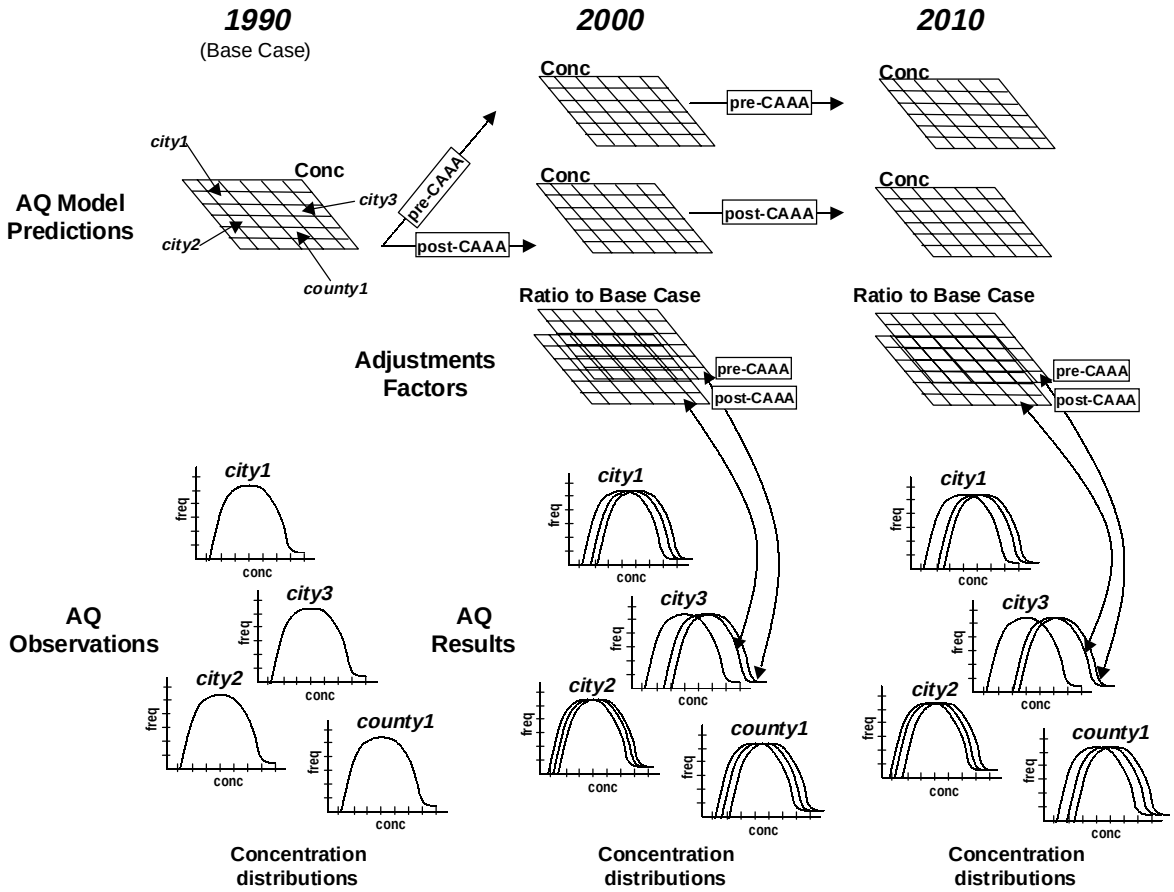
The air quality modeling component of the 812 prospective analysis involved the application of a variety of complex, sophisticated air quality modeling tools and techniques. Overall, however, the method we used to estimate the impact of changes in emissions on air quality was relatively straight forward. We began by gathering 1990 air quality monitor data for the six criteria pollutants analyzed in this study. These observational data served as the air quality baseline for both the Pre- and Post-CAAA scenarios. We then estimated 2000 and 2010 concentrations of each pollutant under each emissions scenario by applying adjustment factors to the 1990 monitor data. The adjustment factors for each future-year projection scenario were based on the relative change in pollutant concentration between 1990 and the desired future-year, as predicted by air quality simulation modeling. This section presents an overview of the methodology we used to estimate future-year ambient concentrations. For a more detailed description, please refer to Appendix C.

The diagram in Figure 4-1 illustrates the methodology used to estimate ozone and PM concentrations. First, we compiled distributions of observed pollutant concentrations recorded at each air quality monitor in 1990. We obtained these data from EPA's Aerometric Information Retrieval System (AIRS), a publicly accessible database of air quality information. Separately, we then developed distributions of estimated concentrations for each pollutant in 1990 using 1990 emissions data and the appropriate air quality model. Unlike the 1990 observed concentrations that were measured at monitoring sites, the 1990 estimated concentrations were calculated at the center of each cell of a grid covering the domain of the applicable air quality model. Using future-year emission inventory estimates for the Pre-CAAA and Post-CAAA scenarios (developed as described in Chapter 2 and Appendix A) and the appropriate air quality models, we next developed distributions of model-estimated concentrations at each grid cell for each of four future-year projection scenarios: 2000 Pre-CAAA, 2010 Pre-CAAA, 2000 Post-CAAA, and 2010 Post-CAAA. These results were used to derive adjustment factors for each air quality monitor, based on the simulation results for the grid cell in which the monitor is located. The future-year/scenario adjustment factor for each pollutant represents the ratio of the simulated future-year/scenario concentration to the 1990 model-estimated concentration. These factors were calculated by matching future-year and 1990 concentrations at regular intervals in each distribution. Finally, four sets of model-derived adjustment factors were applied to the distribution of observed 1990 concentrations at each monitor to forecast distributions of concentrations for each of the four future-year projection scenarios. It is these concentrations that serve as inputs into the CAAA benefits modeling.

An illustrative example follows. Assume the median observed concentration of Pollutant A at Monitor X in 1990 was 0.24 ppm. Air quality modeling for the grid cell in which Monitor X is located predicts a median Pollutant A concentration of 0.30 ppm in 1990 and 0.15 ppm in 2010 under the post-CAAA scenario. The 2010 Post-CAAA adjustment factor for the median Pollutant A concentration would be 0.5, and the predicted 2010 Post-CAAA median concentration at Monitor X would be 0.5 (=0.15/0.30) times the 1990 monitor value of 0.24 ppm, or 0.12 ppm.

Our approach for forecasting concentrations of $SO_2$, $NO_x$, and CO involved the same basic approach described above. However, instead of applying model-derived adjustment factors to the 1990 observed distribution of concentrations, we adjusted the 1990 distribution using the ratio of future-year emissions to 1990 emissions in the vicinity of the monitor for each of the four future-year projection scenarios. For more information about this approach, please refer to Appendix C.

**Figure 4-1**
**Schematic diagram of the future-year concentration estimation methodology**




NOTE: Figure illustrates how model results and observations are used to produce the air quality profiles (concentration distributions) for the benefits analysis. The figure shows model runs at the top; four sets of "ratios" of model results in space in the middle; and frequency distributions of pollutant monitor concentrations and the space-dependent scaling of these by the ratios of the model predictions on the bottom.

## Air Quality Model Results

This section presents a summary representation of the air quality modeling results. We discuss the model-simulated concentration estimates and the adjusted future-year concentration predictions with a focus on the change in air quality resulting from the implementation of the 1990 CAAA.

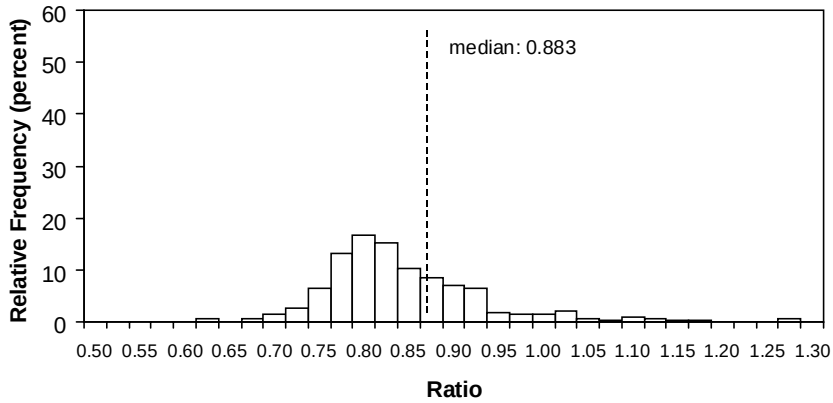
### *Ozone*

We modeled ozone concentrations separately for the eastern U.S., western U.S., San Francisco Bay area, Los Angeles area, and Maricopa County (Phoenix, AZ) area. Examination of base-year and future-year model concentration estimates shows expected increases in Pre-CAAA ozone concentrations and expected decreases in Post-CAAA ozone concentrations in the eastern U.S. In this part of the country, UAM-V predicts Pre-CAAA ozone concentration increases will occur primarily over the states of Virginia, North Carolina, Kentucky, Tennessee, Georgia, and Alabama; while Post-CAAA decreases will be more widespread. Comparison of Pre- and Post-CAAA model estimates shows that, with the exception of a few isolated areas, ambient ozone levels throughout the East will be reduced in the year 2010 as a result of the CAAA. These lower levels are largely due to significant reductions in area source and motor vehicle VOC emissions and utility, point source, and motor vehicle $NO_x$ emissions.

Regional-scale model results for the western U.S. indicate that ozone concentrations in this portion of the country, just as in the eastern U.S., will generally increase from the 1990 base-year under the Pre-CAAA scenario and decrease from 1990 levels under the Post-CAAA scenario. In the West, we anticipate widespread changes under both scenarios; however, we project that the increases in Pre-CAAA ozone concentrations and decreases in Post-CAAA model concentrations will be smaller than the pre-

dicted changes in ambient ozone levels in the eastern U.S. Furthermore, comparison of 2010 Pre- and Post-CAAA model estimates shows that future-year western ozone concentrations will be lower as a result of the 1990 Amendments, but UAM-V results indicate that the reductions in the West will likely be about half the size of the reductions in the eastern portion of the country. The difference between the change in western ozone concentrations and the change in eastern ozone concentrations is largely due to the more aggressive $NO_x$ controls expected in the East. Specifically, the Post-CAAA scenario incorporates the effects of a $NO_x$ cap-and-trade system for the eastern U.S. (OTAG region). Another reason for the difference between the modeled change in eastern and western ozone concentrations is that we estimated ozone levels in the East and West using different model grid resolutions. The coarser the resolution, the less responsive the model concentration estimates are to localized changes in emissions. Thus, the smaller estimated change in western ozone concentrations than in eastern ozone concentrations may, in part, be attributable to the fact that UAM-V grid-cells covering the western U.S. are larger than those covering the eastern U.S.

**Figure 4-2**
**Distribution of Monitor Level Ratios for 95th Percentile Ozone Concentrations: 2010 Post-CAAA/Pre-CAAA**




Western urban-area modeling results differ from the regional scale results described above. Examination of Pre- and Post-CAAA modeling estimates shows that, in some portions of the urban centers of San Francisco and Los Angeles, future-year Post-CAAA ozone concentrations are expected to be higher than Pre-CAAA estimates. This ozone "disbenefit" is the result of inhibiting a complex chemical reaction termed "$NO_x$ scavenging," during which a reduction in $NO_x$, an ozone precursor, leads to an increase in ozone production instead of the typical decrease.[3] In the area immediately surrounding the two cities, however, and in Maricopa County, model results show that scavenging is not expected to be influential, if it occurs at all, and future-year Post-CAAA ozone concentration estimates are predicted to be lower than Pre-CAAA estimates.

As described above, we used the UAM-V model results to calculate adjustment factors for each of the four future-year projection scenarios. We estimated future-year monitor-level ozone concentrations by applying these factors to 1990 observed concentrations. Examination of the distribution of adjusted monitor concentration ratios for 95th percentile ozone concentrations is one means of analyzing the impact of the CAAA on air pollution. The distribution of ratios of 2010 Pre-CAAA to 1990 base-year ozone concentrations reveals that the majority of future year Pre-CAAA ozone concentration estimates are between zero and 10 percent greater than 1990 levels, with most concentrations falling in the middle of this range. The distribution of ratios of 2010 Post-CAAA to 1990 base-year shows that in nearly all areas of the U.S. ozone concentrations will be lower in 2010 than in the base-year; in the majority of the country, future-year concentrations will be five to 20 percent lower than in the base-year.[4] The histogram in Figure 4-2 depicts the distribution of ratios of 2010 Post-CAAA ozone estimates to 2010 Pre-CAAA ozone estimates. Most of the ratios in the distribution are less than one, with a median of 0.883. This indicates that the 95th percentile level Post-CAAA concentrations, with few exceptions, are lower than the corresponding Pre-CAAA values. The smaller the ratio, the greater the difference between future-year scenarios.

[3] Scavenging occurs in areas, typically cities, with limited VOC and abundant $NO_x$. In VOC-limited areas where there is a relatively high $NO_x$ concentration (regions where the concentration of VOC, not $NO_x$, dictates the amount of ozone that can be formed), these two ozone precursors (VOC and $NO_x$) compete to react with a particular gaseous compound. To produce ozone, this compound must combine with VOC. As a result, if the compound joins with $NO_x$, ozone production is impeded; thus, a decrease in $NO_x$ leads to an increase in ozone concentrations.

[4] See Appendix C for histograms illustrating the change in ozone concentrations from the base-year.

## Particulate Matter

To model Pre- and Post-CAAA particulate matter ($PM_{10}$ and $PM_{2.5}$) concentrations, we used RADM/RPM for the eastern U.S. and REMSAD for the western U.S. Results from both models show PM concentrations are expected to be lower under the Post-CAAA scenario than under the Pre-CAAA scenario. This projected improvement in air quality is widespread throughout the eastern U.S., with 2010 Post-CAAA PM estimates in some parts of the East up to 15 to 30 percent lower than 2010 Pre-CAAA estimates. In the West, projected reductions in future-year PM concentrations (Pre-CAAA minus Post-CAAA) are largely restricted to urban areas.[5] The broad scale improvement in eastern PM concentrations is driven largely by reductions in utility source sulfur dioxide emissions throughout this portion of the country.[6] In the West, however, sulfur dioxide emissions have a much smaller impact on overall PM concentrations. Western PM concentrations are more significantly influenced by area, motor vehicle, and nonroad source emissions of nitrogen oxides and directly emitted PM. These sources are more concentrated in urban areas. As a result, the impact of the CAAA on PM concentrations in the West is primarily restricted to urban areas.

Examination of the distribution of adjusted monitor-level concentration ratios for annual average PM concentrations reveals that 2010 Pre-CAAA $PM_{10}$ and $PM_{2.5}$ estimates are both higher than 1990 base-year estimates in almost all areas of the country. Pre-CAAA 2010 $PM_{10}$ and $PM_{2.5}$ estimates are generally zero to 10 percent greater than 1990 base-year estimates. The average estimated increase in $PM_{2.5}$ concentrations, however, is slightly larger than the average estimated increase in $PM_{10}$.[7] The estimated change in PM concentrations from the base-year to 2010 under the Post-CAAA scenarios is less uniform. While the majority of areas experience a reduction in annual average $PM_{10}$ and $PM_{2.5}$ concentrations, in a number of areas ambient PM levels, more frequently $PM_{2.5}$, increase from the base-year under the Post-CAAA scenario. On average, however, 2010 Post-CAAA $PM_{10}$ and $PM_{2.5}$ concentrations are between zero and five percent and zero and 10 percent, respectively, lower than 1990 base-year concentrations.[8]

As shown in Figures 4-3 and 4-4, the percentage reduction in $PM_{2.5}$ concentrations across the U.S. between the Pre- and Post-CAAA scenarios vary more widely than the percentage reduction in $PM_{10}$. In the emissions analysis we focus on the impact of the CAAA on anthropogenic emissions and, so, hold natural source PM emissions constant at 1990 levels. Natural source emissions make up a much larger portion of $PM_{10}$ concentrations than $PM_{2.5}$ concentrations and dampen the influence of changes in anthropogenic emissions on ambient $PM_{10}$ concentrations.

Comparison of the two distributions in Figures 4-3 and 4-4 shows that, despite the greater variation of $PM_{2.5}$ reductions, the percentage reduction in $PM_{2.5}$ concentrations are larger on average than the percentage reduction in $PM_{10}$ concentrations. The reason for this difference is two fold. First, as described above, $PM_{2.5}$ concentrations are more susceptible to the influence of changes in anthropogenic emissions, which are regulated by the CAAA. Second, the CAAA provisions that influence PM emissions (regulations that focus on secondary PM precursors such as $NO_x$, and $SO_2$, and primary PM sources such as diesel engine exhaust standards) affect the fine particulate ($PM_{2.5}$) subset of $PM_{10}$ to a much greater extent than the coarser fraction that makes up the rest of $PM_{10}$. As a result of these two factors, the projected difference in ambient concentrations between the Pre-CAAA and Post-CAAA scenarios reflect a larger percentage reduction in $PM_{2.5}$ than $PM_{10}$.

[5] Outside the larger urban areas in the West, REMSAD results show little or no change in PM concentrations between Pre- and Post-CAAA estimates.

[6] Sulfur dioxide is a secondary PM precursor.

[7] In some of the figures in this chapter the Pre-CAAA and Post-CAAA scenarios are referred to as Pre-CAAA90 and Post-CAAA90, respectfully.

[8] See Appendix C for histograms illustrating the change in PM concentrations from the 1990 base-year to each of the Pre-CAAA and Post-CAAA future year scenarios.

**Figure 4-3**
**Distribution of Combined RADM/RPM and REMSAD Derived Monitor Level Ratios for Annual Average $PM_{10}$ Concentrations: 2010 Post-CAAA/Pre-CAAA**

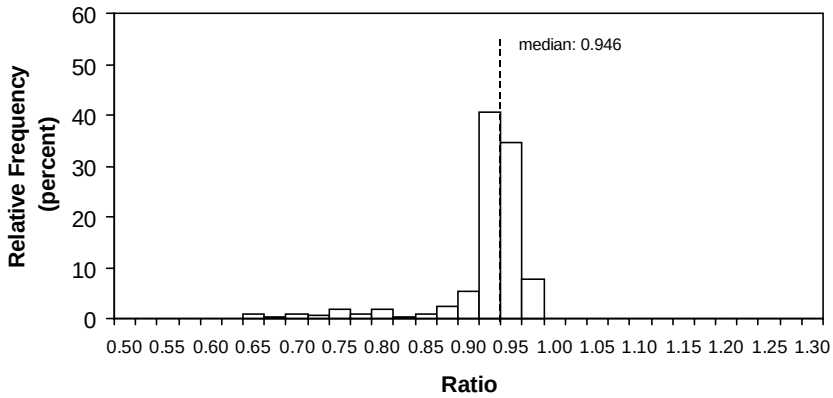
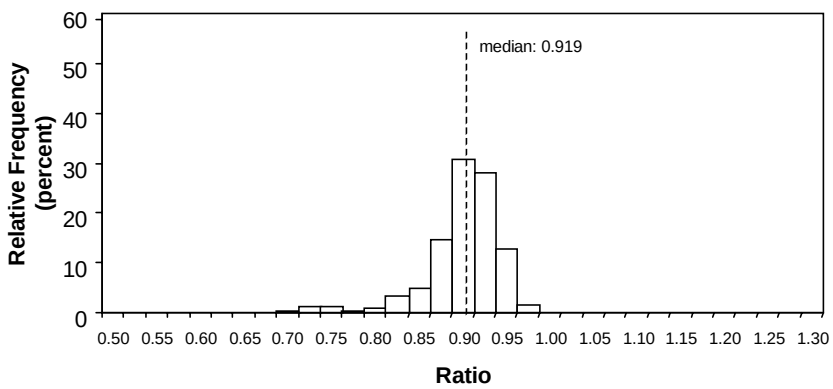



**Figure 4-4**
**Distribution of Combined RADM/RPM and REMSAD Derived Monitor Level Ratios for Annual Average $PM_{2.5}$ Concentrations: 2010 Post-CAAA/Pre-CAAA**




## *Visibility*

We also relied on RADM/RPM and REMSAD to estimate the impact of the CAAA on future-year visibility. Tables 4-2 and 4-3 compare the mean annual visibility (expressed in deciviews)[9] in selected eastern urban areas and National Parks, respectively, as estimated by RADM/RPM under the 1990 base-year and 2010 Pre- and Post-CAAA scenarios. Comparison of these values reveals that, in the eastern U.S., we anticipate that future-year visibility in both urban and rural areas is projected to improve under the Post-CAAA scenario. RADM/RPM predicts that Post-CAAA visibility in 2010 will not only be better than Pre-CAAA visibility, but also, in many areas, it will be better than the visibility in the 1990 base-year. This improvement in visibility is attributable to reductions in the concentration of gaseous and suspended particles, such as PM, that scatter and absorb light, and thus influence visibility.

Visibility in the West is also significantly better under the Post-CAAA scenario than under the Pre-CAAA scenario (see Tables 4-4 and 4-5). Base-year model runs show that visibility in the western U.S. is the poorest in larger metropolitan areas such as Los Angeles, CA; San Francisco, CA; Denver, CO; and Phoenix, AZ. Under the 2010 Pre-CAAA scenario, REMSAD estimates that, throughout much of the West, visibility will remain relatively unchanged from the base-year, and in some cases will even improve. In the metropolitan areas, however, the model predicts visibility degradation. Under the Post-CAAA scenario, however, REMSAD estimates widespread improvement in future-year visibility in the West. In both metropolitan and non-urban areas, deciview levels estimated for 2010 are lower under the Post-CAAA scenario than under the Pre-CAAA scenario. The model suggests Los Angeles and Las Vegas will experience the greatest improvement.

[9] The deciview is a measure of visibility which captures the relationship between air pollution and human perception of visibility. When air is free of the particles that cause visibility degradation, the DeciView Haze Index is zero. The higher the deciview level, the poorer the visibility; a one to two deciview change translates to a just noticeable change in visibility for most individuals.

**Table 4-2**
**Comparison of Visibility in Selected Eastern Urban Areas**

| | | Mean Annual Deciview* | | |
|---|---|---|---|---|
| **Area Name** | **State** | **1990 Base-Year** | **2010 Pre-CAAA** | **2010 Post-CAAA** |
| Atlanta Metro Area | GA | 20.9 | 22.8 | 20.0 |
| Boston Metro Area | MA | 13.2 | 14.0 | 11.9 |
| Chicago Metro Area | IL | 17.5 | 19.1 | 17.0 |
| Columbus | OH | 16.5 | 17.7 | 15.1 |
| Detroit Metro Area | MI | 16.0 | 18.5 | 15.3 |
| Indianapolis | IN | 20.1 | 21.1 | 19.0 |
| Little Rock | AR | 15.0 | 17.2 | 15.1 |
| Milwaukee Metro Area | WI | 15.6 | 18.4 | 15.3 |
| Minn.-St. Paul Metro Area | MN | 10.1 | 12.4 | 10.3 |
| Nashville | TN | 20.4 | 21.5 | 19.0 |
| New York City Metro Area | NY/NJ | 15.2 | 18.0 | 13.9 |
| Pittsburgh Metro Area | PA | 15.8 | 16.9 | 14.2 |
| St. Louis Metro Area | MO | 16.5 | 17.8 | 16.0 |
| Syracuse | NY | 12.4 | 13.2 | 11.5 |
| Washington, DC Metro Area | DC/VA/MD | 17.5 | 19.2 | 16.3 |

*For cities or metropolitan areas not contained by a single RADM/RPM grid cell, the visibility measure presented in this table is a weighted average of the mean annual deciview level from each of the grid cells that together completely contain the selected area. Weighting is based upon the spatial distribution of an area over the various grid cells.

**Table 4-3**
**Comparison of Visibility in Selected Eastern National Parks**

| | | Mean Annual Deciview* | | |
|---|---|---|---|---|
| **Area Name** | **State** | **1990 Base-Year** | **2010 Pre-CAAA** | **2010 Post-CAAA** |
| Acadia NP | ME | 11.1 | 12.0 | 10.4 |
| Everglades NP | FL | 7.6 | 9.2 | 6.9 |
| Great Smoky Mtns. NP | TN | 20.4 | 22.3 | 19.6 |
| Shenandoah NP | VA | 16.5 | 17.8 | 15.2 |

*For national parks not contained by a single RADM/RPM grid cell, the visibility measure presented in this table is a weighted average of the mean annual deciview level from each of the grid cells that together completely contain the selected area. Weighting is based upon the spatial distribution of an area over the various grid cells.

**Table 4-4**
**Comparison of Visibility in Selected Western Urban Areas**

| | | Mean Annual Deciview* | | |
|---|---|---|---|---|
| **Area Name** | **State** | **1990 Base-Year** | **2010 Pre-CAAA** | **2010 Post-CAAA** |
| Denver | CO | 19.4 | 22.6 | 21.0 |
| Las Vegas | NV | 14.6 | 17.9 | 15.2 |
| Los Angeles | CA | 22.7 | 24.6 | 22.0 |
| Phoenix | AZ | 15.4 | 17.1 | 15.3 |
| Salt Lake City | UT | 12.5 | 14.8 | 13.4 |
| San Francisco | CA | 24.4 | 26.1 | 24.6 |
| Seattle | WA | 20.5 | 22.2 | 21.0 |

*For cities not contained by a single REMSAD grid cell, the visibility measure presented in this table is a weighted average of the mean annual deciview level from each of the grid cells that together completely contain the selected area. Weighting is based upon the spatial distribution of an area over the various grid cells.

**Table 4-5**
**Comparison of Visibility in Selected Western National Parks**

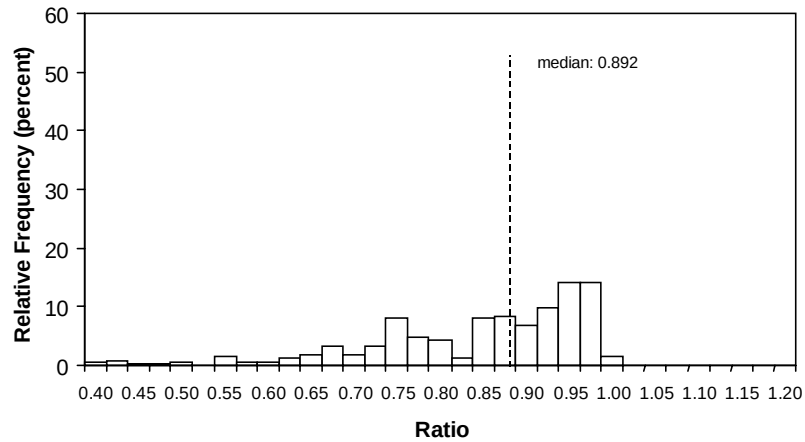
| | | Mean Annual Deciview* | | |
|---|---|---|---|---|
| **Area Name** | **State** | **1990 Base-Year** | **2010 Pre-CAAA** | **2010 Post-CAAA** |
| Glacier NP | MT | 11.2 | 11.9 | 11.5 |
| Grand Canyon NP | AZ | 8.3 | 8.8 | 8.3 |
| Olympic NP | WA | 11.1 | 11.8 | 11.7 |
| Yellowstone NP | WY | 9.0 | 9.7 | 9.5 |
| Yosemite NP | CA | 11.5 | 13.2 | 12.2 |
| Zion NP | UT | 8.0 | 9.0 | 8.4 |

*For national parks not contained by a single REMSAD grid cell, the visibility measure presented in this table is a weighted average of the mean annual deciview level from each of the grid cells that together completely contain the selected area. Weighting is based upon the spatial distribution of an area over the various grid cells.

## Acid Deposition

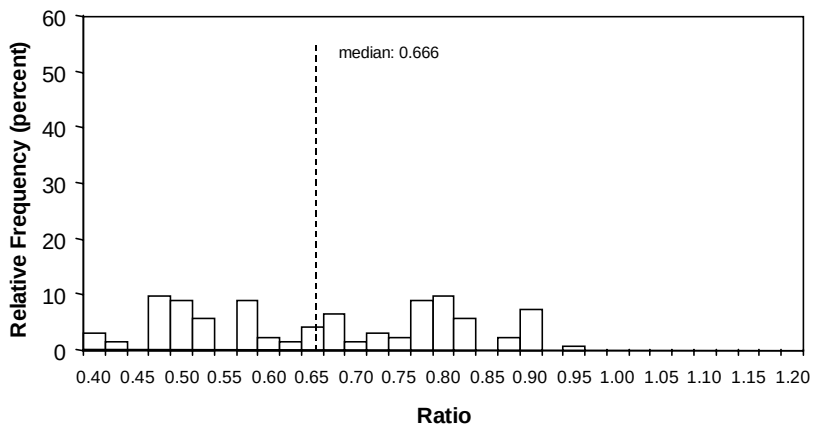
We estimated nitrogen and sulfur deposition for the 1990 base-year and each of the future-year emissions scenarios. Using RADM, we focused on acid deposition in the eastern U.S. where the acidification problem is the most acute. Under the Pre-CAAA scenario, model results show an increase in both nitrogen and sulfur deposition between 1990 and 2010. However, under the Post-CAAA scenario, 2010 deposition projections are not only lower than 2010 Pre-CAAA projections, but also below 1990 base-year levels as well. Average annual acid deposition is expected to decrease as a result of the CAAA. Motor vehicle tailpipe emissions standards and Title IV Acid Rain provisions are expected to significantly reduce both $NO_x$ and $SO_2$ emissions thus contributing to significant reductions in downwind deposition of acidic nitrogen and sulfur compounds. The differences between the Pre-CAAA and Post-CAAA projections, however, imply that the 1990 Amendments will have a larger impact on the percentage reduction in nitrogen deposition than on the percentage reduction in sulfur deposition. One reason for the greater change in nitrogen deposition is the region-wide $NO_x$ emissions cap-and-trade program that is part of the Post-CAAA scenario.

**Figure 4-5**
**Distribution of Monitor - Level Ratios of $SO_2$ Emissions**




Note: 2.4 percent of the distribution of ratios is less than 0.40.

**Figure 4-6**
**Distribution of Monitor - Level Ratios of NO Emissions**




Note: 3.3 percent of the distribution of ratios is less than 0.40.

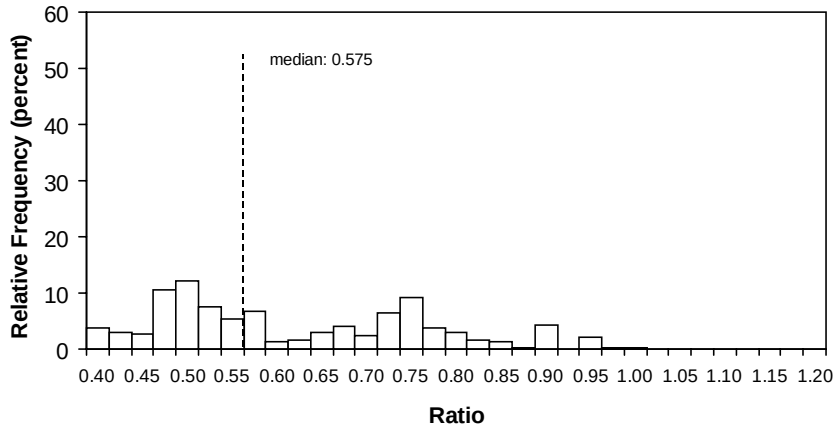
## $SO_2$, NO, $NO_2$, and CO

To estimate future-year $SO_2$, NO, $NO_2$, and CO concentrations we relied on linear emissions scaling, adjusting 1990 base-year concentrations using ratios of future-year to base-year emissions. Ratios greater than one indicate an increase in ambient concentrations relative to the base-year, while ratios less than one indicate a decrease.[10]

Our results indicate that compared to the base-year, future-year concentrations of $SO_2$, NO, $NO_2$, and CO tend to increase under the Pre-CAAA scenario, while Post-CAAA concentrations for all four pollutants except $SO_2$ tend to decrease. For example, the median 2010 Pre-CAAA emission-based ratio for $SO_2$ is roughly 1.35, indicating an increase in median 2010 Pre-CAAA $SO_2$ concentration of approximately 35 percent from the 1990 base-year. The median ratios for NO, $NO_2$, and CO are roughly 1.13, 1.17, and 1.05 respectively. Under the Post-CAAA scenario we estimate that in 2010 NO, $NO_2$, and CO concentrations will tend to be approximately 25 and 30 percent below base-year levels. The median 2010 Post-CAAA emission-based ratios for these three pollutants are roughly 0.74, 0.70, and 0.76 respectively.

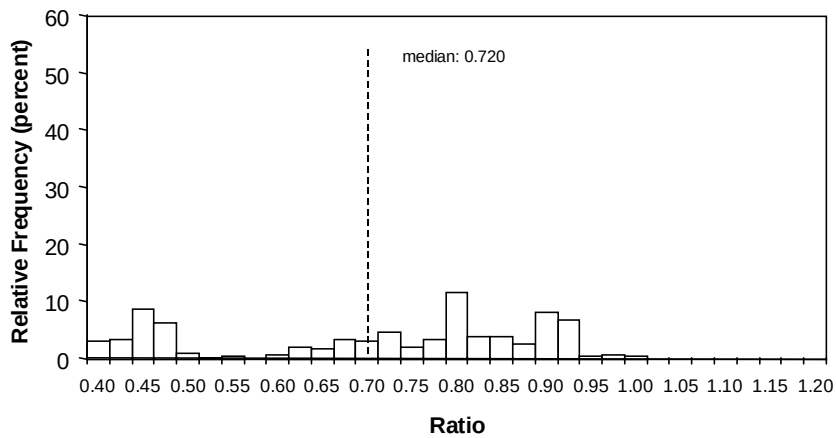
[10] The values in this section represent ratios for actual monitoring site locations. Interpolated data are not included in these figures. We believe, however, that the values presented in this section accurately reflect the impact of the 1990 Amendments on $SO_x$, NO, $NO_2$, and CO ambient concentrations.

**Figure 4-7**
**Distribution of Monitor - Level Ratios of $NO_2$ Emissions**




Note: 2.7 percent of the distribution of ratios is less than 0.40.

**Figure 4-8**
**Distribution of Monitor - Level Ratios of CO Emissions**




Note: 15.7 percent of the distribution of ratios is less than 0.40.

Comparison of Pre- and Post-CAAA emission-based adjustment factors also helps illustrate the effect of the 1990 Amendments on ambient pollution concentrations. The ratio of 2010 Post-CAAA adjustment factors to 2010 Pre-CAAA adjustment factors shows the impact of the 1990 Amendments on ambient concentrations relative to the baseline scenario. Ratios less than one indicate that we estimate that future-year concentrations of $SO_2$, NO, $NO_2$, and CO are lower under the Post-CAAA scenario than under the Pre-CAAA scenario.

Figures 4-5 through 4-8 show the distribution of 2010 Post-CAAA to 2010 Pre-CAAA ratios for summertime $SO_2$, NO, $NO_2$, and CO respectively. These figures illustrate the regional variation in the influence of the 1990 Amendments on ambient concentrations of these pollutants. Although we estimate concentrations in some areas will increase under the Post-CAAA scenario relative to Pre-CAAA estimates, the median summertime 2010 Post- to Pre-CAAA ratios for $SO_2$, NO, $NO_2$, and CO are 0.90, 0.67, 0.58, and 0.72 respectively. These values, each less than one, indicate that the central tendency for summertime 2010 Post-CAAA concentration estimates of these four pollutants is to be lower than 2010 Pre-CAAA estimates.

Table 4-6 displays the median values of the distribution of Post- to Pre-CAAA ratios for the summer months described above and the remaining three seasons. Just as for the summer; spring, autumn, and winter median values are less than one. Averaged over all four seasons, we estimate a median reduction in $SO_2$, NO, $NO_2$, and CO concentrations of approximately 9, 33, 40, and 25 percent respectively. RACT requirements, tailpipe emissions standards, and $NO_x$ emissions trading account for the bulk of the reduc-

**Table 4-6**
**Median Values of the Distribution of Ratios of 2010 Post-CAAA/ Pre-CAAA Adjustment Factors**

| | $SO_2$ | NO | $NO_2$ | CO |
|---|---|---|---|---|
| Spring | 0.904 | 0.669 | 0.598 | 0.790 |
| Summer | 0.892 | 0.666 | 0.575 | 0.720 |
| Autumn | 0.916 | 0.677 | 0.614 | 0.756 |
| Winter | 0.924 | 0.686 | 0.626 | 0.692 |

tion in NO and $NO_2$ concentrations. Title I nonattainment area controls and Title II motor vehicle provisions are responsible for much of the change in CO concentrations, while regulation of utility and motor vehicle emissions account for majority of the decrease in $SO_2$ concentrations.

## Uncertainty in the Air Quality Estimates

Many sources of uncertainty affect the precision and accuracy of the projected changes in air quality presented in this study. These uncertainties arise largely from potential inaccuracies in the emissions inventories used as air quality modeling inputs and potential errors in the structure and parameterization of the air quality models themselves. For example, we estimated changes in PM concentrations in the eastern U.S. based exclusively on changes in the concentrations of sulfate and nitrate particles. By not accounting for changes in organic and primary particulate fractions, we likely underestimate the impact of the CAAA on PM concentrations. Also, by using separate air quality models for individual pollutants and different geographic regions, as opposed to a single integrated model, we were unable to fully capture the interaction among air pollutants or reflect transport of pollutants or precursors across the boundaries of the models covering the western and eastern states. The direction and magnitude of bias these limitations impose on net benefits estimate presented in this analysis can not be determined based on current information.

Some model-related uncertainties, however, may be mitigated because this analysis uses the air quality modeling results in a relative, not absolute, sense. We focus on the change in air quality between the Pre- and Post-CAAA scenarios and not on the ambient concentrations projected by the individual models themselves. Therefore, uncertainties that affect a model's ability to accurately predict the relative change in concentration of a pollutant from one scenario to another are more important in the context of this study than those that affect only the absolute model results.

The relatively coarse grid cells used to model ozone in most areas of the U.S. represents a potential source of uncertainty affecting a model's sensitivity to changes in emissions. Grid size affects chemistry, transport, and diffusion processes that in turn determine the response of pollutant concentrations to changes in emissions. The less accurately a model can predict the impact of changes in emissions on ambient levels, the greater the uncertainty associated with predicted differences between Pre- and Post-CAAA concentration estimates.

Table 4-7 presents the most important specific sources of uncertainty and Appendix C further describes the uncertainties associated with air quality modeling. While the list of potential errors presented in Table 4-7 is not exhaustive, it includes discussion of those factors with the greatest likelihood of contributing to any potential bias in the primary net benefit estimates.

**Table 4-7**
**Key Uncertainties Associated with Air Quality Modeling**

| Potential Source of Error | Direction of Potential Bias for Net Benefits Estimate | Likely Significance Relative to Key Uncertainties in Net Benefit Estimate* |
|---|---|---|
| $PM_{10}$ and $PM_{2.5}$ concentrations in the East (RADM domain) are based exclusively on changes in the concentrations of sulfate and nitrate particles, omitting the effect of anticipated reductions in organic or primary particulate fractions. | Underestimate. | Potentially major. Nitrates and sulfates constitute major components of PM, especially $PM_{2.5}$, in most of the RADM domain and changes in nitrates and sulfates may serve as a reasonable approximation to changes in total $PM_{10}$ and total $PM_{2.5}$. Of the other components, primary crustal particulate emissions are not expected to change between scenarios; primary organic carbon particulate emissions are expected to change, but an important unknown fraction of the organic PM is from biogenic emissions, and biogenic emissions are not expected to change between scenarios. If the underestimation is major, it is likely the result of not capturing reductions in motor vehicle primary elemental carbon and organic carbon particulate emissions. |
| The number of $PM_{2.5}$ ambient concentration monitors throughout the U.S. is limited. As a result, cross estimation of $PM_{2.5}$ concentrations from $PM_{10}$ (or TSP) data was necessary in order to complete the "monitor-level" observational dataset used in the calculation of air quality profiles. | Unable to determine based on the current information. | Potentially major. $PM_{2.5}$ exposure is linked to mortality, and avoided mortality constitutes a large portion of overall CAAA benefits. Cross estimation of $PM_{2.5}$, however, is based on studies that account for seasonal and geographic variability in size and species composition of particulate matter. Also, results are aggregated to the annual level, improving the accuracy of cross estimation. |
| Use of separate air quality models for individual pollutants and for different geographic regions does not allow for a fully integrated analysis of pollutants and their interactions. | Unable to determine based on current information. | Potentially major. There are uncertainties introduced by different air quality models operating at different scales for different pollutants. Interaction is expected to be most significant for PM estimates. However, important oxidant interactions are represented in all PM models and the models are being used as designed. The greatest likelihood of error in this case is for the summer period in areas with NOx inhibition of ambient ozone (e.g., Los Angeles). |
| Future-year adjustment factors for seasonal or annual monitoring data are based on model results for a limited number of simulation days. | Overall, unable to determine based on current information. | Probably minor. RADM/RPM and REMSAD PM modeling simulation periods represent all four seasons and characterize the full seasonal distribution. Potential overestimation of ozone, due to reliance on summertime episodes characterized by high ozone levels and applied to the May-September ozone season, is mitigated by longer simulation periods, which contain both high and low ozone days. Also, underestimation of UAM-V western and UAM-IV Los Angeles ozone concentrations (see below) may help offset the potential bias associated with this uncertainty. |

**Table 4-7**
**Key Uncertainties Associated with Air Quality Modeling (continued)**

| Potential Source of Error | Direction of Potential Bias for Net Benefits Estimate | Likely Significance Relative to Key Uncertainties in Net Benefit Estimate* |
|---|---|---|
| Comparison of modeled and observed concentrations indicates that ozone concentrations in the western states were somewhat under-predicted by the UAM-V model, and ozone concentrations in the Los Angeles area were underestimated by the UAM-IV model. | Unable to determine based on current information. | Probably minor. Because model results are used in a relative sense (i.e., to develop adjustment factors for monitor data) the tendency for UAM-V or UAM to underestimate absolute ozone concentrations would be unlikely to affect overall results. To the extent that the model is not accurately estimating the relative changes in ozone concentrations across regulatory scenarios, the effect could be greater. |
| Ozone modeling in the eastern U.S. relies on a relatively coarse 12 km grid, suggesting $NO_x$ inhibition of ambient ozone levels may be under represented in some eastern urban areas. Coarse grid may affect both model performance and response to emissions changes. | Unable to determine based on current information. | Probably minor. Though potentially major for eastern ozone results in those cities with known $NO_x$ inhibition, ozone benefits contribute only minimally to net benefit projections in this study. Grid size affects chemistry, transport, and diffusion processes which in turn determine the response to changes in emissions, and may also affect the relative benefits of low-elevation versus high-stack controls. However, the approach is consistent with current state-of-the-art for regional-scale ozone modeling. |
| UAM-V modeling of ozone in the western U.S. uses a coarser grid than the eastern UAM-V (OTAG) or UAM-IV models, limiting the resolution of ozone predictions in the West. | Unable to determine based on current information. | Probably minor. Also, probably minor for ozone results. Grid cell-specific adjustment factors for monitors are less precise for the west and may not capture local fluctuations. However, exposure tends to be lower in the predominantly non-urban west, and models with finer grids have been applied to three key population centers with significant ozone concentrations. May result in underestimation of benefits in the large urban areas not specifically modeled (e.g., Denver, Seattle) with finer grid. |
| Emissions estimated at the county level (e.g., area source and motor vehicle $NO_x$ and VOC emissions) are spatially and temporally allocated based on land use, population, and other surrogate indicators of emissions activity. Uncertainty and error are introduced to the extent that area source emissions are not perfectly spatially or temporally correlated with these indicators. | Unable to determine based on current information. | Probably minor. Potentially major for estimation of ozone, which depends largely on VOC and $NO_x$ emissions; however, ozone benefits contribute only minimally to net benefit projections in this study. |
| The REMSAD model under-predicted western PM concentrations during fall and winter simulation periods. | Unable to determine based on current information. | Probably minor. Because model results are used in a relative sense (i.e., to develop adjustment factors for monitor data) REMSAD's underestimation of absolute PM concentrations would be unlikely to significantly affect overall results. To the extent that the model is not accurately estimating the relative changes in PM concentrations across regulatory scenarios, or the individual PM components (e.g., sulfates, primary emissions) do not vary uniformly across seasons, the effect could be greater. |

**Table 4-7**
**Key Uncertainties Associated with Air Quality Modeling (continued)**

| Potential Source of Error | Direction of Potential Bias for Net Benefits Estimate | Likely Significance Relative to Key Uncertainties in Net Benefit Estimate* |
|---|---|---|
| Lack of model coverage for acid deposition in Western states. | Underestimate | Probably minor. Because acid deposition tends to be a more significant problem in the eastern U.S. and acid deposition reduction contributes only minimally to net monetized benefits, the monetized benefits of reduced acid deposition in the western states would be unlikely to significantly alter the total estimate of monetized benefits. |
| Uncertainties in biogenic emissions inputs increase uncertainty in the AQM estimates. | Unable to determine based on current information. | Probably minor. Potentially major impacts for ozone outputs, but ozone benefits contribute only minimally to net benefit projections in this study. Uncertainties in biogenics may be as large as a factor of 2 to 3. These biogenic inputs affect the emissions-based VOC/$NO_x$ ratio and, therefore, potentially affect the response of the modeling system to emissions changes. |

* The classification of each potential source of error reflects the best judgement of the section 812 Project Team. The Project Team assigns a classification of "potentially major" if a plausible alternative assumption or approach could influence the overall monetary benefit estimate by approximately five percent or more; if an alternative assumption or approach is likely to change the total benefit estimate by less than five percent, the Project Team assigns a classification of "probably minor."

# Human Health Effects of Criteria Pollutants

Chapter 5

Health benefits resulting from improved air quality constitute a significant portion of the overall benefits of the Clean Air Act Amendments of 1990. As part of the prospective analysis of these amendments, we have identified and, where possible, estimated the magnitude of the health benefits that Americans are likely to enjoy in future years as a result of the CAAA. These health benefits are expressed as avoided cases of air-pollution related health effects such as premature mortality, heart disease, and respiratory illness. This chapter presents an overview of our approach to modeling these changes in adverse health effects, discusses key assumptions associated with this approach, and summarizes modeling results for major health effect categories. Although this chapter focuses predominantly on the human health effects associated with exposure to criteria pollutants, the final section of this chapter presents a discussion of the effects associated with air toxics and stratospheric ozone.

In general, this analysis finds that the CAAA will result in significant reductions in mortality, respiratory illness, heart disease, and other adverse health effects, with much of these reductions resulting from decreases in ambient particulate matter concentrations.

## Analytical Approach

We estimate the impact of the CAAA on human health by analyzing the difference in the expected incidence of adverse health effects between the Pre-CAAA and Post-CAAA regulatory scenarios. As described in Chapter 2, the Pre-CAAA scenario assumes no further controls on criteria pollutant emissions besides those already in place in 1990, while the Post-CAAA scenario assumes full implementation of the 1990 CAAA. For each regulatory scenario, we use the Criteria Air Pollutant Modeling System (CAPMS) to estimate the incidence of health effects for 1990 (base-year), 2000, and 2010. Modeling the incidence of adverse health effects resulting from exposure to criteria air pollutants requires three types of inputs: (1) estimates of the changes in air quality for the Pre- and Post-CAAA scenarios in 2000 and 2010; (2) estimates of the number of people exposed to air pollutants at a given location; and (3) concentration-response (C-R) functions that link changes in air pollutant concentrations with changes in adverse health effects. We discuss each of these inputs in greater detail below.

### *Air Quality*

The development of criteria pollutant concentration estimates for use in the CAPMS model consists of two steps. First, air quality modeling and 1990 base-year monitoring data are used to project ambient pollution levels at monitors throughout the 48 contiguous states. Second, because air quality monitors are neither uniformly nor pervasively distributed across the country, concentration data at monitors are extrapolated to non-monitored areas in order to generate a more comprehensive air quality data set covering the 48 contiguous states and the District of Columbia.

The projections of criteria pollutant concentrations at air pollution monitors are developed as summarized in Chapter 4 and described in detail in Appendix C. Briefly, baseline 1990 concentrations at each monitor are adjusted using monitor- and pollutant-specific adjustment factors to produce estimates of concentrations in 2000 and 2010 for each regulatory scenario. Each adjustment factor reflects the relative change in the concentration of a pollutant in a specific geographic area between 1990 and the target year, as predicted by air quality modeling.

To develop pollutant concentration estimates for the entire continental U.S. we extrapolate the 1990 monitor data and the future-year estimates to the eight kilometer by eight kilometer CAPMS grid cells that cover the 48 contiguous states. Within each of these cells, we calculate an estimated pollutant concentration using data from nearby monitors according to a distance-weighted averaging method described in Appendix D. We then use these grid cell pollutant concentration estimates to predict changes in health effects among the population residing within each cell.

### Population

Health benefits resulting from the CAAA are related to the change in air pollutant exposure experienced by individuals. Because the expected changes in pollutant concentrations vary from location to location, individuals in different parts of the country may not experience the same level of health benefits. This analysis apportions benefits among individuals by matching the change in air pollutant concentration in a CAPMS grid cell with the size of the population that experiences that change.

As a result, we require an estimate of the distribution of the U.S. population among CAPMS grid cells. The grid-cell-specific population counts for 1990 are derived from U.S. Census Bureau block level population data. Grid cell population estimates for future years are extrapolated from 1990 levels using the ratio of future-year and 1990 state-level population estimates provided by the U.S. Bureau of Economic Analysis.[1]

### Concentration-Response Functions

We calculate the benefits attributable to the CAAA as the avoided incidence of adverse health effects. Such benefits can be measured using C-R functions specific to each health effect. C-R functions are equations that relate the change in the number of individuals in a population exhibiting a "response" (in this case an adverse health effect such as respiratory disease) to a change in pollutant concentration experienced by that population. The C-R functions used in CAPMS generate changes in the incidence of an adverse health effect using three values: the grid-cell-specific change in pollutant concentration, the grid-cell-specific population, and an estimate of the change in the number of individuals that suffer an adverse health effect per unit change in air quality.[2] As described in Appendix D, we derive this last factor, as well as the specific form of the C-R equation, from the published scientific literature for each pollutant/health effect relationship of interest.

Using the appropriate C-R functions, CAPMS generates estimates for each grid-cell of the change in incidence of a set of adverse health effects resulting from the incremental change in exposure between the Pre- and Post-CAAA scenarios in 2000 and 2010. For each health effect, CAPMS then generates national health benefits estimates by summing the annual incidence change across all grid cells.

Each criteria pollutant evaluated in the 812 prospective analysis has been associated with multiple adverse health effects. The published scientific literature contains information that supports the estimation of some, but not all, of these effects. Thus, it is not possible currently to estimate all of the human health benefits attributable to the CAAA. In addition, for some of the health effects we do quantify, the current economic literature does not support the estimation of the economic value of these effects. For each of the criteria pollutants we evaluate in this analysis, Table 5-1 presents the health effects that are quantitatively estimated and those that can not currently be quantified. The sixth criteria pollutant, lead (Pb), is not included in this analysis since airborne emissions of lead were virtually eliminated by pre-1990 Clean Air Act programs.

## Key Analytical Assumptions

The modeling of health benefits attributable to the CAAA involves numerous judgments and assumptions to address data limitations and other constraints. Each of these analytical assumptions affects both the accuracy and precision with which we can estimate health benefits of the CAAA, but some as-

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

[2] An estimate of the baseline incidence of the adverse health effect may also be required for certain C-R functions.

**Table 5-1**
**Human Health Effects of Criteria Pollutants**

| Pollutant | Quantified Health Effects | Unquantified Health Effects† |
|---|---|---|
| Ozone | Respiratory symptoms<br>Minor restricted activity days<br>Respiratory restricted activity days<br>Hospital admissions-<br>All Respiratory and<br>All Cardiovascular<br>Emergency room visits for asthma<br>Asthma attacks | Mortality‡<br>Increased airway responsiveness to stimuli<br>Inflammation in the lung<br>Chronic respiratory damage / Premature aging of the lungs<br>Acute inflammation and respiratory cell damage<br>Increased susceptibility to respiratory infection<br>Non-asthma respiratory emergency room visits |
| Particulate Matter ($PM_{10}$, $PM_{2.5}$) | Mortality*<br>Bronchitis - Chronic and Acute<br>New asthma cases<br>Hospital admissions -<br>All Respiratory and<br>All Cardiovascular<br>Emergency room visits for asthma<br>Lower respiratory illness<br>Upper respiratory illness<br>Shortness of breath<br>Respiratory symptoms<br>Minor restricted activity days<br>All restricted activity days<br>Days of work loss<br>Moderate or worse asthma status (asthmatics) | Neonatal mortality‡<br>Changes in pulmonary function<br>Chronic respiratory diseases<br>other than chronic bronchitis<br>Morphological changes<br>Altered host defense mechanisms<br>Cancer<br>Non-asthma respiratory emergency room visits |
| Carbon Monoxide | Hospital Admissions -<br>All Respiratory and<br>All Cardiovascular | Behavioral effects<br>Other hospital admissions<br>Other cardiovascular effects<br>Developmental effects<br>Decreased time to onset of angina<br>Non-asthma respiratory emergency room visits |
| Nitrogen Oxides | Respiratory illness<br>Hospital Admissions -<br>All Respiratory and<br>All Cardiovascular | Increased airway responsiveness to stimuli<br>Chronic respiratory damage / Premature aging of the lungs<br>Inflammation of the lung<br>Increased susceptibility to respiratory infection<br>Acute inflammation and respiratory cell damage<br>Non-asthma respiratory emergency room visits |
| Sulfur Dioxide | Hospital Admissions -<br>All Respiratory and<br>All Cardiovascular<br>In exercising asthmatics:<br>Chest tightness,<br>Shortness of breath, or<br>Wheezing | Changes in pulmonary function<br>Respiratory symptoms in non-asthmatics<br>Non-asthma respiratory emergency room visits |

† Some of the unquantified adverse health effects of air pollution may be associated with adverse health endpoints that we have quantitatively evaluated (e.g., chronic respiratory damage and premature mortality). However, it is likely that the value assigned to the quantified endpoint may not fully capture the value of the associated health effect (e.g., chronic respiratory damage may result in significant pain and suffering prior to mortality). As a result, we include such effects separately in the unquantified health effects column.

‡Appendix D includes detailed discussion of the scientific evidence for these potential health effects and includes illustrative benefit calculations for them. Current uncertainties in our understanding of these effects do not support including these quantitative estimates in the overall CAAA benefits estimate. However, ozone-related mortality may be implicitly quantified in the overall analysis as part of the PM mortality estimate because of the significant correlation between ozone and PM concentrations.

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

sumptions introduce greater uncertainty into the results than others. This section characterizes these key assumptions and the associated uncertainties to allow the reader to gain a better understanding of the potential for misestimation of avoided health effects. In addition, health benefits are presented as ranges to reflect the aggregate effect of the uncertainty in key variables (see Results section below). This section discusses the most important analytical assumptions of this modeling effort, grouped into the following categories: (1) exposure analysis, (2) selection and application of C-R functions, and (3) estimation of changes in PM-related mortality.

## Exposure Analysis

The key analytical assumptions involved in estimating exposure to criteria air pollutants relate to two steps: the extrapolation of air quality data from monitors and the mapping of population data to air quality data.

As discussed above, actual ambient air pollution data are available only for a limited number of monitor sites that are not uniformly distributed across the U.S. Thus, to estimate the impact of air pollution changes on the health of the U.S. population, data from monitors are extrapolated to the cells of a grid that covers the 48 contiguous states and are matched with population data for each grid-cell. Essentially, the extrapolation method uses data from the closest set of monitors surrounding a grid-cell to compute a weighted average concentration for that cell. Monitors closer to the grid cell are assumed to yield a more accurate estimate of air quality in the cell; thus data from these monitors receive more weight than data from more distant monitors when calculating an air quality estimate for the cell.[3] The resulting estimates are uncertain because the geography, weather, land use, and other factors influencing air pollution may differ significantly between a grid cell and the monitor or monitors used to generate estimates of air quality, especially as the monitor-to-grid-cell distance grows.[4] As a result, they may not sufficiently capture local variation in air pollution levels (e.g., hot spots).

However, since the uncertainty in these extrapolated values is inversely proportional to the density of monitors in a given area, and since air quality monitors are more prevalent in high pollution areas than in low pollution areas, this extrapolation method estimates the air quality in high pollution areas (where the potential benefits of the CAAA are greatest) with greater certainty than in low pollution areas. Thus, grid-cell ozone estimates in the eastern U.S., where ozone levels and ozone monitor density are higher, are likely to be more accurate than those in the west, where monitor coverage is more sparse. Also, estimates of concentrations of criteria pollutants, which are measured by a greater number of monitors nationwide (PM, ozone, $SO_2$), are expected to be less uncertain than estimates for CO and $NO_x$, which are measured by considerably fewer monitors.

Air pollutant concentration changes are mapped to grid-cell population data derived from U.S. Census bureau data, and extrapolated to future years using population growth estimates from the U.S. Bureau of Economic Analysis. There are two key assumptions associated with this population mapping. First, we assume the population in each grid cell grows at the same rate as the state population as a whole. As a result, exposures (and potential benefits) in individual grid cells may be either under- or over-estimated if population growth varies from the state average during the 1990 to 2010 period. This uncertainty is likely to be more significant in larger states such as California and Texas, which may have more geographic variability in growth patterns. Also, the effect of this assumption may be less significant for large population centers because their growth rate better approximates the growth rate of the state as a whole. Second, we assume in the exposure analysis that the population in the grid cell is similar in terms of its activity patterns and demographic characteristics to the populations in the studies from which the C-R functions are derived. This is a potentially significant uncertainty which is discussed further in the next section and in Appendix D.

[3] Specifically, monitor data are weighted based on the inverse of the distance between the monitor and the grid-cell center. Additional information on the extrapolation method is provided in Appendix D.

[4] In order to address this issue for long-distance extrapolation (i.e., grid cells greater than 50 kilometers from a monitor), the method is modified to also incorporate air quality modeling predictions for the source and target locations. See Appendix D for details.

## Selection and Application of C-R Functions

We rely on the most recent available, published scientific literature to ascertain the relationship between air pollution and adverse human health effects. The uncertainties underlying those published studies and our method for selecting studies that could be used to derive C-R functions likely contributes to the uncertainty of the health effects results. For example, the uncertainty associated with the current state of the published scientific literature could potentially have two contradictory influences on the results of this analysis. First, to the extent that the published literature may collectively overstate the effects of pollution, our analysis will overstate the benefits of CAAA-related pollution reduction. This overestimation is possible because scientific journals tend to publish research reporting significant associations between pollution and disease more often than research that fails to find such associations. On the other hand, our analysis may underestimate overall health benefits of the CAAA because, as the state of the science evolves, current pollutant/health effect associations may be found to be stronger than previously thought, and new associations may be identified. For example, in recent years, studies have shown the potential health benefits from reductions in ambient PM to be much greater than previously believed. To the extent that the present analysis does not include health effects whose link to air pollution has not been subject to adequate scientific inquiry, this analysis may understate CAAA-related health benefits.

Our method of identifying appropriate C-R functions for use in the benefits analysis may also introduce uncertainty. We evaluate studies using the nine selection criteria summarized in Table 5-2 and described in detail in Appendix D. These criteria include consideration of whether the study was peer-reviewed, the study design and location, and characteristics of the study population, among others. The selection of C-R functions for the benefits analysis is guided by the goal of achieving a balance between comprehensiveness and scientific defensibility. However, to the extent that this selection process may lead to the exclusion of valid studies, the process introduces uncertainty into the analysis. The overall effect of this uncertainty is expected to be minor, given the emphasis of the selection process on scientific validity. Appendix D lists the studies selected for each category of health effects, and presents the associated C-R functions for each criteria pollutant.

Once the C-R functions have been selected, uncertainty may also enter the analysis due to both within-study and across-study variation in C-R functions for individual health effects. Within-study variation refers to the uncertainty and error that may surround a given study's estimate of a C-R function. Health effects studies provide both "best estimates" of the relationship between air quality changes and health effects and a measure of the statistical uncertainty of the relationship. We use statistical simulation modeling techniques to evaluate the overall uncertainty of the results given the uncertainties associated with individual studies. Across-study variation refers to the fact that different published studies of the same pollutant/health effect relationship typically do not report identical findings; in some instances the differences are substantial. These differences can exist even between equally reputable studies and may result in health effect estimates that vary considerably.

Across-study variation can result from two possible causes. One possibility is that studies report different estimates of the single true relationship between a given pollutant and a health effect due to differences in study design, random chance, or other factors. For example, a hypothetical study conducted in New York and one conducted in Seattle may report different C-R functions for the relationship between PM and mortality in part because of differences between these two study populations (e.g., demographics, activity patterns). Alternatively, study results may differ because they are in fact estimating different relationships; that is, the same reduction in PM in New York and Seattle may result in different reductions in premature mortality. This may result from a number of factors, such as differences in the relative sensitivity of these two populations to PM pollution and differences in the composition of PM in these two locations.[5] In either case, where we identify multiple studies that are appro-

[5] PM is a mix of particles of varying size and chemical properties. The composition of PM can vary considerably from one region to another depending on the sources of particulate emissions in each region.

**Table 5-2**
**Summary of Considerations Used in Selecting C-R Functions**

| Consideration | Comments |
|---|---|
| Peer reviewed research | Peer reviewed research is preferred to research that has not undergone the peer review process. |
| Study type | Among studies that consider chronic exposure (e.g., over a year or longer) prospective cohort studies are preferred over cross-sectional studies (a.k.a. "ecological studies") because they control for important confounding variables that cannot be controlled for in cross-sectional studies. If the chronic effects of a pollutant are considered more important than its acute effects, prospective cohort studies may also be preferable to longitudinal time series studies because the latter type of study is typically designed to detect the effects of short-term (e.g. daily) exposures, rather than chronic exposures. |
| Study period | Studies examining a relatively longer period of time (and therefore having more data) are preferred, because they have greater statistical power to detect effects. More recent studies are also preferred because of possible changes in pollution mixes, medical care, and life style over time. |
| Study population | Studies examining a relatively large sample are preferred. Studies of narrow population groups are generally disfavored, although this does not exclude the possibility of studying populations that are potentially more sensitive to pollutants (e.g., asthmatics, children, elderly). However, there are tradeoffs to comprehensiveness of study population. Selecting a C-R function from a study that considered all ages will avoid omitting the benefits associated with any population age category. However, if the age distribution of a study population from an “all population” study is different from the age distribution in the assessment population, and if pollutant effects vary by age, then bias can be introduced into the benefits analysis. |
| Study location | U.S. studies are more desirable than non-U.S. studies because of potential differences in pollution characteristics, exposure patterns, medical care system, and life style. |
| Pollutants included in model | Models with more pollutants are generally preferred to models with fewer pollutants, though careful attention must be paid to potential collinearity between pollutants. Because PM has been acknowledged to be an important and pervasive pollutant, models that include some measure of PM are highly preferred to those that do not. |
| Measure of PM | $PM_{2.5}$ and $PM_{10}$ are preferred to other measures of particulate matter, such as total suspended particulate matter (TSP), coefficient of haze (COH), or black smoke (BS) based on evidence that $PM_{2.5}$ and $PM_{10}$ are more directly correlated with adverse health effects than are these other measures of PM. |
| Economically valuable health effects | Some health effects, such as forced expiratory volume and other technical measurements of lung function, are difficult to value in monetary terms. These health effects are not quantified in this analysis. |
| Non-overlapping endpoints | Although the benefits associated with each individual health endpoint may be analyzed separately, care must be exercised in selecting health endpoints to include in the overall benefits analysis because of the possibility of double counting of benefits. Including emergency room visits in a benefits analysis that already considers hospital admissions, for example, will result in double counting of some benefits if the category “hospital admissions” includes emergency room visits. |

priate for estimating a given health effect, we use the multiple C-R estimates, applied to the entire U.S., to derive a range of possible results for that health effect.

Whether this analysis estimates the C-R relationship between a pollutant and a given health endpoint using a single function from a single study or using multiple C-R functions from several studies, each C-R relationship is applied throughout the U.S. to generate health benefit estimates. However, to the extent that pollutant/health effect relationships are region-specific, applying a location-specific C-R function at all locations in the U.S. may result in overestimates of health effect changes in some locations and underestimates of health effect changes in other locations. It is not possible, however, to know the extent or direction of the overall effect on health benefit estimates introduced by application of a single C-R function to the entire U.S. This may be a sig-

nificant uncertainty in the analysis, but the current state of the scientific literature does not allow for a region-specific estimation of health benefits.

## PM-Related Mortality

This section discusses the estimation of one of the most serious health impacts of air pollution: premature mortality associated with PM exposure. This section consists of three parts. It begins with a discussion of the uncertainties surrounding the PM/mortality relationship. Then, it presents specific factors to consider when selecting a PM mortality C-R function. It ends with a brief discussion of the advantages and disadvantages of the study we selected for the PM mortality analysis: Pope et al., 1995.

### Uncertainties in the PM Mortality Relationship

Health researchers have consistently linked air pollution, especially PM, with excess mortality. A substantial body of published scientific literature recognizes a correlation between elevated PM concentrations and increased mortality rates. However, there is much about this relationship that is still uncertain.[6] These uncertainties include:

- **Causality.** For this analysis, we assume a causal relationship between exposure to elevated PM and premature mortality, based on the evidence of a correlation between PM and mortality reported in the scientific literature. This assumption is necessary because the epidemiological studies on which this analysis relies, by design, can not definitively prove causation.

- **Other Pollutants.** PM concentrations are correlated with the concentrations of other criteria pollutants, such as ozone and CO, and it is unclear how much each pollutant may influence elevated mortality rates. Recent studies have explored whether ozone and CO may have mortality effects independent of PM, but we do not view the evidence as sufficient to include such effects in the overall CAAA-related health benefits estimate.[7] As a result, we use the reported PM/mortality relationship as a proxy for the mortality effects of the air pollutant mixture.

- **Shape of the C-R Function.** The shape of the true PM mortality C-R function is uncertain, but this analysis assumes the C-R function to have a log-linear form (as derived from the literature) throughout the relevant range of exposures.[8] If this is not the correct form of the C-R function, or if certain scenarios (e.g., 2010 Pre-CAAA) predict concentrations well above the range of values for which the C-R function was fitted, avoided mortality may be mis-estimated.

- **Regional Differences.** As discussed earlier, significant variability exists in the results of different PM studies. This variability may reflect regionally-specific C-R functions resulting from regional differences in factors such as the physical and chemical composition of PM. If true regional differences exist, applying these C-R functions to regions other than the study location would result in mis-estimation of effects in these regions.

- **Exposure/Mortality Lags.** It is currently unknown whether there is a time lag — a delay between changes in PM exposures and changes in mortality rates — in the chronic PM/mortality relationship. The existence of such a lag could be important for the valuation of benefits, if one were to assume that lagged incidences of premature mortality should be discounted over the period between when the fatal increment of exposure is experienced and premature mortality actually occurs. Although there is no specific scientific evidence of the existence or structure of a PM effects lag, current scientific literature on adverse health effects such as those associated with PM (e.g., smoking-related disease) leaves us skeptical that all inci-

[6] The morbidity studies used in this analysis may also be subject to many of the uncertainties listed in this section.

[7] Appendix D discusses the evidence linking both ozone and CO with mortality. It also describes and presents the results of an illustrative analysis estimating CAAA-related reductions in ozone-related mortality using currently available studies.

[8] C-R functions for other health effects may be assumed to be linear or log-linear. See Appendix D for more details.

dences of premature mortality associated with a given incremental change in PM exposure would occur in the same year as the exposure reduction. This same literature implies that lags of up to a few years are plausible, and we chose to assume a five-year lag structure, with 25 percent of deaths occurring in the first year, another 25 percent in the second year, and 16.7 percent in each of the remaining three years.

- **Cumulative Effects.** We attribute the PM/mortality relationship used in this study (Pope et al., 1995) primarily to PM-associated cumulative damage to the cardiopulmonary system, since the short-term mortality estimates reported in time-series studies account for only a minor fraction of total excess mortality. However, the relative roles of exposure duration and exposure level remain unknown at this time.

## Selection of a PM Mortality C-R Function

In addition to the study selection criteria listed in Table 5-2, we consider three additional factors when selecting a PM mortality function. The first focuses on the PM indicator (i.e., $PM_{10}$ or $PM_{2.5}$), the second focuses on whether the study measured short-term or long-term PM exposure, and the third focuses on whether the study used a cohort or ecologic design.

Current research suggests that particle size matters when estimating the health impacts of PM. Particulate matter is a heterogeneous mixture that includes particles of varying sizes. Fine PM is generally viewed as having a more harmful impact than coarse PM, especially for coarse particles larger than 10μm in aerodynamic diameter, although it is not clear to what extent this may differ by the type of health effect or the exposed population. While one cannot necessarily assume that coarse PM has no adverse impact on health, we prefer the use of $PM_{2.5}$ as the best currently available measure of the impact of PM on mortality.[9]

Two types of exposure studies (short-term and long-term) have been used to estimate a PM/mortality relationship. Short-term exposure studies attempt to relate short-term (often day-to-day) changes in PM concentrations and changes in daily mortality rates up to several days after a period of elevated PM concentrations. Long-term exposure studies examine the potential relationship between longer-term (e.g., annual) changes in exposure to PM and annual mortality rates. Researchers have found significant correlations using both types of studies; however, for this analysis, we rely exclusively on long-term studies to quantify PM mortality effects, though the short-term studies provide additional scientific evidence supporting the PM/mortality relationship.

Because short-term studies focus only on the acute effects associated with daily peak exposures, they are unable to evaluate the degree to which observed excess mortality is premature,[10] and they may underestimate the C-R coefficient because they do not account for the cumulative mortality effects of long-term exposures (i.e., exposures over many years rather than a few days). Long-term studies, on the other hand, are able to discern changes in mortality rates due to long-term exposure to elevated air pollution concentrations, and are not limited to measuring mortalities that occur within a few days of a high-pollution event (though they may not predict cases of premature mortality that were only hastened by a few days). Consequently, the use of C-R functions derived from long-term studies is likely to result in a more complete assessment of the effect of air pollution on mortality risk. However, to the extent that long-term studies fail to capture acute mortality effects related to peak exposures, the use of long-term mortality studies may underestimate CAAA-related avoided mortality benefits.

Among long-term PM studies, we prefer studies using a prospective cohort design to those using an ecologic or population-level design. Prospective

[9] Due to the relative abundance of studies using $PM_{10}$, however, and the reasonably good correlation between $PM_{2.5}$ and $PM_{10}$, the 812 prospective analysis also uses $PM_{10}$ studies to estimate the impact of PM on non-mortality health effects.

[10] This can be important in cost-benefit analysis if benefits are estimated in terms of life-years lost. In short-term studies evaluating peak pollution events, 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.

cohort studies follow individuals forward in time for a specified period, periodically evaluating each individual's exposure and health status. Population-level ecological studies assess the relationship between population-wide health information (such as counts for daily mortality) and ambient levels of air pollution. Prospective cohort studies are preferred because they are better at controlling a source of uncertainty known as "confounding." Confounding is the mis-estimation of an association that results if a study does not control for factors that are correlated with both the outcome of interest (e.g., mortality) and the exposure of interest (e.g., PM exposure). For example, smoking is associated with mortality. If populations in high PM areas tend to smoke more than populations in low PM areas, and a PM exposure study does not include smoking as a factor in its model, then the mortality effects of smoking may be erroneously attributed to PM, leading to an overestimate of the risk from PM. Prospective cohort studies are better at controlling for confounding than ecologic studies because the former follow a group of individuals forward in time and can gather individual-specific information on important risk factors such as smoking. However, it is always possible, even in well-designed studies, that a relevant risk factor (e.g., climate, the presence of other pollutants) may not have been adequately considered or controlled for. As a result, it is possible that differences in mortality rates 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 addressed in the exposure models.

## The Pope Study

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). Of these three studies, we prefer using the Pope et al. study as the basis for developing the primary PM mortality estimates in this analysis. Pope et al. studied the largest cohort, had the broadest geographic scope, and effectively controlled for potentially significant sources of confounding.

Pope et al. examined a much larger population (over 295,000) and many more locations (50 metropolitan areas) than either the Dockery study or the Abbey study. The Dockery study covered a cohort of over 8,000 individuals in six U.S. cities, and the Abbey study covered a cohort of 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 unique in that it followed a largely white and middle class population. The use of this study population reduces the potential for confounding because it decreases the likelihood that differences in premature mortality across locations were attributable to differences in socioeconomic status or related factors rather than PM. However, the demographics of the study population may also produce a downward bias in the PM mortality coefficient, because short-term studies indicate that the effects of PM tend to be significantly greater among groups of lower socioeconomic status.

Although it is the strongest of the PM cohort studies, Pope et al. does have some limitations. For example, Pope et al. did not consider the migration of cohort members across study cities, which would cause exposures to be more similar across individuals than those indicated by assigning city-specific annual average pollution levels to each member of the cohort. As intercity migration increases among cohort members, the exposure experienced by migrating individuals will tend toward an intercity mean. If this migration is significant and is ignored, approximating true differences in exposure levels by differences in city-specific annual average PM levels will exaggerate changes in exposure, resulting in a downward bias of the PM coefficient. This occurs because a given difference in mortality rates is being associated with a larger difference in PM levels than that actually experienced by individuals in the study cohort. When the relationship between elevated PM exposure and premature mortality derived from the

Pope et al. study is applied in the present analysis, the effect of the potential mis-specification of exposure due to migration in the underlying study is to underestimate PM-related mortality reduction benefits attributable to the CAAA.

Also, Pope et al. only included PM when estimating a C-R function. Because PM concentrations are correlated with the concentrations of other criteria air pollutants (e.g., ozone), and because these other pollutants may be correlated with premature mortality (see Appendix D), the PM risk estimate may be overestimated because it includes the mortality impacts of these confounders. However, in an effort to avoid overstating benefits, and because the evidence associating mortality with PM exposure is stronger than for other pollutants, the 812 Prospective analysis uses PM as a surrogate for PM and related criteria pollutants.

Although we use the Pope study exclusively to derive our primary estimates of avoided mortality, the C-R function based on Dockery et al. (1993) may provide a reasonable alternative estimate. While the Dockery et al. study used a smaller sample of individuals from fewer cities than the study by Pope et al., it features improved exposure estimates, a slightly broader study population (adults aged 25 and older), and a follow-up period nearly twice as long as that of Pope et al. We present an alternative estimate of the premature adult mortality associated with long-term PM exposure based on Dockery et al. (1993) in Chapter 8 and in Appendix D. We emphasize, however, that the estimate based on Pope et al. (1995) is our primary estimate of the effect of the 1990 Amendments on this important health effect.

## Health Effects Modeling Results

This section presents a summary of the differences in health effects resulting from improvements in air quality between the Pre-CAAA and Post-CAAA scenarios. Table 5-3 summarizes the CAAA-related avoided health effects in 2010 for each study included in the analysis. The mean estimate is presented as the Primary Central estimate, the 5th percentile observation from the statistical uncertainty modeling is presented as the Primary Low estimate, and the 95th percentile observation is presented as the Primary High estimate of the number of avoided cases of each endpoint.[11] To provide context for these results, Table 5-3 also expresses the mean reduction in incidence for each adverse health effect as a percentage of the baseline incidence of that effect (extrapolated to the appropriate future year) for the population considered (e.g., adults over 30 years of age). In general, because the differences in air quality between the Pre- and Post-CAAA scenarios are expected to increase from 1990 to 2010 and because population is also expected to increase during that time, the health benefits attributable to the CAAA are expected to increase consistently from 1990 to 2010. More detailed results are presented in Appendix D.

### *Avoided Premature Mortality Estimates*

Table 5-3 summarizes the avoided mortality due to reductions in PM exposure in 2010 between the Pre- and Post-CAAA scenarios. As this table shows, our Primary Central estimate implies that PM reductions due to the CAAA in 2010 will result in 23,000 avoided deaths, with a Primary Low and Primary High bound on this estimate of 14,000 and 32,000 avoided deaths, respectively. The Primary Central estimate of 23,000 avoided deaths represents roughly one percent of the projected annual non-accidental mortality of adults aged 30 and older in the year 2010. Additionally, Table 5-4 summarizes the distribution of avoided mortality for 2010 by age cohort, along with the expected remaining life-span (i.e., the life years lost) for the average person in each age cohort. The majority of the estimated deaths occur in people over the age of 65 (due to their higher baseline mortality rates), and this group has a shorter life expectancy relative to other age groups. The life years lost estimates might be higher if data were available for PM-related mortality in the under 30 age group.

[11] The Primary Low, Primary Central and Primary High health benefit estimates represent points on a distribution of estimated incidence changes for each health effect. This distribution reflects the uncertainty associated with the coefficient of the C-R function for each health endpoint. More information about C-R function uncertainty and the uncertainty modeling that generates the results distributions is presented in Appendix D.

**Table 5-3**
**Change in Incidence of Adverse Health Effects Associated with Criteria Pollutants in 2010 (Pre-CAAA minus Post-CAAA) – 48 State U.S. Population (avoided cases per year)**

| | | 2010 | | | % of Baseline Incidences for the mean estimates [a] |
|---|---|---|---|---|---|
| **Endpoint** | **Pollutant** | **5th %** | **mean** | **95th %** | **2010** |
| **Mortality** | | | | | |
| ages 30 and older | PM | 14,000 | 23,000 | 32,000 | 1.00% |
| **Chronic Illness** | | | | | |
| chronic bronchitis | PM | 5,000 | 20,000 | 34,000 | 3.14% |
| chronic asthma | $O_3$ | 1,800 | 7,200 | 12,000 | 3.83% |
| Hospitalization | | | | | |
| respiratory admissions | PM, CO, $NO_2$, $SO_2$, $O_3$ | 13,000 | 22,000 | 34,000 | 0.62% |
| cardiovascular admissions | PM, CO, $NO_2$, $SO_2$, $O_3$ | 10,000 | 42,000 | 100,000 | 0.86% |
| emergency room visits for asthma | PM, $O_3$ | 430 | 4,800 | 14,000 | 0.55% |
| **Minor Illness** | | | | | |
| acute bronchitis | PM | 0 | 47,000 | 94,000 | 5.06% |
| upper respiratory symptoms | PM | 280,000 | 950,000 | 1,600,000 | 0.86% |
| lower respiratory symptoms | PM | 240,000 | 520,000 | 770,000 | 3.57% |
| respiratory illness | $NO_2$ | 76,000 | 330,000 | 550,000 | 10.44% |
| moderate or worse asthma[c] | PM | 80,000 | 400,000 | 720,000 | 0.24% |
| asthma attacks[c] | $O_3$, PM | 920,000 | 1,700,000 | 2,500,000 | 1.04% |
| chest tightness, shortness of breath, or wheeze | $SO_2$ | 290 | 110,000 | 520,000 | 0.003% |
| shortness of breath | PM | 26,000 | 91,000 | 150,000 | 1.69% |
| work loss days | PM | 3,600,000 | 4,100,000 | 4,600,000 | 0.94% |
| minor restricted activity days / any of 19 respiratory symptoms[d] | $O_3$, PM | 25,000,000 | 31,000,000 | 37,000,000 | 2.15% |
| restricted activity days[c] | PM | 10,000,000 | 12,000,000 | 13,000,000 | 1.00% |

[a] The baseline incidence generally is the same as that used in the C-R function for a particular health effect. However, there are a few exceptions. To calculate the baseline incidence rate for respiratory-related hospital admissions, we used admissions for persons of all ages for International Classification of Disease (ICD) codes 460-519; for cardiovascular admissions, we used admissions for persons of all ages for ICD codes 390-429; for emergency room visits for asthma, we used the estimated ER visit rate for persons of all ages; for chronic bronchitis we used the incidence rate for individuals 27 and older; for the pooled estimate of minor restricted activity days and any-of-19 respiratory symptoms, we used the incidence rate for minor restricted activity days.

[b] Percentage is calculated as the ratio of avoided mortality to the projected baseline annual non-accidental mortality for adults aged 30 and over. Non-accidental mortality was approximately 95% of total mortality for this subpopulation in 2010.

[c] These health endpoints overlap with the "any-of-19 respiratory symptoms" category. As a result, although we present estimates for each endpoint individually, these results are not aggregated into the total benefits estimates.

[d] Minor restricted activity days and any-of-19 respiratory symptoms have overlapping definitions and are pooled.

### Non-Fatal Health Impacts

We report non-fatal health effects estimates in a similar manner 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 2010 only is characterized in Table 5-3 with 5th percentile, mean, and 95th percentile estimates which correspond to the Primary Low, Primary Central, and Primary High estimates, respectively. All estimates are expressed as new cases avoided in 2010, with the following exceptions. Hospital admissions reflect admissions for a range of respiratory and cardiovascular diseases, and these results, along with emergency room visits for asthma, do not necessarily represent the avoidance of new cases of disease (i.e., air pollution may simply exacerbate an existing condition, resulting in an emergency room visit or hospital admission). Further, each admission is only counted once, regardless of the length of stay in the hospital. "Shortness of breath" is expressed in terms of symptom 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.

## Avoided Health Effects of Other Pollutants

This section discusses the health effects associated with non-criteria air pollutants regulated by the Clean Air Act Amendments of 1990. It first discusses the effects of pollutants known as "air toxics", and then summarizes the effects associated with stratospheric ozone depleting substances.

### Avoided Effects of Air Toxics

In addition to addressing the control of criteria pollutants, the Clean Air Act Amendments revamped regulations for air toxics — defined as non-criteria pollutants which can cause adverse effects to human health and to ecological resources — under section 112 of the Act. Among other changes, the 1990 Amendments establish a list of air toxics to be regulated, require EPA to establish air toxic emissions standards based on maximum achievable control technology (MACT standards), and include a provision that requires EPA to establish more stringent air toxic standards if MACT controls do not sufficiently protect the public health against residual risks. Control of air toxics is expected to result both from these changes and from incidental control due to changes in criteria pollutant programs.

**Table 5-4**
**Mortality Distribution by Age in Primary Analysis (2010 only), Based on Pope et al. (1995)[a]**

| Age Group | Proportion of Premature Mortality by Age [b] | Life Expectancy (years) |
|---|---|---|
| Infants | not estimated | -- |
| 1-29 | not estimated | -- |
| 30-34 | 1% | 48 |
| 35-44 | 4% | 38 |
| 45-54 | 6% | 29 |
| 55-64 | 12% | 21 |
| 65-74 | 24% | 14 |
| 75-84 | 30% | 9 |
| 85+ | 24% | 6 |

[a] Results based on PM-related mortality incidence estimates for the 48 state U.S. population.
[b] Percentages may not sum to 100 percent due to rounding.

For several decades, the primary focus of risk assessments and control programs designed to reduce air toxics has been cancer. According to present EPA criteria, over 100 air toxics are 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).[12] We note, however, that the results of this analysis are based, in part, on conservative, upper-bound estimates of chemical specific risk factors.

In addition to cancer, inhalation of air toxics compounds can cause a wide variety of health effects, including neurotoxicity, respiratory problems, and adverse reproductive and developmental effects. However, there has been considerably less work done to assess the magnitude of non-cancer effects from air toxics.

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.

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.[13]

Unfortunately, the effects of air toxics emissions reductions could not be quantified for the present study. Unlike criteria pollutants, monitoring data for air toxics are relatively scarce, and the data that do exist cover only a handful of pollutants. Emissions inventories are very limited and inconsistent, and air quality modeling has only been performed for a few source categories. In addition, the scientific literature on the effects of air toxics is generally much weaker than that available for criteria pollutants. Appendix I presents 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 prospective studies.

## Avoided Health Effects for Provisions to Protect Stratospheric Ozone

We estimate benefits of stratospheric ozone protection programs by relying on analyses conducted to support a series of regulatory support documents for these provisions. The series of basic steps to arrive at physical effects estimates — from emissions estimation, atmospheric modeling, exposure assessment, and dose-response characterization — is similar to that used to estimate effects of criteria pollutants, but the details of each modeling step are vastly different. The emissions and atmospheric modeling yields estimates of changes in ultraviolet-b (UV-b) radiation, and the exposure and dose-response analyses then yield estimates of the effects of changes in UV-b radiation, including human health, welfare, and ecological effects. Appendix G provides a detailed description of the methodology and sources used to generate these estimates. Several of the benefits can be identified but cannot yet be reliably quantified, and so are described qualitatively.

The quantified physical effects estimates of sections 604 and 606 of Title VI, the provisions that provide the primary controls on production and release of CFCs and HCFCs generate about 98 percent of the monetized quantified benefits estimate. The quantified health benefits include the following: reduced incidences of mortality and morbidity associated with skin cancer (melanoma and nonmelanoma); and reduced incidences of cataracts

[12] These pollutants included PIC (products of incomplete combustion), 1,3-butadiene, hexavalent chromium, benzene, formaldehyde, chloroform, asbestos, arsenic, ethylene dibromide, dioxin, gasoline vapors, and ethylene dichloride. See U.S. EPA, Cancer Risk from Outdoor Exposure to Air Toxics. EPA-450/1-90-004f. Prepared by EPA/OAR/OAQPS.

[13] 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.

and their associated pain and suffering.[14] Using the change in UV radiation dose, we estimate the number of additional cases of skin cancer (melanoma and nonmelanoma) and cataracts. Because the baseline levels of all of these UV-related health effects tend to be higher for older people and for those with lighter skins, EPA's method for projecting future incremental skin cancers and cataracts incorporates these factors in its benefits estimates.[15] We present a brief summary of these benefits in Table 5-5, and the analysis is described in detail in Appendix G.

To calculate the number of deaths from melanoma, the model uses a dose response function similar to the C-R functions for criteria pollutants. For nonmelanoma, the model estimates the number of deaths by assuming that a fixed percentage of the total nonmelanoma cases will result in death.[16] We estimate that from 1990 to 2165 sections 604 and 606 will result in 6.3 million avoided deaths from skin cancer, 27.5 million avoided cataract cases, and 299.0 million cases of non-fatal skin cancers (melanoma and nonmelanoma). The unquantified effects of sections 604 and 606 include avoided pain and suffering from skin cancer and human health and environmental benefits outside the United States.

**Table 5-5**
**Major Health Benefits of Provisions to Protect Stratospheric Ozone (CAAA Sections 604, 606, And 609)**

| Health Effects- Quantified | Estimate | Basis for Estimate |
|---|---|---|
| Melanoma and nonmelanoma skin cancer (fatal) | 6.3 million lives saved from skin cancer in the U.S. between 1990 and 2165 | Dose-response function based on UV exposure and demographics of exposed populations.[1] |
| Melanoma and nonmelanoma skin cancer (non-fatal) | 299 million avoided cases of non-fatal skin cancers in the U.S. between 1990 and 2165 | Dose-response function based on UV exposure and demographics of exposed populations.[1] |
| Cataracts | 27.5 million avoided cases in the U.S. between 1990 and 2165 | Dose-response function uses a multivariate logistic risk function based on demographic characteristics and medical history. [1] |
| **Health Effects- Unquantified** | | |
| Skin cancer: reduced pain and suffering | | |
| Reduced morbidity effects of increased UV. For example,<br>• reduced actinic keratosis (pre-cancerous lesions resulting from excessive sun exposure)<br>• reduced immune system suppression. | | |

Notes:
[1] For more detail see EPA's *Regulatory Impact Analysis: Protection of Stratospheric Ozone* (1988).
[2] Note that the ecological effects, unlike the health effects, do not reflect the accelerated reduction and phaseout schedule of section 606.
[3] Benefits due to the section 606 methyl bromide phaseout are not included in the benefits total because annual incidence estimates are not currently available.

[14] Quantitative estimates presented in Appendix G also include reduced crop damage associated with UV-b radiation and tropospheric ozone; reduced damage to fish harvests associated with UV-b radiation; and reduced polymer degradation from UV-b radiation. The derivation of these effects is described in more detail in Chapter 7.

[15] The dose-response equation is (fractional change in incidence) = (fractional change in UV-b dose + 1)$^b$ -1, where b (the biological amplification factor) equals the percent change in incidence associated with a one percent change in dose. More information about the origins of the models can be found in Appendix G.

[16] Scotto, Fears, and Fraumeni, U.S. Department of Health and Human Services, NIH, "Incidence of Nonmelanoma Skin Cancer in the United States," 1981, pages 2, 7, and 13.

## Uncertainty in the Health Effects Analysis

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 (i.e., overestimation or underestimation), and the potential significance of each of these uncertainties for the overall net benefit results of the analysis are summarized in Table 5-6. As shown in this table, the decisions made to overcome the problems of limited data, the inadequacy of the currently available scientific literature, and other constraints do not clearly bias the overall results of this analysis in one particular direction.

**Table 5-6**
**Key Uncertainties Associated with Human Health Effects Modeling (continued)**

| Potential Source of Error | Direction of Potential Bias for Net Benefits Estimate | Likely Significance Relative to Key Uncertainties in Net Benefit Estimate* |
|---|---|---|
| Application of C-R relationships only to those subpopulations matching the original study population. | Underestimate. | Potentially major. The C-R functions for several health endpoints (including PM-related premature mortality) were applied only to subgroups of the U.S. population (e.g., adults over 30) and thus may underestimate the whole population benefits of reductions in pollutant exposures. In addition, the demographics of the study population in the Pope et al. study (largely white and middle class) may result in an underestimate of PM-related mortality, because the effects of PM tend to be significantly greater among groups of lower socioeconomic status. |
| No quantification of health effects associated with exposure to air toxics. | Underestimate | Potentially major. According to EPA criteria, over 100 air toxics are known or suspected carcinogens, and many air toxics are also associated with adverse health effects such as neurotoxicity, reproductive toxicity, and developmental toxicity. Unfortunately, current data and methods are insufficient to develop (and value) quantitative estimates of the health effects of these pollutants. |
| Use of long-term global warming estimates in Title VI analysis that show more severe warming than is now generally anticipated. | Overestimate (for Title VI estimate only) | Potentially major. Global warming can accelerate the pace of stratospheric ozone recovery; if warming is less severe than anticipated at the time the Title VI analyses were conducted, the modeled pace of ozone recovery may be overestimated, suggesting benefits of the program could be delayed, perhaps by many years. The magnitude of estimated Title VI benefits suggests that the impact of delaying benefits could be major. |
| The quantitative analysis of Title VI (see next section) does not account for potential increases in averting behavior (i.e., people's efforts to protect themselves from UV-b radiation). | Unable to determine based on current information. | Potentially major. Murdoch and Thayer (1990) estimate that the cost-of-illness estimates for nonmelanoma skin cancer cases between 2000 and 2050 may be almost twice the estimated cost of averting behavior (application of sunscreen). Our Title VI analysis relies on epidemiological studies, which incorporate averting behavior as currently practiced. Omission of future increases in averting behavior, however, may overstate the benefits of reduced emissions of ozone-depleting chemicals. Benefits could be understated if individuals alter their behaviors in ways that could increase exposure or risk (e.g., sunbathing more frequently). A recent European study by Autier et al. (1999) found that the use of high sun protection factor (SPF) sun screen is associated with increased frequency and duration of sun exposure. |

**Table 5-6**
**Key Uncertainties Associated with Human Health Effects Modeling (continued)**

| Potential Source of Error | Direction of Potential Bias for Net Benefits Estimate | Likely Significance Relative to Key Uncertainties in Net Benefit Estimate* |
|---|---|---|
| Analysis assumes a causal relationship between PM exposure and premature mortality based on strong epidemiological evidence of a PM/mortality association. However, epidemiological evidence alone cannot establish this causal link. | Unable to determine based on current information. | Potentially major. A basic underpinning of this analysis, this assumption is critical to the estimation of health benefits. However, the assumption of causality is suggested by the epidemiologic evidence and is consistent with current practice in the development of a best estimate of air pollution-related health benefits. At this time, we can identify no basis to support a conclusion that such an assumption results in a known or suspected overestimation bias. |
| Across-study variance / application of regionally derived C-R estimates to entire U.S. | Unable to determine based on current information. | Potentially major. The differences in the expected changes in health effects calculated using different underlying studies can be large. If differences reflect real regional variation in the PM/mortality relationship, applying individual C-R functions throughout the U.S. could result in considerable uncertainty in health effect estimates. |
| Estimate of non-melanoma skin cancer mortality resulting from reductions in stratospheric ozone is calculated indirectly, by assuming the mortality rate is a fixed percentage of non-melanoma incidence. | Unable to determine based on current information. | Potentially major. New data on the death rate for non-melanoma skin cancer may significantly influence the Title VI mortality estimate. Some preliminary estimates suggest that this estimate may need to be adjusted downward. |
| The baseline incidence estimate of chronic bronchitis based on Abbey et al. (1995) excluded 47 percent of the cases reported in that study because those reported "cases" experienced a reversal of symptoms during the study period. These "reversals" may constitute acute bronchitis cases that are not included in the acute bronchitis analysis (based on Dockery et al., 1996). | Underestimate. | Probably minor. The relative contribution of acute bronchitis cases to the overall benefits estimate is small compared to other health benefits such as avoided mortality and avoided chronic bronchitis. |
| CAAA fugitive dust controls implemented in PM non-attainment areas would reduce lead exposures by reducing the re-entrainment of lead particles emitted prior to 1990. This analysis does not estimate these benefits. | Underestimate | Probably minor. While the health and economic benefits of reducing lead exposure can be substantial (e.g., see section 812 Retrospective Study Report to Congress), most additional fugitive dust controls implemented under the Post-CAAA scenario (e.g., unpaved road dust suppression, agricultural tilling controls, etc) tend to be applied in relatively low population areas. |
| Exclusion of C-R functions from short-term exposure studies in PM mortality calculations. | Underestimate | Probably minor. Long-term PM exposure studies may be able to capture some of the impact of short-term peak exposure on mortality; however the extent of overlap between the two study types is unclear. |

**Table 5-6**
**Key Uncertainties Associated with Human Health Effects Modeling (continued)**

| Potential Source of Error | Direction of Potential Bias for Net Benefits Estimate | Likely Significance Relative to Key Uncertainties in Net Benefit Estimate* |
|---|---|---|
| Age-specific C-R functions for PM related premature mortality not reported by Pope et al. (1995). Estimation of the degree of life-shortening associated with PM-related mortality used a single C-R function for all applicable age groups. | Unable to determine based on current information. | Unknown, possibly major 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 that PM-related mortality occurs over a period of five-years following the critical PM exposure. Analysis assumes that 25 percent of deaths occur in year one, 25 percent in year two, and 16.7 percent in each of the remaining three years. | Unable to determine based on current information. | Probably minor. If the analysis underestimates the lag period, benefits will be overestimated, and vice-versa. However, available epidemiological studies do not provide evidence of the existence or potential magnitude of a lag between exposure and incidence. Thus, an underestimate of the lag seems unlikely. If the assumed lag structure is an overestimate, even if benefits are fully discounted from the future year of death, application of reasonable discount rates over this period would not significantly alter the monetized benefit estimate. |
| Extrapolation of criteria pollutant concentrations to populations distant from monitors. | Unable to determine based on current information. | Probably minor. Extrapolation method is most accurate in areas where monitor density is high. Monitor density tends to be highest in areas with high criteria pollutant exposures; thus most of this uncertainty affects low exposure areas where benefits are likely to be low. In addition, an enhanced extrapolation method incorporating modeling results is used for areas far (> 50 km) from a monitor. |
| Exposure analysis in areas beyond 50 km is based on a new technique that relies on the direct use of air quality modeling results in combination with adjusted monitor data. | Unable to determine based on current information. | Probably minor. The new technique is used for less than 10 percent of the country for PM exposure, and less than 15 percent for ozone. The approach we use should be more accurate than the alternative approach of linear interpolation over long distances. The new method nonetheless requires further testing against monitor data to access its accuracy. |
| Pope et al. (1995) study did not include pollutants other than PM. | Unable to determine based on current information. | Probably minor. If ozone and other criteria pollutants correlated with PM contribute to mortality, that effect may be captured in the PM estimate. Thus, PM is essentially used as a surrogate for a mix of pollutants. This uncertainty does make it difficult to disaggregate avoided mortality benefits by pollutant, however other studies (besides Pope) suggest that PM is the dominant factor in premature mortality. |

* The classification of each potential source of error reflects the best judgement of the section 812 Project Team. The Project Team assigns a classification of "potentially major" if a plausible alternative assumption or approach could influence the overall monetary benefit estimate by approximately five percent or more; if an alternative assumption or approach is likely to change the total benefit estimate by less than five percent, the Project Team assigns a classification of "probably minor."

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# Economic Valuation of Human Health Effects

Chapter 6

The reduced incidence of physical effects is a valuable measure of health benefits for individual endpoints; however, to compare or aggregate benefits across endpoints, the benefits must be monetized. Assigning a dollar value to avoided incidences of each effect permits us to sum monetized benefits realized as a result of the CAAA, and compare them with the associated costs.

In the 812 prospective analysis, we have two broad categories of benefits, health and welfare benefits. Human health effects include mortality and morbidity endpoints, which are presented in this chapter. Welfare effects include agricultural and ecological benefits, visibility, and worker productivity, which are covered in the following chapter. We obtain valuation estimates from the economic literature, and report them in "dollars per case reduced for health effects" and "dollars per unit of avoided damage for welfare effects".[1] Similar to estimates of physical effects provided by health studies, we report each of the monetary values of benefits applied in this analysis in terms of a central estimate and a probability distribution around that value. The statistical form of the probability distribution varies by endpoint. For example, we use a Weibull distribution to describe the estimated dollar value of an avoided premature mortality, while we assume the estimate for the value of a reduced case of acute bronchitis is uniformly distributed between a minimum and maximum value.

Although human health benefits of the 1990 Amendments are attributed to reduced emissions of criteria pollutants (Titles I through V) and reduced emission of ozone depleting substances (Title VI), this chapter focuses only on the valuation of human health effects attributed to the reduction of criteria pollutants. The chapter begins with an brief review of the economic concepts behind valuing human health effects in a cost-benefit context and a summary of the unit values applied to health endpoints. We follow with a discussion of how we derive valuation estimates for specific health effects. We then present the results of this analysis. We conclude the chapter with a review of the uncertainties associated with benefits valuation.

Our analysis indicates that the benefit of avoided premature mortality risk reduction dominates the overall net benefit estimate. This is, in part, due to the high monetary value assigned to the avoidance of premature mortality relative to the unit value of other health endpoints. Because of the critical importance of this endpoint in the study's results, this chapter pays particular attention to the major challenges to valuing mortality risk reductions and the limitations of the estimate we apply in this analysis. There are also significant reductions in short term and chronic health effects and a substantial number of health (and welfare) benefits that we could not quantify or monetize.

## Valuation of Benefit Estimates

In an environmental benefit-cost analysis, the dollar value of an environmental benefit (e.g., a health-related improvement due to environmental quality) enjoyed by an individual is the dollar amount such that the person would be indifferent between experiencing the benefit and possessing the money. In general, the dollar amount required to compensate a person for exposure to an adverse effect is roughly the same as the dollar amount a person is willing to pay to avoid the effect. Thus, economists speak of "willingness-to-pay" (WTP) as the appropriate measure of the value of avoiding an adverse

[1] The literature reviews and process for developing valuation estimates are described in detail in Appendix I and in referenced supporting reports.

effect. For example, the value of an avoided respiratory symptom would be a person's WTP to avoid that symptom.

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, a 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 products that result in similar effects or risk reductions, (e.g., non-toxic cleaners or bike helmets). Alternatively, surveys may be used in an attempt to directly elicit WTP for an environmental improvement.

Wherever possible in this analysis, we use estimates of mean WTP. In cases where WTP estimates are not available, we use the cost of treating or mitigating the effect as an alternative estimate. For example, for the valuation of hospital admissions we use the avoided medical costs as an estimate of the value of avoiding the health effects causing the admission. These costs of illness (COI) estimates generally understate the true value of avoiding a health effect. They tend to reflect the direct expenditures related to treatment and not the utility an individual derives from improved health status or avoided health effect. As noted above, we use a range of values for most environmental effects, to support the primary central estimate of net benefits. Table 6-1 summaries the mean unit value estimates that we use in this analysis. We present the full range of values in Appendix H, including those used to derive the primary low and primary high estimates, as well as values used to generate an alternative value for avoiding premature mortality.

### *Valuation of Premature Mortality*

Some forms of air pollution increase the probability that individuals will die prematurely. We use concentration-response functions for mortality that express the increase in mortality risk as cases of "ex-

**Table 6-1**
**Health Effects Unit Valuation (1990 dollars)**

| Endpoint | Pollutant | Valuation (mean est.) | |
|---|---|---|---|
| Mortality | $PM_{10}$ | $4,800,000 | per case |
| Chronic Bronchitis | $PM_{10}$ | $260,000 | per case |
| Chronic Asthma | $O_3$ | $25,000 | per case |
| Hospital Admissions | | | |
| All Respiratory | $SO_2$, $NO_2$, $PM_{10}$ & $O_3$ | $6,900 | per case |
| All Cardiovasular | $SO_2$, $NO_2$, & CO $PM_{10}$ & $O_3$ | $9,500 | per case |
| Emergency Room Visits for Asthma | $PM_{10}$ & $O_3$ | $194 | per case |
| Respiratory Illness and Symptoms | | | |
| Acute Bronchitis | $PM_{10}$ | $45 | per case |
| Asthma Attack or Moderate or Worse Asthma Day | $PM_{10}$ & $O_3$ | $32 | per case |
| Acute Respiratory Symptoms | $SO_2$, $NO_2$, $PM_1$, & $O_3$ | $18 | per case |
| Upper Respiratory Symptoms | $PM_1$ | $19 | per case |
| Lower Respiratory Symptoms | $PM_{10}$ | $12 | per case |
| Shortness of Breath, Chest Tightness, or Wheeze | $PM_{10}$ & $SO_2$ | $5.30 | per day |
| Work Loss Days | $PM_{10}$ | $83 | per day |
| Mild Restricted Activity Days | $PM_{10}$ & $O_3$ | $38 | per day |

cess premature mortality" per time period (e.g., per year). The benefit, however, is the avoidance of small increases in the risk of mortality. By summing individuals' WTP to avoid small increases in risk over enough individuals, we can infer the value of a statistical premature death avoided.[2] For expository purposes, we express this valuation as "dollars per mortality avoided," or "value of a statistical life" (VSL), even though the actual valuation is of small changes in mortality risk experience by a large number of people. The economic benefits associated with avoiding premature mortality were the largest category of monetized benefits in the section 812 CAA retrospective analysis (U.S. EPA 1997) and continue to be the largest source of monetized benefits for this prospective analysis. Mortality benefits, however, are also the largest contributor to the range of uncertainty in monetized benefits. For a more detailed discussion of the factors affecting the valuation of premature mortality see Appendix H.

The health science literature on air pollution indicates that several human characteristics affect the degree to which mortality risk affects an individual. For example, some age groups appear to be more susceptible to air pollution than others (e.g., the elderly and children). Health status prior to exposure also affects susceptibility. At risk individuals include those who have suffered strokes or are suffering from cardiovascular disease and angina (Rowlatt, et al. 1998). An ideal economic benefits estimate of mortality risk reduction would reflect these human characteristics, in addition to an individual's willingness to pay (WTP) to improve one's own chances of survival plus WTP to improve other individuals' survival rates.[3] The ideal measure would also take into account the specific nature of the risk reduction commodity that is provided to individuals, as well as the context in which risk is reduced. To measure this value, it is important to assess how reductions in air pollution reduce the risk of dying from the time that reductions take effect onward, and how individuals value these changes. Each individual's survival curve, or the probability of surviving beyond a given age, should shift as a result of an environmental quality improvement. For example, changing the current probability of survival for an individual also shifts future probabilities of that individual's survival. This probability shift will differ across individuals because survival curves are dependent on such characteristics as age, health state, and the current age to which the individual is likely to survive

Although a survival curve approach provides a theoretically preferred method for valuing the economic benefits of reduced risk of premature mortality associated with reducing air pollution, the approach requires a great deal of data to implement. The economic valuation literature does not yet include good estimates of the value of this risk reduction commodity. As a result, in this study we value avoided premature mortality risk using the value of statistical life approach, supplemented by an alternative valuation based on a value of statistical life years lost approach. We provide a review of the relevant literature and a more detailed discussion of our selected approach in Appendix H.

As in the retrospective, we use a mortality risk valuation estimate which is based on an analysis of 26 policy-relevant value-of-life studies (see Table 6-2). Five of the 26 studies are contingent valuation (CV) studies, which directly solicit WTP information from subjects; the rest are wage-risk studies, which base WTP estimates on estimates of the additional compensation demanded in the labor market for riskier jobs. We 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. We discuss this issue in detail later in this chapter and in Appendix H.

In addition, we developed alternative calculations based on a life-years lost approach. To employ the value of statistical life-year (VSLY) approach, we first estimated the age distribution of those lives projected to be saved by reducing air pollution. Based on life expectancy tables, we calculate the life-years saved

[2] 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.

[3] For a more detailed discussion of altruistic values related to the value of life, see Jones-Lee (1992).

**Table 6-2**
**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 |
| Garen (1988) | Labor Market | 13.5 |

Source: Viscusi, 1992 and EPA analysis.

from each statistical life saved within each age and gender cohort. To value these statistical life-years, we hypothesized a conceptual model which depicted the relationship between the value of life and the value of life-years. As noted in Chapter 5, the average number of life-years saved across all age groups for which data were available is 14 for PM-related mortality. The average for PM, in particular, differs from the 35-year expected remaining lifespan derived from existing wage-risk studies.[4] Using the same distribution of value of life estimates used above (i.e. the Weibull distribution with a mean estimate of $4.8 million), we estimated a distribution for the value of a life-year and combined it with the total number of estimated life-years lost. The details of these calculations are presented in Appendix H.

## *Valuation of Specific Health Effects*

### *Chronic Bronchitis*

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

We use the mean of a distribution of WTP estimates as the central tendency estimate of WTP to avoid a pollution-related case of chronic bronchitis in this analysis. The distribution incorporates 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, we derive a distribution of WTP to avoid a pollution-related case of CB by statistical uncertainty analysis techniques.[5] The expected value of this distribution,

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

[5] The statistical uncertainty analysis technique we used, which is also known as simulation modeling, is a probabilistic approach to characterizing the uncertainty or the distribution of potential values around a central estimate.

which is about $260,000, is taken as the central tendency estimate of WTP to avoid a pollution-related case of CB. We describe the three underlying distributions, and the generation of the resulting distribution of WTP, in Appendix H.

### Chronic Asthma

The valuation of this health endpoint requires an estimate which reflects an individual's desire to avoid the effects of chronic asthma throughout his or her lifetime. We derive this valuation estimate from two studies that solicit values from individuals diagnosed as asthmatics. Blumenschein and Johannesson (1998) generate an estimate of monthly WTP, while O'Conor and Blomquist (1997) generate an annual WTP estimate. In order to extend monthly and annual WTP estimates over an individual's lifetime, we adjusted the reported estimates to reflect the average life-years remaining and age distribution of the adult U.S. population, given that chronic asthma is not expected to affect the average life expectancy. The mean estimate of WTP to avoid a case of chronic asthma resulting from this method is approximately $25,000.

### Respiratory-Related Ailments

In general, the values we assign to the respiratory-related ailments in Table 6-1 are a combination of WTP estimates for individual symptoms comprising 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. While that study defines upper respiratory symptoms as one suite of ailments (runny or stuffy nose; wet cough; and burning, aching, or red eyes), the valuation literature reports individual WTP estimates for three closely matching symptoms (head/sinus congestion, cough, and eye irritation). We therefore use these available WTP estimates and a benefits transfer procedure to estimate the value of avoiding those symptoms defined in the concentration-response study.

To capture the uncertainty associated with the valuation of respiratory-related ailments, we 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 pollution-related symptom or combination of 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 report an estimate for WTP to avoid a day of minor restricted activity. Therefore, we derive for this analysis a value 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, a distributional form is used which reflects our expectations that the actual value is likely to be closer to the central estimate than either extreme.

### Hospital Admissions, Cardiovascular and Respiratory

The valuation of this benefits category reflects the value of reduced incidences of hospital admissions due to respiratory or cardiovascular conditions. We use avoided hospital admissions as a measure as opposed to the number of avoided cases of respiratory or cardiovascular conditions, because of the availability of C-R relationships for the hospital admissions endpoint. Hospital admissions reflect a class of health effects linked to air pollution which are acute in nature but more severe than the symptom-day measures discussed above.

As described in Chapter 5, our approach to estimating the number of incidences for this category involves reliance on several concentration-response (C-R) functions. Each concentration response func-

tion provides an alternative definition of either respiratory effects or cardiovascular effects, and may be based on different pollutants. For valuation of the incidences, the current literature provides well-developed and detailed cost estimates of hospitalization by health effect or illness. Using illness-specific estimates of avoided medical costs and avoided costs of lost work-time, developed by Elixhauser (1993), we construct cost of illness (COI) estimates that are specific to the suite of health effects defined by each C-R function. For example, we use twelve distinct C-R functions to quantify the expected change in respiratory admissions.[6] Consequently in this analysis, we develop twelve separate COI estimates, each reflecting the unique composition of health effects considered in the individual studies.

The use of COI estimates suggests we likely understand the WTP to avoid these effects. The valuation of any given health effect, such as hospitalization, should reflect the value of avoiding associated pain and suffering and lost leisure time, in addition to medical costs and lost work time. While the probability distributions in this analysis characterize a range of potential costs associated with hospitalization, they do not account for the omission of factors from the COI estimates such as pain and suffering. Consequently, the valuations for these endpoints most likely understate the true social values for avoiding hospital admissions due to respiratory or cardiovascular conditions.

### *Stratospheric Ozone Provisions*

We develop monetary estimates of the health benefits due to stratospheric ozone provisions based on estimated incidences presented in a series of existing regulatory support analyses. To ensure consistency with the valuation strategy of this analysis, however, we adjust certain parameters used in the existing regulatory analyses of Title VI provisions. Specifically, we re-evaluate the physical effects change projected in the RIAs using the discount rate and the value of statistical life adopted throughout the rest of our present study. The net effect of these changes is to reduce the estimates of benefits from those found in the regulatory source support documents. The most important change is the discount rate. Because the benefits of stratospheric ozone protection accrue over several hundred years, the discount rate chosen can have an especially large influence on the benefits estimate. The central estimate employed in this analysis is five percent; the rate used in the source documents is two percent.

The value of statistical life (VSL) estimate is also an important factor in the calculations, because the vast majority of benefits of stratospheric ozone protection result from avoided fatal skin cancer cases. To reflect the uncertainty of the VSL estimates, we employ the same statistical uncertainty aggregation approach used in the criteria pollutant analysis, using a Weibull distribution of VSL estimates as an input. Appendix G describes the details of these and other changes made to ensure consistency between our stratospheric ozone provision benefits analysis and our criteria pollutant analyses.

## Results of Benefits Valuation

We combine the number of reduced incidences of our health endpoints with our estimated values of avoiding the health effect to generate total annual monetized human health benefits in 2000 and 2010. We attribute to Titles I through V of the CAAA total annual human health benefits of $68 billion in 2000 and $110 billion in 2010. We summarize the Post-CAAA 2010 monetized benefit in Table 6-3. The table provides our central estimate, in addition to the 5th and 95th percentile estimates for each benefit category.

There are two aspects of our results that warrant discussion. The first is the valuation of premature mortality due to PM exposure. The second is our strategy to avoid double-counting when aggregating health benefits. As discussed in Chapter 5, premature mortality is attributed to PM exposure and our primary estimate reflects a lag between PM exposure and premature mortality. While this lag does not alter the number of estimated incidences, it does alter the monetization of benefits. Because we value the "event" rather than the present risk, in this analysis we assume that the value of avoided future premature mortality should be discounted. Therefore, the type of lag structure employed plays a direct role in the valuation of this endpoint.

[6] For more detailed discussion of the various health effects considered by each C-R function and methodology for estimating the number of avoided hospital admissions, see Appendix D.

**Table 6-3**
**Results of Human Health Benefits Valuation, 2010**

| | Monetary Benefits (in millions 1990$) | | |
|---|---|---|---|
| | 5th %ile | Mean | 95th %ile |
| **Mortality** | | | |
| Ages 30+ | $ 14,000 | $ 100,000 | $ 250,000 |
| **Chronic Illness** | | | |
| Chronic Bronchitis | $ 360 | $ 5,600 | $ 18,000 |
| Chronic Asthma | 40 | 180 | 300 |
| **Hospitalization** | | | |
| All Respiratory | $ 75 | $ 130 | $ 200 |
| Total Cardiovascular | 93 | 390 | 960 |
| Asthma-Related ER Visits | 0.1 | 1 | 3 |
| **Minor Illness** | | | |
| Acute Bronchitis | $ 0 | $ 2 | $ 5 |
| URS | 4 | 19 | 39 |
| LRS | 2 | 6 | 12 |
| Respiratory Illness | 1 | 6 | 15 |
| Mod/Worse Asthma[1] | 2 | 13 | 29 |
| Asthma Attacks[1] | 20 | 55 | 100 |
| Chest tightness, Shortness of Breath, or Wheeze | 0 | 0.6 | 3 |
| Shortness of Breath | 0 | 0.5 | 1.2 |
| Work Loss Days | 300 | 340 | 380 |
| MRAD/Any-of-19 | 680 | 1,200 | 1,800 |
| **Total Benefits in 2010[2]** | - | $ 110,000 | - |

Note:

[1] Moderate to worse asthma and asthma attacks are endpoints included in the definition of MRAD/Any-of-19 respiratory effects. Although valuation estimates are presented for these categories, the values are not included in total benefits to avoid the potential for double-counting.

[2] Summing 5th and 95th percentile values would yield a misleading estimate of the 5th and 95th percentile estimate of total health benefits. For example, the likelihood that the 5th percentile estimates for each endpoint would simultaneously be drawn during the statistical uncertainty analysis is much less than 5 percent. As a result, we present only the total mean.

The primary analysis reflects a five-year lag structure. Under this scenario, 50 percent of the estimated cases of avoided mortality occur within the first two years. The remaining 50 percent are then distributed across the next three years. Our valuation of avoided premature mortality applies a five percent discount rate to the lagged estimates over the periods 2000 to 2005 and 2010 to 2015. We discount over the period between the initial PM exposure change (either 2000 or 2010) and timing of the incidence.

Many of the monetized health benefit categories include overlapping health endpoints, creating the potential for double-counting. In an effort to avoid overstating the benefits, we do not aggregate all of the quantified health effects. For example, asthma attacks and moderate to worse asthma are considered components of the endpoint, "Any of 19 Respiratory Symptoms". Consequently, we present the results but do not include them in our reported total benefits figures. In other cases, there are endpoints included in our aggregation of benefit that appear to have overlapping health effects. For those benefit categories that describe similar health effects, it is important to keep in mind that estimated incidences are based on unique portions of the population.

## Valuation Uncertainties

We addressed many valuation uncertainties explicitly and quantitatively by expressing values as distributions (see Appendix H for a complete description of distributions employed), using a computerized statistical technique to apply the valuations to physical effects (see Chapters 5 and 8) with the mean of each valuation distribution providing the foundation for the primary central estimate of total net benefits. 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, we assume 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 cost of illness values, which probably underestimate WTP). There are, however, a limited number of health endpoints

for which a different valuation approach may yield results significantly different from out primary central benefit estimate. For example, using a value of statistical life year approach in lieu of the value of statistical life method for valuing avoided premature mortality yields a mean estimate for this benefit which is approximately 45 percent lower than our primary central estimate. For those few endpoints where reasonable alternative valuation paradigms yield significantly different results from our preferred approach, see our discussion in Chapter 8.

The potential for biases as introduced by benefits transfer methodology is applicable to all benefits categories and, as noted in Table 6-4, the direction of its bias is unknown. Because changes in mortality risk are the single most important component of aggregate benefits, mortality risk valuation is also the dominant component of the quantified uncertainty. This category accounts for over 90 percent of total annual estimates under the Post-CAAA scenario. The second largest benefits category, reduced risk of chronic bronchitis, valued at approximately $5.6 billion per year in 2010, accounts for roughly five percent of the total estimated benefits. 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.

### *Mortality Risk Benefits Transfer*

One issue that merits special attention is the uncertainties and possible biases related to the "benefits transfer" from the 26 valuation source studies to valuation of reductions in PM-related mortality rates. Given the limitations of the current literature, we address this source of uncertainty qualitatively in this section. Although each of the mortality risk valuation source studies (see Table 6-2) estimate the average WTP for a given reduction in mortality risk, the degree of reduction in risk being valued varies 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, but that the actual mortality risk reduction resulting from a given pollutant reduction is 1/10,000. If WTP for reductions in mortality risk is linear in risk reduction, then a WTP of $50 for a reduction of 1/100,000 implies a WTP of $500 for a risk reduction of 1/10,000 (which is 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. This assumption has been shown to be reasonable provided the change in the risk being valued is within the range of risks evaluated in the underlying studies (Rowlatt et al. 1998).

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

**Table 6-4**
**Valuation of CAAA Benefits: Potential Sources and Likely Direction of Bias**

| Benefits Category | Factor | Likely Direction of Bias in WTP Estimates Used in this Study |
|---|---|---|
| Premature Mortality | Age | Uncertain, perhaps overestimate |
| | Degree of Risk Aversion | Underestimate |
| | Income | Uncertain |
| | Voluntary vs. Involuntary | Underestimate |
| | Catastrophic vs. Protracted Death | Uncertain, perhaps underestimate |
| | Discounting over a latency period | Uncertain, perhaps underestimate |
| Chronic Bronchitis | Severity-level | Uncertain |
| | Elasticity of WTP with respect to severity | Uncertain |
| All other benefit endpoints | Benefits Transfer | Uncertain |

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.[7] The average age of subjects in wage-risk studies, in contrast, would be well under 65, and probably closer to 40 years of age.

The direction of bias resulting from the age difference is unclear. We could argue 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. This would imply that older populations are likely to select occupations that are relatively less risky than workers represented in wage-risk studies or 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, because 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). This implies that individuals with higher incomes and/or greater wealth should be willing to pay more to reduce risk, all else equal, than individuals with lower incomes or wealth. The comparison between the income, both actual and potential, 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. 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, or generally assumed to be, whereas air pollution-related risks are incurred involuntarily. There is some evidence[8] that people will pay more to reduce involuntarily incurred risks than risks incurred voluntarily. If this is the case, WTP estimates based on wage-risk studies may understate WTP to reduce involuntarily incurred air pollution-related mortality risks.

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

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

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

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.

Economic assessment of WTP for lagged mortality effects also introduces uncertainty. For lack of a more refined technique, our analysis relies on the simplifying assumption that lagged mortality risks can be valued at the time of the occurrence of death, rather than at the time of exposure. In subsequent development of the annual and present value estimates, we therefore discount the dollar benefits estimate as if the full benefit accrues only in the year of death. There are several reasons to believe that this approach underestimates willingness to pay. Most importantly, while death may occur after a lag period, morbidity effects may appear at any time prior to death, including immediately upon exposure. It is not clear that other dose-response assessments capture the full range of morbidity effects, direct and indirect, that might be associated with a latent fatal exposure. Other potentially important factors include the use of a financial discount rate, which may or may not accurately represent the rate at which individuals might discount delayed health benefits and the effect of knowledge of a fatal exposure on valuation of a delayed effect, in other words whether the valuation is affected by a prior diagnosis of a fatal condition.

We summarize 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 in Table 6-4; the overall effect of these multiple biases is addressed in Table 6-5. 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;[9] 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 6-4, add additional uncertainty regarding the transferability of WTP estimates from wage-risk studies to environmental policy and program assessments.

[9] Chestnut, 1995; IEc, 1992.

**Table 6-5**
**Key Uncertainties Associated with Valuation of Health Benefits**

| Potential Source of Error | Direction of Potential Bias for Net Benefits | Likely Significance Relative to Key Uncertainties on Net Benefits Estimate[1] |
|---|---|---|
| Benefits transfer for mortality risk valuation, including differences in age, income, degree of risk aversion, the nature of the risk, and treatment of latency between mortality risks presented by PM and the risks evaluated in the available economic studies. | Unable to determine based on currently available information | Potentially major. The mortality valuation step is clearly a critical element in the net benefits estimate, so any uncertainties can have a large effect. As discussed in the text, however, information on the combined effect of these known biases is relatively sparse, and it is therefore difficult to assess the overall effect of multiple biases that work in opposite directions. |
| Benefits transfer for chronic bronchitis, including adjustments made to better match the severity of the risks modeled in the available economic studies. | Unable to determine based on currently available information | Probably minor. Benefits of avoided chronic bronchitis account for about five percent of total benefits, limiting the effect on net benefits to a maximum of about seven percent. Steps taken in the study to adjust for severity using the best available empirical information likely limit the effect to much less than this maximum value. |
| Inability to value some quantifiable morbidity endpoints, such as impaired lung function. | Underestimate | Probably minor. Reductions in lung function are a well-established effect, based on clinical evaluations of the impact of air pollutants on human health, and the effect would be pervasive, affecting virtually every exposed individual. There is therefore a potential for a major impact on benefits estimates. The lack of a clear symptomatic presentation of the effect, however, could limit individual WTP to avoid lung function decrements. |

Note: [1] The classification of each potential source of error reflects the best judgement of the section 812 Project Team. The Project Team assigns a classification of "potentially major" if a plausible alternative assumption or approach could influence the overall monetary benefit estimate by approximately five percent or more; if an alternative assumption or approach is likely to change the total benefit estimate by less than five percent, the Project Team assigns a classification of "probably minor."

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# Ecological and Other Welfare Effects

Chapter 7

EPA's traditional focus in environmental benefits assessment has been on quantifying beneficial impacts of environmental regulation on human health. As we have learned more about the effects of anthropogenic stressors on ecological systems, however, pursuit of environmental programs targeted on reductions of damage to the environment have become more common. The CAAA Title IV provisions, collectively referred to as the Acid Rain Program, are a good example. These provisions are in place largely as the result of a major research effort to better understand and quantify the effects of sulfur and nitrogen oxides on natural systems susceptible to acid rain. Although the benefits of this program include improvements in human health, the initial impetus was protection of ecological resources.

We have designed this first section 812 prospective analysis to be responsive to the increased focus on the importance of ecological resources by devoting a great deal of effort to characterizing and, where possible, quantifying and monetizing the impacts of air pollutants on natural systems. This increased focus is also partly a result of the outcome of EPA's retrospective analysis, in which we identified an increased understanding of and focus on ecological effects as one of the important research directions for the first prospective and subsequent analyses. This chapter presents the results of these efforts.

This chapter consists of four sections. First, we provide an overview of our approach to estimating the effects of air pollution on ecological systems. Second, we provide a characterization of these effects in qualitative terms. The second section concludes with a summary of the process for selecting specific impacts which can be quantified and monetized using currently available methods. Third, we present the results of our quantitative and economic analyses. Finally, we discuss major uncertainties of the ecological and other welfare effects analyses.

## Overview of Approach

Our analysis of ecological effects involves three major steps:

- First, we identify and characterize ecological effects from air pollution.
- Second, we develop and implement selection criteria for more in-depth assessment of ecological impacts.
- Third, we perform quantitative and qualitative analyses to characterize a portion of the benefits of the 1990 CAAA provisions.

The first step involves taking a broad view of pollutants controlled under the CAAA and their documented effects on ecological systems, both as individual pollutants and, to the extent possible, as one component in multiple-stressor effects on ecosystems and their components. We organize our analysis in terms of major pollutant classes and by the level of biological organization at which impacts are measured (e.g., regional ecosystem, local ecosystem, community, population, individual, etc.).

After completing the first step on a broad level, the second step involves narrowing the scope of subsequent analyses. While it is desirable to focus effort on those impacts that are of greatest importance, in practice the state of the science in ecological assessment largely dictates the subsequent focus of the analysis. There exist only a handful of comprehensive ecological assessments from which to draw conclusions about those effects that are most important either ecologically or in economic terms, and those studies are potentially controversial in their methods and conclusions, in part because of the incomplete understanding of many of these effects. As a result, the categories of effects ultimately chosen for assessment here are necessarily limited by available

methods and data. As scientific understanding and impact assessment methods grow more comprehensive, however, we expect that the focus of subsequent analyses will be on those effects whose avoidance would have the greatest potential ecological and/or economic value.

The third step involves implementing a wide range of analyses to more exhaustively characterize specific effects of air pollution on ecological systems. We provide quantitative estimates of the benefits of the 1990 CAAA for the following effects:

- eutrophication of estuaries associated with airborne nitrogen deposition;
- acidification of freshwater bodies associated with airborne nitrogen and sulfur deposition; and
- reduced forest growth associated with ozone exposure.

In addition, in this chapter we present the methods and results for quantitative analysis of other welfare effects, including reduced agricultural yields associated with ozone exposure, the impact of ambient particulate matter on visibility, the effects of ozone on farm worker productivity, and the effects of stratospheric ozone on crop and fisheries yields. These effects have been identified as important categories of benefits in many previous analyses, including the section 812 retrospective analysis. As a result, these effects were not considered in the same three step process used for other service flows.

We attempted to conduct quantitative analyses of two other benefits categories: the accumulation of toxics in freshwater fisheries associated with airborne toxics deposition; and aesthetic degradation of forests associated with ozone and airborne toxics exposure. However, we found that, while some quantitative methods exist to evaluate these benefits, key links are missing in the analytic process. This in turn prevents development of defensible benefits estimates which can be reasonably associated with the air quality and air pollutant deposition patterns developed from our Post-CAAA and Pre-CAAA scenarios. See Appendix E for more detailed discussion of these service flows. In addition, in assessing nitrogen deposition impacts to estuarine systems, we relied on a displaced cost approach with results that we chose to omit from the primary benefits estimate because of uncertainties in the methodology. These results are nonetheless reported in this chapter, but are used for the purposes of sensitivity testing only.

Because the breadth and complexity of air pollutant-ecosystem interactions do not allow for comprehensive quantitative analysis of all the ecological benefits of the CAAA, we stress the importance of continued consideration of those impacts not valued in this report in policy decision-making and in further technical research. Judging from the geographic breadth and magnitude of the relatively modest subset of impacts that we find sufficiently well-understood to quantify and monetize, it is apparent that the economic benefits of the CAAA's reduction of air pollution impacts on ecosystems are substantial.

## Characterization of Impacts of Air Pollution on Ecological Systems

The purpose of this section is to provide an overview of potential interactions between air pollutants and the natural environment. We identify major single pollutant-environment interactions, as well as the synergistic impacts of ecosystem exposure to multiple air pollutants. Although a wide variety of complex air pollution-environment interactions are described or hypothesized in the literature, for the purposes of this analysis we focus on major aspects of ecosystem-pollutant interactions. We do this by limiting our review according to the following criteria:

- Pollutants regulated by the CAAA.
- Known interactions between pollutants and natural systems as documented in peer-reviewed literature.
- Pollutants present in the atmosphere in sufficient amounts after 1990 to cause significant damages to natural systems.

Our understanding of air pollution effects on ecosystems has progressed considerably during recent decades. Previously, air pollution was regarded primarily as a local phenomenon and concern was associated with the vicinity of industrial facilities,

power plants or urban areas. The pollutants of concern were gaseous (e.g., sulfur dioxide and ozone) or heavy metals (e.g., lead) and the observed effects were visible stress- specific symptoms of injury (e.g., foliar chlorosis). The most typical approach to documenting the effects of specific pollutants was a dose-response experiment, where the objective was to develop a regression equation describing the relationship between exposure and some easily measured effect (e.g., growth, yield or mortality). As analytic methods improved and ecology progressed, a broader range of effects of air pollutants was identified and understanding of the mechanisms of effect improved. Observations made on various temporal scales (e.g., long-term studies) and spatial scales (e.g., watershed studies) led to the recognition that air pollution can affect all organizational levels of biological systems.

Our current understanding of ecosystem impacts can be organized by the pollutants of concern and by the level of biological organization at which impacts are directly measured. We attempt to address both dimensions of categorization in this overview. In Table 7-1 we summarize the major pollutants of concern, and the documented acute and long-term ecological impacts associated with them.

The summary in Table 7-1 is a highly condensed version of the results of our characterization of ecological impacts. In addition to the pollutant-specific effects outlined in the table, it is important to identify the level of biological organization and types of ecosystems that are susceptible to these types of effects. Tables 7-2 through 7-4 provide more detail on pollutant-specific impacts at a range of levels of biological organization. It is important to note that the interactions listed are intended to illustrate the range of possible adverse effects. For a more complete review of air-pollutant-induced effects on ecosystems, see Appendix E.

## *Effects of Mercury and Ozone*

Table 7-2 summarizes the effects of mercury and ozone on ecological systems. To illustrate the nature of our review of effects, consider the second row in Table 7-2. This row summarizes the effects of the air pollutants mercury and ozone at the "individual" level of biological organization. As indicated in the table, in a general sense air pollutants can induce a direct physiological response in individuals (analogous to that experienced by humans exposed to pollutants), or an indirect effect either through impacts on the individual's surroundings or by weakening the individual and making it more susceptible to other stressors. Mercury has several direct effects to fauna, including effects to the central nervous system and the liver, while the documented direct effects of ozone tend to be to a variety of plant functions. Indirect effects of mercury are not well understood, but the indirect effects of ozone may serve to compound the direct effects to plants by also making the plants more susceptible to drought or heat stress, for example. This type of cataloging of

**Table 7-1**
**Classes of Pollutants and Ecological Effects**

| Pollutant Class | Major Pollutants and Precursor Emissions | Acute Effects | Long-term Effects |
|---|---|---|---|
| Acidic Deposition | Sulfuric acid, nitric acid<br>Precursor emissions: Sulfur dioxide, nitrogen oxides | Direct toxic effects to plant leaves and aquatic organisms. | Progressive deterioration of soil quality. Chronic acidification of surface waters. |
| Nitrogen Deposition | Nitrogen compounds (e.g., nitrogen oxides) | | Saturation of terrestrial ecosystems with nitrogen. Progressive nitrogen enrichment of coastal estuaries. |
| Hazardous Air Pollutants (HAPs) | Mercury, dioxins | Direct toxic effects to animals. | Conservation of mercury and dioxins in biogeochemical cycles and accumulation in the food chain. |
| Ozone | Tropospheric ozone<br>Precursor emissions: Nitrogen Oxides and Volatile Organic Compounds (VOCs) | Direct toxic effects to plant leaves. | Alterations of ecosystem wide patterns of energy flow and nutrient cycling. |

**Table 7-2**
**Interactions of Mercury and Ozone with Natural Systems At Various Levels of Organization**

| | | Examples of Interactions | |
|---|---|---|---|
| **Spatial Scale** | **Type of Interaction** | **Mercury in streams and lakes** | **Ozone** |
| Molecular and cellular | Chemical and biochemical processes | Mercury enters the body of vertebrates and binds to sulfhydril groups (i.e. proteins). | Oxidation of enzymes of plants. Disruption of the membrane potential. |
| Individual | Direct physiological response. | Neurological effects in vertebrates. Behavioral abnormalities. Damages to the liver. | Direct injuries include visible foliar damage, premature needle senescence, reduced photosynthesis, altered carbon allocation, and reduction of growth rates and reproductive success. |
| | Indirect effects: Response to altered environmental factors or alterations of the individual's ability to cope with other kinds of stress. | Few interactions known. Damages through increased sensitivity to other environmental stress factors could occur, for example, through impairment of immune response. | Increased sensitivity to biotic and abiotic stress factors like pathogens and frost. Disruption of plant-symbiont relationship (mychorrhiza), and symbionts. |
| Population | Change of population characteristics like productivity or mortality rates. | Reduced reproductive success of fish and bird species. Increased mortality rates, especially in earlier life stages. | Reduced biological productivity. Selection for less sensitive individuals. Possibly microevolution for ozone resistance. |
| Community | Changes of community structure and competitive patterns | Loss of species diversity of benthic invertebrates. | Alteration of competitive patterns. Selective advantage for ozone-resistant species. Loss of ozone sensitive species and individuals. Reduction in productivity. |
| Local Ecosystem (e.g.,landscape element) | Changes in nutrient cycle, hydrological cycle, and energy flow of lakes, wetlands, forests, grasslands, etc. | Not well understood. | Alterations of ecosystem-wide patterns of energy flow and nutrient cycling. |
| Regional Ecosystem (e.g., watershed) | Biogeochemical cycles within a watershed. Region-wide alterations of biodiversity. | Not well understood. | Region-wide loss of sensitive species. |

effects, while limited in its direct usefulness in a cost-benefit framework, nonetheless does convey the wide range of documented effects of air pollutants on ecological resources. These tables and the accompanying text, found in Appendix E, also provide a framework for determining the extent to which important factors may not be well characterized by quantitative analysis, setting the stage for prioritization of research needs.

## *Effects of Nitrogen Deposition*

Table 7-3 provides a summary of the effects of nitrogen deposition on natural systems. These impacts are manifest in both terrestial and coastal estuarine systems. In both types of systems, nitrogen can be a growth-enhancing nutrient. As shown in the rows characterizing individual and population level impacts, the effects on many varieties of plants are beneficial. This growth can have other harmful effects, however. For example, excessive growth of

**Table 7-3**
**Interactions Between Nitrogen Deposition and Natural Systems At Various Levels of Organization**

| | | Examples of Interactions | |
|---|---|---|---|
| **Spatial Scale** | **Type of Interaction** | **Eutrophication and Nitrogen Saturation of Terrestrial Landscapes** | **Eutrophication of Coastal Estuaries** |
| Molecular and cellular | Chemical and biochemical processes | Assimilation of nitrogen by plants and microorganisms | Assimilation of nitrogen by plants and microorganisms. |
| Individual | Direct physiological response. | Increases in leaf- size of terrestrial plants. | Increase in growth of marine plants. |
| | Indirect effects: Response to altered environmental factors or alterations of the individual's ability to cope with other kinds of stress. | Decreased resistance to biotic and abiotic stress factors like pathogens and frost. Disruption of plant-symbiont relationships with mycorrhiza fungi. | Injuries to marine fauna through oxygen depletion of the environment. Loss of physical habitat due to loss of sea-grass beds. Injury through increased shading. Toxic blooms of plankton. |
| Population | Change of population characteristics like productivity or mortality rates. | Increase in biological productivity and growth rates of some species. | Increase in biological productivity. Increase of growth rates (esp. of algae and marine plants). |
| Community | Changes of community structure and competitive patterns | Alteration of competitive patterns. Selective advantage for fast growing species and individuals that efficiently use additional nitrogen. Loss of species adapted to nitrogen-poor environments. | Excessive algal growth. Changes in species composition. Decrease in sea-grass beds. |
| Local Ecosystem (e.g., landscape element) | Changes in nutrient cycle, hydrological cycle, and energy flow of lakes, wetlands, forests, grasslands, etc. | Magnification of the biogeochemical nitrogen cycle. Progressive saturation of microorganisms, soils, and plants with nitrogen. | Magnification of the nitrogen cycle. Depletion of oxygen, increased shading through algal growth. |
| Regional Ecosystem (e.g., watershed) | Biogeochemical cycles within a watershed. Region-wide alterations of biodiversity. | Leaching of nitrogen from terrestrial sites to streams and lakes. Acidification of aquatic bodies. Eutrophication of estuaries. | Additional input of nitrogen from nitrogen-saturated terrestrial sites within the watershed. |

marine organisms can lead to eutrophy, a state where the enhanced surface growth of plants shields bottom growing plants from sunlight, causing them to die and, in extreme cases, lead to low dissolved oxygen, or anoxic, conditions that impair a wide range of species and ecological functions. These effects are described in the table in the rows characterizing effects at the community and ecosystem levels. For this reason, isolated analysis of the effects of nitrogen on individuals or populations may provide misleading results; by the same token, analyses which ignore the beneficial effects of nitrogen in certain types of systems may lead to similarly misleading results. These complex linkages across biological levels of organization suggest that, when feasible, a systems level approach to ecological assessments is preferable to isolated analyses of effects at lower orders of organization.

## *Effects of Acid Deposition*

Table 7-4 provides a summary of the effects of acid deposition on forest and freshwater systems. The direct effects of acid deposition in lakes and streams include effects on fish species, as charaterized in the row describing individual-level effects. These

**Table 7-4**
**Interactions Between Acid Deposition and Natural Systems At Various Levels of Organization**

| | | Examples of Interactions | |
|---|---|---|---|
| **Spatial Scale** | **Type of Interaction** | **Acidification of Forests** | **Acidification of Streams and Lakes** |
| Molecular and cellular | Chemical and biochemical processes | Damages to epidermal layers and cells of plants through deposition of acids. | Impairment of ion interactions of fish at the cellular level. |
| Individual | Direct physiological response | Increased loss of nutrients via foliar leaching. | Decreases in pH and increase in aluminum ions causes pathological changes in gill structure of fish. |
| | Indirect effects: Response to altered environmental factors or alterations of the individual's ability to cope with other kinds of stress. | Cation depletion in the soil causes nutrient deficiencies in plants. Concentrations of aluminum ions in soils can reach phytotoxic levels. Increased sensitivity to other stress factors like pathogens and frost. | Aluminum ions in the water column can be toxic to many aquatic organisms through impairment of gill regulation. Acidification can indirectly affect submerged plant species, because it reduces the availability of dissolved carbon dioxide ($CO_2$). |
| Population | Change of population characteristics like productivity or mortality rates. | Decrease of biological productivity of sensitive organisms. Selection for less sensitive individuals. Microevolution of resistance. | Decrease of biological productivity of sensitive organisms. Selection for less sensitive individuals. Microevolution of resistance. |
| Community | Changes of community structure and competitive patterns | Alteration of competitive patterns. Selective advantage for acid-resistant species. Loss of acid sensitive species and individuals. Decrease in productivity. Decrease of species richness and diversity. | Alteration of competitive patterns. Selective advantage for acid-resistant species. Loss of acid sensitive species and individuals. Decrease in productivity. Decrease of species richness and diversity. |
| Local Ecosystem (e.g., landscape element) | Changes in nutrient cycle, hydrological cycle, and energy flow of lakes, wetlands, forests, grasslands, etc. | Progressive depletion of nutrient cations in the soil. Increase in the concentration of mobile aluminum ions in the soil. | Measurable declines of decomposition of some forms of organic matter, potentially resulting in decreased rates of nutrient cycling. |
| Regional Ecosystem (e.g., watershed) | Biogeochemical cycles within a watershed. Region-wide alterations of biodiversity. | Leaching of sulfate, nitrate, aluminum, and calcium to streams and lakes. Acidification of aquatic bodies. | Additional acidification of aquatic systems through processes in terrestrial sites within the watershed. |

effects are not as straightforward as they might appear, however, because it is not only the acidity (pH) of the water itself that causes the effect but the increased leaching of metals, particularly aluminum, which takes place in acidic (low pH) environments that contributes substantially to the effects on fish. These effects will vary widely from place to place according to the mineral content of the soil near the lake and the lakebed sediment, as well as the natural resistance of the lake in absorbing acid deposition (i.e., its buffering capacity). Other important effects characterized in the table include the ability of acid deposition to deplete cation concentrations in terrestrial ecosystems; increase the concentration of aluminum in soils; and leach nutrients, sulfates, and metals to surrounding streams and lakes. Effects of note at the individual level include foliar damage to trees.

A few general points emerge from our review of ecological effects:

- Air pollutants have indirect effects that are at least as important as direct toxic effects on living organisms. Indirect effects include those in which the pollutant alters the physical or chemical environment (e.g., soil properties), the plant's ability to compete for limited resources (e.g., water, light), or the plant's ability to withstand pests or pathogens. Examples are excessive availability of nitrogen, depletion of nutrient cations in the soil by acid deposition, mobilization of toxic elements such as aluminum, and changes in winter hardiness. As is true for other complex interactions, indirect effects are more difficult to observe than direct toxic relationships between air pollutants and biota, and there may be a variety of interactions that have not yet been detected.

- There is a group of pollutants that tend to be conserved in the landscape after they have been deposited to ecosystems. These conserved pollutants are transformed through biotic and abiotic processes within ecosystems, and accumulate in biogeochemical cycles. These pollutants include, but are not limited to, hydrogen ions (H+), sulfur (S) and nitrogen (N) containing substances, and mercury (Hg). Chronic deposition of these pollutants can result in progressive increases in concentrations and cause injuries due to cumulative effects. Indirect, cumulative damages caused by chronic exposure (i.e., long-term, moderate concentrations) to these pollutants may increase in magnitude over time frames of decades or centuries with very subtle annual increments of change. Examples are N-saturation of terrestrial ecosystems, cation depletion of terrestrial ecosystems, acidification of streams and lakes, and accumulation of mercury in aquatic food webs.

- Damages to ecosystems are most likely caused by a combination of environmental stress factors. These include anthropogenic factors such as air pollution and other environmental stress factors such as low temperature, excess or limited water, and limited availability of nutrients. The specific combinations of factors differ among regions and ecosystems where declines have been observed. Accurately predicting the impacts of multiple stress factors is an extremely difficult task, but this is an area of very active research among ecologists.

- Pollutant-environment interactions are complicated by the fact that biotic and abiotic factors in ecosystems change dramatically over time. Besides oscillations on a daily basis, and changes in a seasonal rhythm, there are long-range successional developments over time periods of years, decades, or even centuries. These temporal variations occur in polluted and pristine ecosystems, and no single point in time or space can be defined as representative of the entire system.

## Selection of Service Flows Potentially Amenable to Economic Analysis

Based on this broad overview of effects, we identify a set of pollutant-environment interactions which are amenable to more detailed quantification and monetization. We evaluate the long list of effects and seek categories where a defensible link exists between changes in air pollution emissions and the quality or quantity of the ecological service flow, and where economic models are available to monetize these changes. The use of these criteria greatly constrains the range of impacts that can be treated quantitatively. While the previous section identifies many pollutant-ecosystem interactions, only a handful are understood and have been modeled to an extent sufficient to reliably quantify their impact.

The theoretical basis of economic benefits assessment is that ecosystems provide services to mankind, and that those services have economic value. The application of this theory requires the isolation of service flows that have market values or are otherwise amenable to available methods for determining value in the absence of formal markets. Available methods do not exist to comprehensively value all service flows for any particular ecosystem or aggregation of ecosystems. Generally, we are limited

to those service flows that are either sources of material inputs or associated with natural amenities that involve active recreation. Impacts to these service flows that can be valued tend to manifest themselves immediately and can be readily measured and assessed in terms of the established cause and effect relationships.

Based on the constraints of economic valuation methods and data, we select from the host of ecosystem impacts identified in the previous section a set of service flows as candidate endpoints for analysis. The list of service flows establishes the potential scope of economic analysis for ecological effects feasible in the context of the present study. Table 7-5 presents the service flow impacts that we quantitatively estimate in this analysis plus those effects that currently cannot be quantified for each of the four ecological pollutant categories discussed in Table 7-1.

From the list of effects in Table 7-5, we further limited the quantitative and qualitative analyses conducted to reflect the available model coverage. The results are summarized in Table 7-6. The relatively short list of effects in Tables 7-5 and 7-6 demonstrates that, of the great number of known impacts of air pollution, only a subset can be assessed quantitatively. Note that for one category of effects, nitrogen deposition impacts to estuarine systems, we relied on a displaced cost approach (described below)

**Table 7-5**
**Ecological Effects of Air Pollutants**

| Pollutant | Quantified Effects | Unquantified Effects |
|---|---|---|
| Acidic Deposition | Impacts to recreational freshwater fishing | Impacts to commercial forests (e.g., timber, non-timber forest products) |
| | | Impacts to commercial freshwater fishing |
| | | Watershed damages (water filtration flood control) |
| | | Impacts to recreation in terrestrial ecosystems (e.g. forest aesthetics, nature study) |
| | | Reduced existence value and option values for nonacidified ecosystems (e.g. biodiversity values) |
| Nitrogen Deposition | Additional costs of alternative or displaced nitrogen input controls for eastern estuaries | Impacts to commercial fishing, agriculture, and forests |
| | | Watershed damages (water filteration, flood control) |
| | | Impacts to recreation in estuarine ecosystems (e.g. Recreational fishing, aesthetics, nature study) |
| | | Reduced existence value and option values for non-eutrophied ecosystems (e.g. biodiversity values) |
| Tropospheric Ozone Exposure | Reduced commercial timber yields and reduced tons of carbon sequestered | Impacts to recreation in terrestrial ecosystems (e.g. forest aesthetics, nature study) |
| | | Reduced existence value and option values for ozone-impacted ecosystems |
| Hazardous Air Pollutant (HAPS) Deposition | No service flows quantified | Impacts to commercial and recreational fishing from toxification of fisheries |
| | | Reduced existence value and option values for non-toxified ecosystems (e.g. biodiversity values) |

**Table 7-6**
**Summary of Endpoints Selected for Quantitative Analysis**

| Endpoint | Analysis | Geographic Scope |
|---|---|---|
| Lake acidification impacts on recreational fishing | Quantification of improved fishing with monetization of recreational value | Case study of New York State |
| Estuarine eutrophication impacts on recreational and commercial fishing | Quantification of improved fishing with monetization of displaced costs of alternative eutrophication control methods | Case studies of Chesapeake Bay, Long Island Sound, and Tampa Bay (with illustrative extensions to East Coast estuaries) |
| Ozone impacts on commercial timber sales | Quantification of improved timber growth with monetization of commercial timber revenues | National assessment |
| Ozone impacts on carbon sequestration in commercial timber | Quantification of improved carbon sequestration | National assessment |

that we chose to omit from the primary benefits estimate because of uncertainties in the methodology. These results are nonetheless reported in this chapter, but are used for the purposes of sensitivity testing only. In the next section we discuss the methods, results, and caveats of the analyses of these selected endpoints.

## Results

In this section we summarize the methods used for, and results obtained from, our quantitative and economic analyses of selected service flows. We first review the methods for each analysis, and then present a summary of key quantitative results. For a more detailed description of methods and results, see Appendix E.

### *Estuarine Eutrophication Associated with Airborne Nitrogen Deposition*

Atmospherically derived nitrogen makes up a sizable fraction of total nitrogen inputs in estuaries in the eastern United States. Airborne nitrogen deposition accounts for a significant fraction of the total nitrogen loads to coastal estuaries, particularly on the East and Gulf coasts. For example, the most recent estimates for the Chesapeake Bay indicate airborne deposition accounts for over 40 percent of the total nitrogen load to the estuary; in Galveston Bay, the share is almost 50 percent. When nitrogen enters estuaries it can cause eutrophication, or an increased nutrient load that, in excess, changes the ecosystem's structure and function and affects ecological service flows. Many state governments and multi-state regional authorities have expressed increasing concern about the control of airborne nitrogen deposition as an important source of nitrogen loading.

Our analysis of the effects of nitrogen deposition followed two tracks. We first attempted to quantify the service flows affected by and the damages associated with eutrophication, and derive dose-response relationships and valuation strategies for each of the key service flow categories (for example, recreational fishing). The derivation of dose-response relationships between atmospheric nitrogen loading and ecological effects, however, is complicated by the dynamic nature of ecological systems. In addition to being characterized by non-linear, "threshold" type responses, estuarine ecosystems are simultaneously influenced by a variety of stressors (both anthropogenic and natural). This makes it difficult to quantify the nature and magnitude of ecological changes expected to result from a change in a single stressor such as nutrient loading. Further, if the state of the ecosystem has changed (as from oligotrophic[1] to eutrophic) the removal of the initial stressor does not necessarily mean a rapid return to the prior state. This complicates the quantitative benefits assessment of controlling nitrogen deposition through the CAAA.

[1] Oligotrophy refers to a state of relatively low nutrient enrichment and low productivity of aquatic ecosystems. In contrast, eutrophy refers to a state of relatively high nutrient loading and higher productivity, sometimes leading to overenrichment and reduction in ecological service flows due to water quality decline.

Our second track relies on a displaced cost approach to benefit estimation. To reduce excess nutrient loads (including nitrogen) to local estuaries, many coastal communities are pursuing a range of abatement options. These options include wastewater and stormwater discharge point source controls as well as urban non-point and agricultural non-point source controls for runoff from the land. If atmospheric nitrogen depostion is reduced, the need for these types of expenditure to control other sources of nitrogen loading is also lessened, and the displaced control expenditures represent a benefit to society.

Displaced or avoided cost approaches are not always justified. In order to establish that the costs would truly be avoided, and to ensure that the avoidance of that cost represents a real benefit to society, we need to show that realistic and enforceable nitrogen reduction goals exist for each evaluated estuary. Without specific targets or reduction goals, it is not possible to suggest that there are specific control expenditures to be displaced. Therefore, we choose case study estuaries that most closely meet this criterion: Chesapeake Bay, Long Island Sound, and Tampa Bay. These areas have established nitrogen reduction programs that rely primarily on reductions of effluent from point sources as well as reductions in non-point source discharges. Information on the reduction goal and potential abatement options for meeting those goals allows us to estimate the portion of the goal that can be met by the CAAA, as well as the associated cost savings.[2]

The benefits valuation derived using the displaced-costs approach should be interpreted cautiously for two reasons. First, it is an estimation of capital costs that serve more purposes than mitigating nitrogen inputs into the estuaries of concern. Water treatment works are intended to provide waste water treatment for a variety of pollutants and may be required even in the absence of deposition of airborne nitrogen. Second, the nitrogen loading targets for the estuaries are not concrete, strictly enforced limits, based on certain knowledge of the capacity of the estuaries to accept nitrogen inputs. Instead, the targets may change over time as knowledge of the effects of nitrogen to these estuaries change. For these reasons, and because of the uncertainty about the ability of local and regional entities to enforce the nitrogen reduction targets, we calculate estimates of displaced costs for these three estuaries but do not include them in the primary benefits estimate for the CAAA.

Our approach involves three basic steps. First, we estimate the total loading of nitrogen to each of the three target estuaries. We use nitrogen deposition estimates from the RADM model, generated for each 80 km x 80 km grid cell in the eastern U.S. We then estimate the ultimate fate of deposited nitrogen through a GIS-based model of nitrogen "pass-through." The pass-through is the share of nitrogen deposited that is ultimately transported to the estuarine waters rather than retained by the land. Pass-through factors vary by land use, from about 20 percent (for forests and wetlands) to 100 percent (for open water). We estimate the nitrogen loading for each scenario, and the within-year, cross-scenario differences are the reduced nitrogen deposition attributed to the CAAA. We present these estimates in the second column of Table 7-7.

Second, we estimate the marginal costs of alternative abatement actions which could be implemented in the three case study estuaries. We develop our displaced-cost estimate by assuming that decision makers will choose to forego the most costly nitrogen abatement projects first. That is, we assume that reduced deposition and the resulting loadings reduction will eliminate the need for additional point or non-point source controls at the high end of the marginal cost curve. We summarize those results in the third and fourth columns of Table 7-7.

Third, we multiply the reduced nitrogen loading attributed to the CAAA by the marginal cost estimate to arrive at a range of estimates of displaced cost, ensuring that the reduction in airborne nitrogen is less than or equal to the potential tonnage reduction achieved by the displaced, high marginal cost abatement strategies. We present our results in the last column of Table 7-7. Our estimates suggest that the displaced cost is substantial for the large Chesapeake Bay and Long Island Sound estuaries, and more modest for Tampa Bay. The Chesapeake

[2] With increasing populations, controls of alternative sources (e.g., automobile and utility emissions) may be needed simply to meet the original target or goal. If the CAA amendments are necessary just to achieve the target reductions, then we are actually measuring alternative costs and not avoided costs.

**Table 7-7**
**Estimated Displaced Costs for Three Estuaries**

| Estuary | Reduced N Deposition in 2010(millions of pounds) | Low Marginal Cost($/lb/yr.) | High Marginal Cost ($/lb/yr.) | Estimated Annual Displaced Costs in 2010 ($millions) |
|---|---|---|---|---|
| Long Island Sound | 12.8 | $2 | $8 | $26-$100<br>Central Estimate: $63 |
| Chesapeake Bay | 58.1 | $6 | $22 | $350-$1,300<br>Central Estimate: $820 |
| Tampa Bay | 1.8 | $6 | $38 | $11 - $68<br>Central Estimate: $40 |

Bay and Long Island Sound watersheds together account for about 40 percent of the total estuarine watershed area on the East (Atlantic) coast that is sensitive to nitrogen deposition, while Tampa Bay accounts for about two percent of the sensitive watershed area for the Gulf coast.

## *Acidification of Freshwater Fisheries*

During the 1970s and 1980s, "acid rain" came to be known to the public as a phenomenon that injures trees, forests, and water bodies throughout Europe and in some areas of the United States and Canada. One of the goals of the CAAA was to address the problem of acidification of terrestrial and aquatic ecosystems caused by acidic deposition. To assess this effect we conducted a quantitative analysis of benefits derived from a reduction in acidification of aquatic bodies as they relate to recreational fishing in the Adirondacks region of New York State.

As discussed earlier in this chapter, acidification of water bodies is a complex process. Airborne acids, in the form of sulfur and nitrogen compounds, are deposited to water bodies and surrounding drainage areas, with the potential to change the pH of the water body. Many water bodies are relatively resistant and can absorb a great deal of deposition before pH changes substantially. This buffering capacity is referred to as acid neutralizing capacity (ANC). Once pH begins to be affected, a series of interactions occur, the most important of which is the leaching of aluminum from sediments and surrounding soil and the suspension of this metal in the water column. While acidic pH presents a direct stress to aquatic organisms, it is the combined effect of pH and aluminum exposure that presents the greatest risk. Lakes in the Adirondacks region of New York State are particularly susceptible to acidification because they have low baseline ANC, relative to water bodies in other areas of the country.

Because of these complex physical and chemical interactions, acidification stress is typically evaluated by application of a model that simulates these processes, and requires data on individual lake chemistry and sediment composition. We relied on the scenario-specific atmospheric deposition data (both sulfur and nitrogen) from the RADM air quality model (see Chapter 4 and Appendix C) as an input to EPA's Model of Acidification of Groundwater in Catchments (MAGIC). MAGIC generates several measures of the impact of sulfur and nitrogen deposition on lake acidity, including ANC and pH.[3] We used the pH outputs to classify lakes where recreational fishing might be impaired, and those estimates were used in an economic model of recreational fishing behavior in New York State to develop economic estimates of the impact of acid rain on recreational fishing resources in that state.

We summarize the results of our analysis of economic benefits of avoided Adirondacks acidification attributable to the CAAA in 2010 in Table 7-8. The range of annual benefits from the CAAA are $12 million to $49 million using the low-end assumptions on the threshold of effect (pH 5.0), and $82 to $88 million for the high-end assumptions on the effects threshold (pH 5.4). Higher pH (or, less acidic) threshold assumptions lead to greater damage estimates, because more lakes cross the less acidic threshold. We calculate our benefits results by comparing

[3] For more information on EPA's MAGIC model see Cosby et al. (1985a), as referenced in Appendix E.

**Table 7-8**
**Annual Economic Impact of Acidification in 2010 (Millions of 1990 Dollars)**

| | | Range of Economic Impact | | |
|---|---|---|---|---|
| **Year** | **Scenario** | **Low Estimate** | **Central Estimate** | **High Estimate** |
| 1990 | Base Year | $61 | | $320 |
| 2010 | Post-CAAA | $24 to $61 | | $261 to $281 |
| | Pre-CAAA | $73 | | $349 to $363 |
| **Range of CAAA Benefits in 2010** | | **$12 to $49** | **$50** | **$82 to $88** |

the suite of Post-CAAA 2010 estimates of total damages to the corresponding suite of estimates using Pre-CAAA deposition. The impact of nitrogen saturation in the surrounding terrestrial environment is reflected in the range of estimates presented in Table 7-8. If surrounding soils are saturated, less deposited nitrogen will remain on the land and more nitrogen will enter the water bodies, increasing the stress on the aquatic ecosystem. This phenomenon is reflected by the higher damage estimates for saturated versus non-saturated scenarios, other factors equal, although our model shows no effect of saturation in the 2010 Pre-CAAA low estimate. The results we present are in line with those generated from previous analyses that find annual benefits to the Adirondacks of halving utility emissions to be approximately in the millions to tens of millions of dollars.[4]

## *Reduced Timber Growth Associated with Ozone Exposure*

The third category of effects we quantify is improved commercial timber growth through the reduction of tropospheric ozone concentrations attributable to the CAAA. There is substantial scientific evidence to suggest that elevated ozone concentrations in the troposphere disrupt ecosystems by damaging and slowing the growth of vegetation. In this analysis, we examine one aspect of these impacts, reduced commercial timber growth. Much of the literature on the effects of ozone on tree growth is based on laboratory exposures of seedlings or leaf-scale experiments in the field. Estimates from those studies have been used in previous analyses, making use of professional judgment as an interpretive tool, but always with strong caveats about the potential applicability of the seedling and leaf-scale results to tree growth and, in particular, the rate of accumulation of wood mass that is important for commercial timber production.[5] In an attempt to overcome these issues, we sought to find a concentration-response relationship that would provide a more defensible and broadly applicable basis for estimating effects on tree growth.

Our analysis makes use of the Net Photosynthesis and Evapo-Transpiration model II (PnET II), a biological model of timber stand productivity to estimate the impacts of ozone on timber yields. The PnET II model was designed to estimate the combined effects of several stressors on the rate of net primary productivity (NPP), a measure of the rate of photosynthesis. NPP in a tree does not necessarily all go towards accumulation of wood mass; some may be allocated to root growth, leaf growth, or other tree functions. The PnET II model provides a means to measure both NPP and wood mass growth, as well as the effect on trees of several stressors combined. One important stressor to acknowledge in an analysis of the effects of ozone on trees is drought stress. Ozone has the effect of reducing water loss in trees by stimulating the closing of stomata through which water is transpired. As a result, in drought stress conditions, ozone can have beneficial effects on tree growth. The PnET II model reflects the impact of this factor in combination with other direct effects of ozone on tree function.

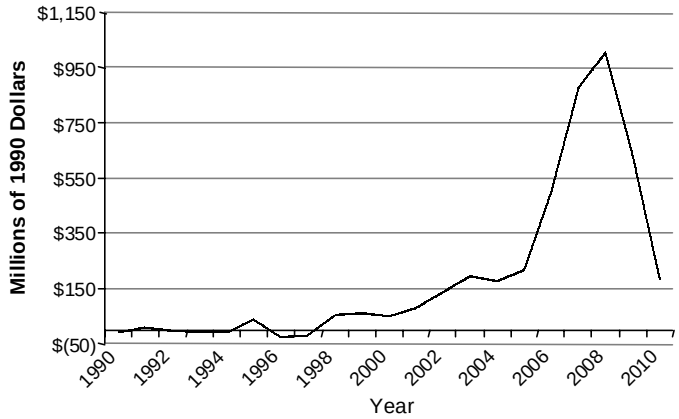
We used the PnET II model to provide estimates of timber stand responses to ozone exposure under each of the scenarios examined in this analysis. We aggregated tree growth results by region, with separate estimates for hardwoods and softwoods, and used them as inputs to the Timber Assessment Market

[4] For alternative estimates see, for example, Englin et al. (1991), Mullen and Menz (1985), and Morey and Shaw (1990), as referenced in Appendix E.

[5] See de Steiger et al. (1990) for an example of the generation of tree growth dose-response estimates based on professional judgement.

Model (TAMM), an economic model of the forest sector maintained by the United States Forest Service. There are three stages to the economic estimation. First, forest growth rate information generated by PnET II is provided to the Aggregate Timber Land Assessment System (ATLAS), the forest inventory tracking component of TAMM. Growth rate information is provided for each of the forest production regions defined by TAMM.[6] Second, ATLAS generates an estimate of forest inventories in each major region, which in turn serves as input to the market component of TAMM. Third, TAMM estimates the future harvests and market responses in each region.

**Figure 7-1**
**Annual Economic Welfare Benefit of Mitigating Ozone Impacts on Commercial Timber: Difference Between the Pre-CAAA and Post-CAAA Scenarios**




Our analysis suggests that there is a significant and measurable difference in timber harvests attributable to ozone exposure under the Post-CAAA and Pre-CAAA scenarios. At the outset of our modeling period, the early 1990s, virtually no change is measured in forest harvest volumes. This result occurs because increases in growth rates do not substantively affect timber volume over a short period of time. By the end of our modeling period, nearing 2010, increased growth rates over the previous decade(s) begin to affect overall forest yields of harvestable timber. This is observed in Figure 7-1 as an increasing annual benefit estimate over the modeling period. The shape of the benefits time-series reveals a production spike in the 2007 to 2008 period. This spike is due to a large anticipated harvest of Southeast U.S. timber due to forest maturity during this period. The spike would occur even in the absence of the CAAA, but is elevated by the CAAA due to increased growth rates projected under the Post-CAAA scenario. Although this change is small in percentage terms relative to total economic surplus generated by the timber sector, it contributes to a large portion of the commercial timber benefits estimate over the 1990-2010 period.

We calculate the cumulative value of annual benefits based on the discounted stream of the annual differences in consumer and producer surplus from commercial timber harvests under the Post-CAAA and Pre-CAAA ozone exposure scenarios from 1990 to 2010. Discounting annual benefits to 1990 using a five percent discount rate, the total cumulative benefits estimate is approximately $1.9 billion. These estimates are incorporated into the primary central estimate by developing a range of annual estimates for the year 2000, based on model results for the period 1998 to 2002, and the year 2010, based on model results for the period 2005 to 2010. The averaging of results across several years to generate our target year results avoids the potential problem of a particular year's results (such as for 2010) mischaracterizing the full time series of estimates when we later calculate the net present value of effects.

## *Reduced Carbon Sequestration Associated with Reduced Timber Growth*

Forest ecosystems help mitigate increasing atmospheric concentrations of carbon dioxide by sequestering carbon from the atmosphere. These ecosystems convert atmospheric carbon into biological structures (e.g., wood) or substances needed in the tree's physiological processes. As described above, however, ozone reduces the growth of forests, thereby limiting the amount of carbon that is sequestered. Sequestered carbon can help mitigate global climate change that has been linked to anthropogenic emissions of carbon and other greenhouse gases.

[6] TAMM includes Canadian as well as U.S. timber production regions because of the important influence of Canadian timber supply on the U.S. market. This analysis reflects modeling of Canadian timber regions and their impact on U.S. production, but we did not simulate changes in ozone in Canadian regions.

We used the timber inventory output of the TAMM/ATLAS modeling system (described above), in combination with a forest carbon model (FORCARB), to estimate changes in carbon storage in each of four ecosystem components: trees, forest understory, forest floor, and soil. The estimates from FORCARB, however, do not account for "leakages" of carbon back to the atmosphere as wood or wood products decay and decompose over time. To estimate the amount of carbon that is sequestered over the long-term, we used a second model, HARVCARB, to estimate the life-cycle of harvested forest timber and thereby adjust the forest carbon sequestration estimates of FORCARB.

The results of these calculations yield estimates of long-term increases in carbon storage as a result of the CAAA provisions of 8 million metric tons of carbon per year by the year 2000, and 29 million metric tons of carbon per year by the year 2010. Because of the great uncertainties in assessing the mitigating effect of carbon sequestration on global climate change, and the economic value of avoiding climate change, we do not attempt to monetize this category of benefit.

## Other Categories of Ecological Benefits

There were two additional categories of ecological effects for which we considered developing economic estimates; however, we abandoned the exercise when key portions of the analysis proved to be excessively problematic. Aesthetic degradation of forests, the first of these additional categories, was supported by a benefits transfer of contingent valuation studies of individual willingness to pay to avoid foliar damage. This category of effects, however, proved too difficult to link to the specific air quality scenarios we evaluated. In other words, available scientific methods and data on the visual appearance of forest stands and their impact on perceived forest aesthetics make it difficult to precisely describe changes in forest aesthetics. Evaluation of the second additional effect category, toxification of freshwater fisheries, was limited by the lack of toxic deposition and exposure data as well as by the limitations of available economic estimates of the impacts of toxics on recreational and commercial fishery resources. (See Appendix E for a more detailed discussion of these service flows). These and many other ecological benefit categories could not be quantified given current data and methods and are thus not reflected in our overall benefits estimates.

## Valuation of Other Effects

### Agricultural Benefits

As discussed earlier in this chapter, tropospheric ozone affects the growth of a wide range of plant species, including agricultural crops. Our agricultural benefits analysis relies on crop-yield loss C-R functions derived from the National Crop Loss Assessment Network (NCLAN) research and a national economic model of the agricultural sector (AGSIM). The NCLAN-derived relationships use a sum of hourly ozone concentration at or above 0.06 ppm (SUM06) as a measure of ozone exposure for the May to September ozone season; these exposure estimates are derived from the ozone air quality modeling results discussed in Chapter 4. Where the C-R functions require a longer time period of ozone concentrations, for example, for winter crops or when the growing or harvest season for summer crops extends beyond the end of September, we rely on 1990 monitor data to estimate ozone exposure, conservatively using the same estimates for both Pre-CAAA and Post-CAAA scenarios. The NCLAN functions cover the following crops: corn, cotton, peanuts, sorghum, soybeans, and winter wheat.

The AGSIM agricultural sector model takes the yield loss information, incorporates agricultural price, farm policy, and other data for each year, and then estimates production levels for each crop and the economic benefits to consumers and producers associated with these production levels. The crop coverage in the AGSIM model includes a wider range of crops than the NCLAN data inputs, adding barley, oats, hay, rice, and cottonseed. The broader crop coverage ensures that the model addresses price and production quantity effects on potential substitute crops that might be related to the effects in the six NCLAN crops. We estimate economic effects using a range of C-R outcomes for several crops, to reflect the variation in ozone sensitivity among the various crop cultivars. Our central estimate is the expected value of the range of results that emerge from the economic model.

Our results indicate significant beneficial effects of ozone reductions in the agricultural sector. Our Primary Central estimate of the benefit in 2000 is $450 million; the annual benefit rises to $550 million in 2010. Our estimated uncertainty around the Primary Central estimates, however, is very broad. For example, in 2010, the Primary Low estimate is $7.1 million, and the Primary High is $1,100 million. The uncertainty range reflects variation in the ozone response of crop cultivars and uncertainty about the suitability of alternative crop cultivars for the soil types and climate conditions in various agricultural regions. See Appendix F for more details on the methods and results of the C-R functions and economic modeling for agricultural effects.

## Visibility

As outlined in Chapter 4, air pollution impairs visibility in both residential and recreational settings. An individual's willingness to pay to avoid reductions in visibility differs in these two settings. Impairments in residential visibility are experienced throughout an individual's daily life and activities. Visibility in recreational settings, on the other hand, is experienced by visitors to areas with notable vistas. For the purposes of this report, we interpret recreational settings applicable for this category of effects to include National Parks throughout the nation. Other recreational settings may also be applicable, for example National Forests, state parks, or even hiking trails or roadside areas, but a lack of suitable economic valuation literature to identify these other areas, as well as a lack of visitation data in some cases, prevents us from generating estimates for those recreational vista areas.

We derive a residential visibility valuation function from the Chestnut and Dennis (1997) published estimates for the Eastern U.S. These estimates are based on original research conducted by McClelland et al. (1990) in two Eastern cities (Atlanta and Chicago). Because of technical concerns about the study's methodology, however, we calculate a benefits estimate but omit the results from the primary benefits estimates.[7] For recreational visibility, we derive values from the the Chestnut and Rowe (1989) study of WTP for visibility in three park regions in the Western, Southwestern, and Eastern U.S.[8] In both cases, the valuation function takes the following form:

$$HHWTP = B * \ln(VR1/VR2)$$

where:

HHWTP = annual WTP per household for visibility changes
VR1 = the starting annual average visual range
VR2 = the annual average visual range after the change in air quality
B = the estimated visibility coefficient.

The form of this valuation function is designed to reflect the way individuals perceive and express value for changes in visibility. In general terms, expressed WTP for visibility changes varies with the percentage change in visual range, a measure that is closely related to, though not exactly analogous to, the Deciview index used in Chapter 4. We use a central B coefficient for residential visibility of 141, as reported in Chestnut and Dennis (1997). For recreational visibility, the coefficients vary based on the region of study and whether the household is within or outside of the National Park region studied. In-region coefficients are higher than those for out-of-region households. The in-region estimates for California, the Southwest, and Southeast are $105, $137, and $65, respectively; the corresponding out-of-region estimates are $73, $110, and $40, respectively. The derivation and application of these valuation functions are described in more detail in Appendix H. The results of this procedure suggest visibility is an important category of CAAA benefits; the Primary Central estimate for 2010, for example, indicates annual recreational visibility benefits of $2.9 billion.

## Worker Productivity

We base the valuation of worker productivity on a study that measures the decline in worker pro-

[7] The two technical concerns involve the method of adjusting the contingent valuation survey results for non-response, and the failure to include adjustments for the "warm glow" effect, or the tendency of respondents to indicate higher willingness to pay for an environmental good because of a strong desire to improve the environment in general.

[8] The visibility valuation function, and the sources of estimates for the coefficients for the functions, were originally developed as part of the National Acid Precipitation Assessment Program (NAPAP), and were subjected to peer-review as part of that program.

ductivity among outdoor farm workers exposed to ozone (Crocker and Horst, 1981). In our analysis, we estimate the value of reduced productivity at $1 per 10 percent increase in ozone concentration. This estimate reflects valuing reduced productivity in terms of the reduction in percentage of daily income incurred by the average worker engaged in strenuous outdoor labor.

## *Stratospheric Ozone Provisions*

The quantified benefits of stratospheric ozone protection provisions are dominated by the reduced health effects expected from reductions in UV-b radiation; the derivation of health benefits of these provisions is discussed in Chapter 5. We summarize other categories of benefits associated with reduced UV-b radiation in Table 7-9. The quantified benefits include: reduced crop damage; and reduced polymer degradation. To estimate crop damage, we apply the results of existing studies on the relationship between crops and UV-b radiation to the changes in UV-b radiation predicted by the emissions and atmospheric models.[9] The polymer damage function is based on a study by Horst (1986). The estimated total cumulative benefits associated with these ecological and other welfare effects are about 2 percent of the total cumulative benefits of the Title VI provisions.

[9] Sources of dose-response relationship for crops and UV-b: Teramura and Murali (1986) and Rowe and Adams (1987). Source of dose-response relationship for crops and tropospheric ozone: Rowe and Adams (1987).

**Table 7-9**
**Quantified and Unquantified Ecological and Welfare Effects of Title VI Provisions**

| Ecological Effects- Quantified | Estimate | Basis for Estimate |
|---|---|---|
| American crop harvests | Avoided 7.5 percent decrease from UV-b radiation by 2075 | Dose-response sources: Teramura and Murali (1986), Rowe and Adams (1987) |
| American crop harvests | Avoided decrease from tropospheric ozone | Estimate of increase in tropospheric ozone: Whitten and Gery (1986). Dose-response source: Rowe and Adams (1987) |
| Polymers | Avoided damage to materials from UV-b radiation | Source of UV-b/stabilizer relationship: Horst (1986) |
| **Ecological Effects- Unquantified** | | |
| Ecological effects of UV. For example, benefits relating to the following:<br>• recreational fishing<br>• forests<br>• marine ecosystem and fish harvests<br>• avoided sea level rise, including avoided beach erosion, loss of coastal wetlands, salinity of estuaries and aquifers<br>• other crops<br>• other plant species<br>• fish harvests | | |
| Ecological benefits of reduced tropospheric ozone relating to the overall marine ecosystem, forests, man-made materials, crops, other plant species, and fish harvests | | |
| Benefits to people and the environment outside the U.S. | | |

Notes:
1) For more detail see EPA's *Regulatory Impact Analysis: Protection of Stratospheric Ozone* (1988).
2) Note that the ecological effects, unlike the health effects, do not reflect the accelerated reduction and phaseout schedule of section 606.
3) Benefits due to the section 606 methyl bromide phaseout are not included in the benefits total because the EPA provides neither annual incidence estimates nor a monetary value.

## Summary of Quantitative Results

Although the effects of air pollutants on ecological systems are likely to be widespread, many effects may be poorly understood and lack quantitative effects characterization methods and supporting data. In addition, many of our quantitative results reflect an incomplete geographic scope of analysis; for example, we generated monetized acidification results only for the Adirondacks region of New York State. As a result, the quantitative results we generate for the purposes of estimating the benefits of the CAAA reflect only a small portion of the overall impacts of air pollution on ecological systems or ecological service flows.

Despite these limitations, it is important to recognize the magnitude of the monetized ecological benefits that we could estimate and reflect those results in the overall estimates of benefits generated in the larger analysis. Table 7-10 provides a tabular summary of the results documented earlier in this chapter. It is not possible to indicate the degree to which ecological benefits are underestimated, but considering the magnitude of benefits estimated for the select endpoints considered in our analysis, it is reasonable to conclude that a comprehensive benefits assessment would yield substantially greater total benefits estimates.

In Table 7-11 we provide a summary of benefits estimates for other welfare effects, including reduced agricultural yields, impaired visibility, and decreased

**Table 7-10**
**Summary of Evaluated Ecological Benefits (millions 1990$)**

| Description of Effect | Air Pollutant | Geographic Scale of Economic Estimate | Range of Annual Impact Estimates in 2010 | Primary Central Estimate for 2010 | Primary Central Cumulative Impact Estimate 1990-2010 | Key Limitations |
|---|---|---|---|---|---|---|
| Freshwater acidification | Sulfur and nitrogen oxides | Regional (Adirondacks) | $12 to $88 | $50 | $260 | - Captures only recreational fishing impact<br>- Incomplete geographic coverage leads to underestimate of benefits |
| Reduced tree growth - Lost commercial timber | Ozone | National | $190 to $1000 | $600 | $1,900 | - Uncertainties in stand-level response to ozone exposure<br>- Uncertainty in future timber markets |
| | | **TOTAL MONETIZED ECONOMIC BENEFIT** | $200 to $1,100 | $650 | $2,200 | - Partial estimate that omits major unquantifiable benefits categories; see text |

Note: Estimates reflect only those benefits categories for which quantitative economic analysis was supported. A comprehensive total economic benefit estimate would likely greatly exceed the estimates in the table. Range of estimates for timber assessment is based on variation in annual point estimates for 2005 through 2010.

**Table 7-11**
**Summary of Other Welfare Benefits (millions 1990$)**

| Description of Effect | Air Pollutant | Geographic Scale of Economic Estimate | Primary Central Annual Estimate 2000 | Primary Central Annual Estimate 2010 | Primary Central Cumulative Estimate 1990-2010 | Key Limitations |
|---|---|---|---|---|---|---|
| Reduced Agricultural Yields | Ozone | National | $450 | $550 | $3,900 | - Covers only major grain crops<br>- Omits effects on fruits and vegetables |
| Impaired Recreational Visibility | Particulate Matter | National | $2,000 | $2,900 | $19,000 | - National Parks only<br>- Omits residential visibility benefits |
| Reduced Worker Productivity | Ozone | National | $460 | $710 | $4,400 | - Reflects effects on workers engaged in strenuous outdoor employment |

Note: Estimates reflect only those benefits categories for which quantitative economic analysis was supported.

worker productivity. These estimates add substantially to the total non-health benefits of the CAAA. In particular, our estimates for the annual value of avoiding visibility impairments is $2,900 million by 2010, even through this estimate does not reflect the value of residential visibility improvements.

## Uncertainty

Because of the limitations in the available methods and data, the benefits assessment in this report does not represent a comprehensive estimate of the economic benefits of the CAAA. Moreover, the potential magnitude of long-term economic impacts of ecological damages mitigated by the CAAA suggests that great care must be taken to consider those ecosystem impacts that are not quantified here. Significant future analytical work and basic ecological and economic research must be performed to build a sufficient base of knowledge and data to support an adequate assessment of ecological benefits. For the current analysis, this incomplete coverage of effects represents the greatest source of uncertainty in the ecological assessment. This and other key uncertainties are summarized in Table 7-12.

Because the chronic ecological effects of air pollutants may be poorly understood, difficult to observe, or difficult to discern from other influences on dynamic ecosystems, our analysis focuses on acute or readily observable impacts. Disruptions that may seem inconsequential in the short-term, however, can have hidden, long-term effects through a series of interrelationships that can be difficult or impossible to observe, quantify, and model. This factor suggests that many of our qualitative and quantitative results may underestimate the overall, long-term effects of pollutants on ecological systems and resources.

**Table 7-12**
**Key Uncertainties Associated with Ecological Effects Estimation**

| Potential Source of Error | Direction of Potential Bias for Net Benefits Estimate | Likely Significance Relative to Key Uncertainties in Net Benefit Estimate* |
|---|---|---|
| Incomplete coverage of ecological effects identified in existing literature, including the inability to adequately discern the role of air pollution in multiple stressor effects on ecosystems. | Underestimate | Potentially major. The extent of unquantified and unmonetized benefits is largely unknown, but the available evidence suggests the impact of air pollutants on ecological systems may be widespread and significant. At the same time, it is possible that a complete quantification of effects might yield economic valuation results that remain small in comparison to the total magnitude of health benefits. |
| Omission of the effects of nitrogen deposition as a nutrient with beneficial effects. | Overestimate | Probably minor. Although nitrogen does have beneficial effects as a nutrient in a wide range of ecological systems, nitrogen in excess also has significant and in some cases persistent detrimental effects that are also not adequately reflected in the analysis. |
| Incomplete assessment of long-term bioaccumulative and persistent effects of air pollutants. | Underestimate | Potentially major. Little is currently known about the longer-term effects associated with the accumulation of toxins in ecosystems, but what is known suggests the potential for major impacts. Future research into the potential for threshold effects is necessary to establish the ultimate significance of this factor. |
| The PnET II modeling of the effects of ozone on timber yields relies on a simplified mechanism of response (i.e., changes in net primary productivity). | Overestimate | Probably minor. Existing evidence suggests that the growth changes PnET II projects are relatively large, however none of the currently available points of conparison fully address such issues as the impact of stand-level competition, and the net primary productivity results are within the range of results of other studies of environmental and anthropogenic stressors. |

*The classification of each potential source of error reflects the best judgement of the section 812 Project Team. The Project Team assigns a classification of "potentially major" if a plausible alternative assumption or approach could influence the overall monetary benefit estimate by approximately five percent or more; if an alternative assumption or approach is likely to change the total benefit estimate by less than five percent, the Project Team assigns a classification of "probably minor."

# Comparison of Costs and Benefits

Chapter 8

In this chapter we present our summary of the primary estimates of monetized benefits of the CAAA from 1990 to 2010, compare the benefits estimates with the corresponding costs, and explore some of the major sources of uncertainty in the benefits estimates. We also present the results of our calculations using alternative assumptions for several key input variables.

## Monetized Benefits of the CAAA

In this section we provide an overview of the three types of analyses conducted to estimate benefits, present the annual estimates of monetized benefits for the human health, ecological, and welfare analyses, and then present an aggregate measure of benefits from all titles of the CAAA for the full study period.

### *Overview of Benefits Analyses*

Our primary estimates of the monetized economic benefits for the 1990 to 2010 period derive from three distinct analyses: (1) the analysis of changes in human health effects associated with reduced exposures to criteria pollutants and the subsequent valuation of these changes, summarized and described in Chapters 5 and 6; (2) the analysis of monetized ecological and other welfare benefits (e.g., visibility), described in Chapter 7; and (3) the analysis of the benefits of stratospheric ozone protection provisions, summarized briefly in Chapters 5, 6, and 7 and described in detail in Appendix G.

We measure the benefits and present the results from each of these analyses in slightly different ways. For the first two analyses, we generate annual estimates of benefits that result from changes in exposures in two target years of the study, 2000 and 2010. These estimates can be directly compared to the estimates of costs incurred in the target years, because for the most part the annual benefits accrue in the same year as the costs are incurred. There is one exception, however: we model the effect of particulate matter on premature mortality to occur over a period of five years from the time of exposure. In this case, we have accounted for the incidence of premature mortality over the assumed lag period, and discounted the valuation of this effect back to the target year.

The annual estimates provide an indication of the trend in benefits accrued over the 20-year study period. To generate a cumulative measure of benefits over the full 20-year period, we must make an assumption about the level of benefits that would be realized in the years between the target years. We interpolate these values, assuming a linear trend in both costs and benefits over the 1990 to 2000 and 2000 to 2010 periods (assuming benefits and costs in the starting year, 1990, are zero). In one portion of the ecological benefits analysis, acidification, we generate only a single annual estimate for the target year 2010. In that case, we assume a linear trend in annual benefits over the full 20-year study period.

The third analysis, assessing changes in stratospheric ozone and the resulting health effects, is different from the criteria pollutant analyses. The long-term nature of the program, and the significant lag effects associated with the processes of ozone depletion over decades-long time scales, make it difficult to generate a meaningful estimate for any single target year. As a result, we could not generate an annual benefit estimate that could be reliably linked to emissions reductions in a single year and, by extension, compared to the costs incurred to achieve that year's allocation of reductions in stratospheric ozone depleting substances. Instead, we generate an annualized equivalent of the cumulative present value of

benefits and costs of the Title VI program. These annualized equivalents cannot be ascribed to any particular target year.

These fundamental differences in the measurement of benefits affect our presentation of benefits estimates in this chapter. Although we generate and report an annual estimate of costs and benefits of Title VI provisions, we encourage the reader to interpret aggregations of these annual estimates with those from other titles of the CAAA with caution. In particular, we discourage the use of these CAAA Title-specific benefit-cost ratios as the sole, or even primary, basis for comparing the relative economic value of Title VI versus other CAAA titles. The comparative benefit-cost ratios are too sensitive to important, highly uncertain analytical assumptions such as the discount rate.

## Summary of Monetized Benefits for Human Health and Welfare Effects

As discussed above, we generate annual estimates for the human health and welfare effects based on exposure analysis conducted for each of the two target years of the analysis, 2000 and 2010. The range of estimates we generate for the monetized benefits of human health effects incorporates both the quantified uncertainty associated with each of the health effect estimates and the quantified uncertainty associated with the corresponding economic valuation strategy. Quantitative estimates of uncertainties in earlier steps of the analysis (i.e., emissions and air quality changes) could not be developed adequately and are therefore not applied in the present study. As a result, the range of estimates for monetized benefits presented in this chapter is more narrow than would be expected with a complete accounting of the uncertainties in all analytical components. The characterization of the uncertainty surrounding economic valuation is discussed in detail in Appendix H. The characterization of the uncertainty surrounding specific health effect estimates is discussed in Appendix D. Below, we discuss the combined effect of these two categories of uncertainty and our techniques for aggregating uncertainty across endpoints and analyses.

We assume that for each endpoint-pollutant combination there are distributions for both the concentration-response function and the valuation coefficients. We combine these distributions by using a computerized, statistical aggregation technique to estimate the mean of the monetized benefit estimate for each endpoint-pollutant combination and to characterize the uncertainty surrounding each estimate.[1]

In the first step of our procedure, we employ statistical analysis to generate mean estimates and quantified uncertainty measures for the C-R function for each endpoint-pollutant combination. For many health and welfare effects, only a single study is available to use as the basis for the C-R function. In this case, the best estimate of the mean of the distribution of C-R coefficients is the reported estimate in the study. The uncertainty surrounding the estimate of the mean C-R coefficient is characterized by the standard error of the reported estimate. This yields a normal distribution, centered at the reported estimate of the mean. If multiple studies are considered for a given C-R function, a normal distribution is derived for each study, centered at the mean estimate reported in the study. On each iteration of the aggregation procedure, a C-R coefficient is selected from an aggregate distribution of C-R estimates for that endpoint. The aggregate distribution of C-R coefficients is determined by a variance-weighted aggregate distribution of values.

In the second step, we estimate incidence for each exposure analysis unit (i.e., 8 km by 8 km cell in a grid pattern) in the 48 contiguous states, and aggregate the results into an estimate of the change in national incidence of the health or welfare effects. Through repeated iterations from the distribution of mean C-R coefficients, we generate a distribution of the estimated change in incidence for each health and welfare effect due to the change in air quality between the Post-CAAA and Pre-CAAA scenarios.

Finally, in the third step we use computerized statistical aggregation methods once again to charac-

[1] The statistical aggregation technique applied is commonly referred to as simulation modeling. The technique involves many re-calculations of results, using different combinations of input parameters each time. For each calculation, values from each input parameter's statistical distribution are selected at random to ensure that the calculation does not always result in extreme values, or rely solely on low end or solely on high end input parameters. The aggregate distribution more accurately reflects a reasonable likelihood of the joint occurrence of multiple input parameters.

terize the overall uncertainty surrounding monetized benefits. For each distinct health and welfare effect, the aggregation procedure selects an estimated incidence change from the distribution of changes for that endpoint, selects a unit value from the corresponding distribution of economic valuation unit values, and multiplies the two to generate a monetized benefit estimate. We then repeat the process many times to generate a distribution of estimated monetized benefits for each endpoint-pollutant combination. Combining the results for the individual endpoints using the aggregation procedure yields a distribution of total estimated monetized benefits for each target year (2000 and 2010).[2] We present the results of this analysis of health effects in Table 6-3 in Chapter 6.

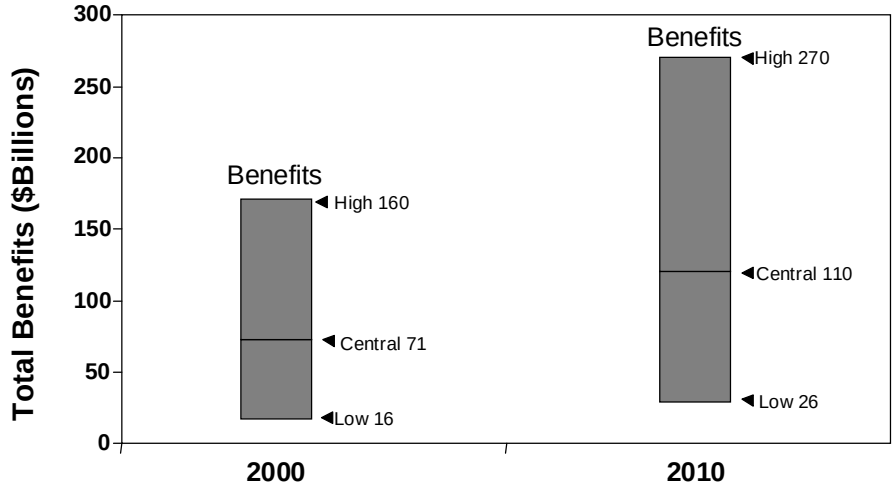
The ecological and welfare results are not currently amenable to the same type of uncertainty analysis. The modeling procedures for estimating the effects of sulfur and nitrogen deposition in acidifying lakes, the effects of ozone in reducing timber and agricultural production, and the effects of particular matter on visibility are all subject to uncertainty and require substantial resources simply to develop single estimates. We describe key uncertainties in Chapter 7 and they are reflected in the ranges of values we present at the end of that chapter. The sources of uncertainty in these estimates, however, cannot as easily be disaggregated among physical effects modeling and valuation components. The endpoints of the ranges we present reflect reasonable alternative choices in key input variables, but the ranges cannot currently be interpreted as points on a statistical distribution of results. For these ecological effects, the central estimate is the midpoint of the ranges of values. We then interpret the endpoints of the range of estimates as the upper and lower bounds of a uniform distribution of values. The uniform distribution is used when we aggregate the ecological and other welfare effects analyses with the analyses of human health.

## Annual Benefits Estimates

We present the results of our aggregation of primary annual benefits estimates for Titles I through V in Figure 8-1 below. The figure provides a characterization of both the primary central estimate and the range of values generated by the aggregation procedure described above, for each of the two target years of the analysis (2000 and 2010). The Primary High estimate corresponds to the 95th percentile value from the aggregation, and the Primary Low estimate corresponds to the 5th percentile value. The total benefits estimates are substantial; the Primary Central estimate in 2010 is $110 billion.

Table 8-1 shows the detailed breakdown of benefits estimates for one of the two target years, 2010. As shown in the table, $100 billion of the $110 billion total benefit estimate in 2010, or roughly 90 percent, is attributable to reductions in premature mortality associated with reductions in ambient particulate matter and associated criteria pollutants. The remaining benefits are divided among two broad categories of benefits: avoided morbidity, the largest component of which is avoided chronic bron-

**Figure 8-1**
**Central, Low, and High Primary Benefits Results for Target Years (in billions of 1990 dollars) - Titles I through V**




[2] This procedure implicitly assumes independence between the specific aggregation simulation draws from the distribution of health and economic valuation estimates.

**Table 8-1**
**Criteria Pollutant Health and Welfare Benefits in 2010**

| | Monetary Benefits (in millions 1990$)* | | |
|---|---|---|---|
| **Benefits Category** | **Primary Low** | **Primary Central** | **Primary High** |
| **Mortality** | | | |
| Ages 30+ | 14,000 | 100,000 | 250,000 |
| **Chronic Illness** | | | |
| Chronic Bronchitis | 360 | 5,600 | 18,000 |
| Chronic Asthma | 40 | 180 | 300 |
| **Hospitalization** | | | |
| All Respiratory | 76 | 130 | 200 |
| Total Cardiovascular | 93 | 390 | 960 |
| Asthma-Related ER Visits | 0.1 | 1.0 | 2.8 |
| Minor Illness | | | |
| Acute Bronchitis | 0.0 | 2.1 | 5.2 |
| URS | 4.2 | 19 | 39 |
| LRS | 2.2 | 6.2 | 12 |
| Respiratory Illness | 0.9 | 6.3 | 15 |
| Mod/Worse Asthma[1] | 1.9 | 13 | 29 |
| Asthma Attacks[1] | 20 | 55 | 100 |
| Chest Tightness, Shortness of Breath, or Wheeze | 0.0 | 0.6 | 3.1 |
| Shortness of Breath | 0.0 | 0.5 | 1.2 |
| Work Loss Days | 300 | 340 | 380 |
| MRAD/Any-of-19 | 680 | 1,200 | 1,800 |
| **Welfare** | | | |
| Decreased Worker Productivity | 710 | 710 | 710 |
| Visibility - Recreational | 2,500 | 2,900 | 3,300 |
| Agriculture (Net Surplus) | 7.1 | 550 | 1,100 |
| Acidification | 12 | 50 | 76 |
| Commercial Timber | 180 | 600 | 1,000 |
| Aggregate Range of Benefits[2] | 26,000 | 110,000 | 270,000 |

Note:
* The estimates reflect air quality results for the entire population in the US.
[1] Moderate to worse asthma, asthma attacks, and shortness of breath are endpoints included in the definition of MRAD/Any of 19 respiratory effects. Although valuation estimates are presented for these categories, the values are not included in total benefits to avoid the potential for double-counting.
[2] The Aggregate Range reflects the 5th, mean, and 95th percentile of the estimated credible range of monetary benefits based on quantified uncertainty, as discussed in the text.

**Table 8-2**
**Present Value of Monetized Benefits for 48 State Population**

| | Present Value (millions 1990$, discounted to 1990 at 5 percent) | | |
|---|---|---|---|
| | **Primary Low** | **Primary Central** | **Primary High** |
| Titles I through V (1990 through 2010) | $160,000 | $690,000 | $1,600,000 |
| Title VI (1990 through 2165) | $100,000 | $530,000 | $900,000 |

chitis, comprises about 60 percent of the non-mortality benefits; and avoided ecological and other welfare effects, the largest component of which is improved recreational visibility, comprises about 40 percent. Note that, because of the aggregation procedure used, and because we round all intermediate results to two significant digits for presentation purposes, the columns of Table 8-1 may not sum to the total estimate presented in the last row.[3]

## Aggregate Monetized Benefits

As discussed earlier in this chapter, we linearly interpolate benefit estimates between 1990 and 2000 and between 2000 and 2010 and then aggregate the resulting annual estimates across the entire 1990 to 2010 period of the study to yield a present discounted value of total aggregate benefits for the period. In this section we discuss issues involved in each stage of aggregation, as well as the results of the aggregation.

As noted earlier, air quality modeling was carried out only for the two target years (2000 and 2010). The resulting annual benefit estimates provide a temporal trend of monetized benefits across the period resulting from the annual changes in air quality. They do not, however, characterize the uncertainty associated with the yearly estimates for intervening years. In an attempt to capture uncertainty associated with these estimates, we relied on the ratios of the 5th percentile to the mean and the 95th percentile to the mean in the two 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 we use to characterize uncertainty about the Primary Central estimate.

In Table 8-2 we present the cumulative monetized benefits aggregated from 1990 to 2010. We present the mean estimate from the aggregation procedure, along with the Primary Low (i.e., 5th percentile of the distribution) and Primary High (i.e., 95th percentile of the distribution) estimates, for all provisions of Titles I through V and, then, separately for Title VI. Aggregating the stream of monetized benefits across years involved discounting the stream of monetized benefits estimated for each year to the 1990 present value (using a five percent discount rate).

## Aggregate Benefits of Title VI Provisions

As described in summary form in Chapters 5, 6, and 7 and in detail in Appendix G, expected human health benefits from Title VI provisions are substantial. The analysis we conducted is based largely on existing results from EPA Regulatory Impact Analyses for individual rules promulgated under Title VI. To the extent possible, we adjusted existing estimates to reflect both the central estimates and uncertainty characterizations used in the criteria pollutant analysis. We made major adjustments for both the value of statistical life (VSL) and the discount rate. We adjusted the VSL estimate to reflect the Weibull distribution of VSL used in our analysis for other provisions. As discussed in the appendix, the choice of the discount rate for estimated benefits which accrue over decades to century-long time spans presents special problems. Although we argue that a two percent discount rate is more appropriate for such long-term discounting, for consistency in this chapter we present estimates using the five percent discount rate used throughout the rest of this study.

[3] 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 8-1. For example, if the fifth percentile benefits of the endpoints shown in Table 8-1 were added, the resulting total would be substantially less than $30 billion, the fifth percentile value of the distribution of aggregate monetized benefits reported in Table 8-1. 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.

The results of the benefits calculations in Appendix G indicate a cumulative central benefit estimate of $530 billion for Title VI (see Appendix G for details). Using the same aggregation techniques for the valuation analysis described above, but only for the mortality valuation step, we generate a 90 percent confidence interval around this central estimate to derive Primary Low and Primary High estimates of $100 billion to $900 billion, respectively. We present these estimates in Table 8-2 above. The annual human health benefits from Title VI provisions steadily increase until about 2045, then decrease until 2165, the last year in the analysis. About 93 percent of the benefits accrue from 2015 to 2165. These benefit estimates only partially reflect potential averting behaviors, such as remaining indoors or increasing use of sun screens or hats, which may mitigate the effects of the UV-b exposure increases estimated under the Pre-CAAA scenario.

## Comparison of Monetized Benefits and Costs

Table 8-3 presents summary quantitative results for the prospective assessment, with costs disaggregated by Title and benefits disaggregated by major category. We present annual, primary estimate results for each of the two target years of the analysis, with all dollar figures expressed as inflation-adjusted 1990 dollars. The final columns provide net present value estimates for costs and benefits from 1990 to 2010 or, in the case of stratospheric ozone protection provisions, 1990 to 2165, discounted to 1990 at five percent. The results indicate that the Primary Central estimate of benefits clearly exceeds the costs of the CAAA, for each of the two target years and for the cumulative estimates of present value over the 1990 to 2010 period.

The estimates in Table 8-3 reflect the difficulty we encountered in reliably disaggregating benefits by CAAA Title or even by pollutant. As the table indicates, a very high percentage of the benefits is attributable to reduced premature mortality associated with reductions in ambient particulate matter and associated criteria pollutants. The CAAA achieves ambient PM reductions through a wide range of provisions controlling emissions of both gaseous precursors of PM that form particles in the atmosphere (sulfur and nitrogen oxides as well as, to a lesser extent, organic constituents) and directly emitted PM (i.e., dust particles). Because the effects of these constituents on ambient PM are nonlinear, and because some precursor pollutants interact with each other in ways which influence the total concentration of particulates in the atmosphere, separating the effects of individual pollutants on the change in ambient PM would require many iterations of our air quality modeling system. These difficulties in separating the effects of individual emissions reductions on the benefits estimates also highlight the need for an integrated air quality modeling system that can more readily analyze multiple scenarios within reasonable time and resource constraints. A tool of this nature could allow us to more reliably and cost-effectively estimate incremental contributions to ambient PM and ozone concentration reductions.

**Table 8-3**
**Summary of Quantified Primary Central Estimate Benefits and Costs (Estimates in million 1990$)**

| Cost or Benefit Category | Annual Estimates 2000 | Annual Estimates 2010 | Present Value |
|---|---|---|---|
| ***Costs:*** | | | |
| Title I | $8,600 | $14,500 | $85,000 |
| Title II | $7,400 | $9,000 | $65,000 |
| Title III | $780 | $840 | $6,600 |
| Title IV | $2,300 | $2,000 | $18,000 |
| Title V | $300 | $300 | $2,500 |
| **Total Costs, Title I-V** | $19,000 | $27,000 | $180,000 |
| **Title VI** | $1,400* | | $27,000* |
| ***Monetized Benefits:*** | | | |
| Avoided Mortality | $63,000 | $100,000 | $610,000 |
| Avoided Morbidity | $5,100 | $7,900 | $49,000 |
| Ecological and Welfare Effects | $3,000 | $4,800 | $29,000 |
| **Total Benefits, Title I-V** | $71,000 | $110,000 | $690,000 |
| **Stratospheric Ozone** | $25,000* | | $530,000* |

* Annual estimates for Title VI stratospheric ozone protection provisions are annualized equivalents of the net present value of costs over 1990 to 2075 (for costs) or 1990 to 2165 (for benefits). The difference in time scales for costs and benefits reflects the persistence of ozone depleting substances in the atmosphere, the slow processes of ozone formation and depletion, and the accumulation of physical effects in response to elevated UV-b radiation levels.

Table 8-4 provides the results of our comparison of primary benefits estimates to primary cost estimates. In the top half of the table we show both annual and present value estimates for Titles I through V, present value estimates for Title VI, and a total present value for all titles. The "monetized benefits" indicate both the Primary Central estimate (the mean) from our statistical aggregation modeling analysis and the Primary Low and Primary High estimates (5th and 95th percentile values, respectively). In the bottom half of the table we present two alternative methods for comparing benefits to costs. "Net benefits" are the Primary Central estimates of monetized benefits less the Primary Central estimates of costs. The table also notes the benefit/cost ratios implied by the benefit ranges.

The conclusion we draw from Table 8-4 is that, given the particular data, models and assumptions we believe are most appropriate at this time, our analysis indicates that the benefits of the CAAA substantially exceed its costs. Furthermore, the results of the uncertainty analysis imply that it is extremely unlikely that the monetized benefits of the CAAA over the 1990 to 2010 period could be less than its costs. Looking at Titles I through V, the central benefits estimate exceeds costs by a factor of four to one, whether we are looking at annual or present value measures, and the high estimate exceeds costs by more than twice that factor (a ratio of nine or ten to one). Using the Primary Low estimate of benefits, the annual estimates of benefits in 2000 and 2010 are slightly less than the annual costs for that year. The data also suggest that costs for criteria

**Table 8-4**
**Summary Comparison of Benefits and Costs (Estimates in millions 1990$)**

| | Titles I through V | | | Title VI | All Titles |
|---|---|---|---|---|---|
| | Annual Estimates | | Present Value Estimate | Present Value Estimate | |
| | 2000 | 2010 | 1990-2010 | 1990-2165 | Total Present Value |
| ***Monetized Direct Costs:*** | | | | | |
| Low [a] | | | *Not Estimated* | | |
| Central | $19,000 | $27,000 | $180,000 | $27,000 | $210,000 |
| High [a] | | | *Not Estimated* | | |
| ***Monetized Direct Benefits:*** | | | | | |
| Low[b] | $16,000 | $26,000 | $160,000 | $100,000 | $260,000 |
| Central | $71,000 | $110,000 | $690,000 | $530,000 | $1,200,000 |
| High[b] | $160,000 | $270,000 | $1,600,000 | $900,000 | $2,500,000 |
| ***Net Benefits:*** | | | | | |
| Low | ($3,000) | ($1,000) | ($20,000) | $73,000 | $50,000 |
| Central | $52,000 | $93,000 | $510,000 | $500,000 | $1,000,000 |
| High | $140,000 | $240,000 | $1,400,000 | $870,000 | $2,300,000 |
| ***Benefit/Cost Ratio:*** | | | | | |
| Low[c] | less than 1/1 | less than 1/1 | less than 1/1 | less than 4/1 | 1/1 |
| Central | 4/1 | 4/1 | 4/1 | 20/1 | 6/1 |
| High[c] | more than 8/1 | more than 10/1 | more than 9/1 | more than 33/1 | 12/1 |

[a] The cost estimates for this analysis are based on assumptions about future changes in factors such as consumption patterns, input costs, and technological innovation. We recognize that these assumptions introduce significant uncertainty into the cost results; however the degree of uncertainty or bias associated with many of the key factors cannot be reliably quantified. Thus, we are unable to present specific low and high cost estimates.

[b] Low and high benefits estimates are based on primary results and correspond to 5th and 95th percentile results from statistical uncertainty analysis, incorporating uncertainties in physical effects and valuation steps of benefits analysis. Other significant sources of uncertainty not reflected include the value of unquantified or unmonetized benefits that are not captured in the primary estimates and uncertainties in emissions and air quality modeling.

[c] The low benefit/cost ratio reflects the ratio of the low benefits estimate to the central costs estimate, while the high ratio reflects the ratio of the high benefits estimate to the central costs estimate. Because we were unable to reliably quantify the uncertainty in cost estimates, we present the low estimate as "less than X," and the high estimate as "more than Y", where X and Y are the low and high benefit/cost ratios, respectively.

pollutant programs grow somewhat more rapidly than benefits from 1990 to 2000, but that benefits grow more rapidly from 2000 to 2010.

The estimates for Title VI indicate that benefits well exceed costs, even at the low benefits estimate. This conclusion holds despite the relatively high discount rate used for the estimates in Table 8-4 (5 percent) a value that most analysts would consider too high for the long time period over which benefits of this program are discounted (175 years).[4] The total estimates for all titles of the CAAA also indicate benefits in excess of costs for the full range of primary benefits.

### Cost-Effectiveness Evaluation

The approach to premature mortality valuation used in our primary estimates is a method that allows us to aggregate the benefits of reducing mortality risks with other monetized benefits of the CAAA. One of the great advantages of the benefit-cost paradigm is that a wide range of quantifiable benefits can be compared to costs to evaluate the economic efficiency of particular actions. Some analysts suggest, however, that presentation of the results of a cost-benefit analysis may mask the key assumptions that are made to quantify all benefits in monetary terms. Another evaluative paradigm, cost-effectiveness analysis, is sometimes suggested as further evidence of whether the benefits of a regulatory program justify its costs. Cost-effectiveness analysis involves estimation of the costs per unit of benefit (e.g., lives saved). This type of analysis is most useful for comparing programs that have similar goals, for example, alternative medical interventions or treatments that can save a life or cure a disease. They are less readily applicable to programs with multiple categories of benefits, such as the CAAA, because the cost-effectiveness calculation is based on quantity of a single benefit category. In other words, we cannot readily convert reductions in new cases of chronic bronchitis, reduced hospital admissions, improvements in visibility, and increased commercial timber and crop yields to a single metric such as "lives saved." For these reasons, we prefer to present our results in terms of monetary benefits.

Despite the risks of oversimplification of benefits, cautiously interpreted cost-effectiveness calculations may provide further evidence of whether the costs incurred to implement the CAAA are a reasonable investment for the nation. The most common cost-effectiveness metric, costs per life saved, can be readily calculated from the information presented in this report. For example, we estimate the total annual direct costs of implementation of Titles I through V in 2010 to be approximately $27 billion. In exchange for this expenditure, in the year 2010 we avoid 23,000 cases of premature mortality and gain estimated non-mortality benefits of about $20 billion. We can generate a net cost per life saved by subtracting from costs the total non-mortality benefits, and then dividing by lives saved. For Titles I through V, we estimate a net cost per life saved of approximately $300,000 ($27 billion minus $20 billion divided by 23,000).[5] Although we are also concerned about many of the uncertain assumptions required to generate cost per life-year saved estimates, we include an estimate for illustrative purposes. For the year 2010, the net cost per life-year saved estimate implied by the primary central case results is $23,000 per life-year ($7 billion divided by 310,000 life-years saved).[6]

## Major Sources of Uncertainty

We can obtain additional insights into key assumptions and findings of the present study through further analysis of potentially important variables and inputs. The estimated uncertainty ranges for each endpoint category summarized in Table 8-1 reflect the measured uncertainty associated with two aspects of the analysis: avoided physical effects (both health and welfare benefits) and economic valuation of benefits. In addition, in Chapter 3 we conduct quantitative sensitivity analyses of key components of the direct cost estimates. For many other aspects of our analysis, however, including emissions esti-

[4] The primary central benefit-cost ratio for Title VI using a 3 percent discount rate is 44 to 1, higher than any of those presented in Table 8-4 (see Table 8-6 below). In addition, the ratio using a 2 percent discount rate, the rate used in the underlying RIAs, is 75 to 1. See Appendix G for more detail on the sensitivity of Title VI benefits to the choice of discount rate.

[5] The illustrative calculations presented here do not reflect discounting of the physical incidence of mortality.

[6] Because of Agency concerns regarding discounting of physical effects, the ratio presented here reflects undiscounted life-years saved. If future years were discounted, the implicit cost per life-year saved would be significantly higher.

mates, air quality modeling, and unquantified categories of benefits, we are unable to conduct quantitative analysis of uncertainty. Instead, we have attempted throughout this report to identify and characterize major sources of uncertainty — we present the results of these efforts at the end of Chapters 2 through 7. In this section, we provide a summary evaluation of the relative importance of key sources of uncertainty.

Table 8-5 below provides a summary of both quantified and unquantified sources of uncertainty and our estimates of the impact of these sources of uncertainty on the primary central estimates of benefits and costs. The table covers seven major categories of uncertainties: measurement uncertainties in physical effects and valuation components of the benefits analysis; measurement uncertainties in estimation of direct costs; alternative assumptions for PM-related mortality valuation; alternative assumptions for PM-related mortality risk; unquantified sources of error in emissions and air quality modeling; and omissions of key benefits categories. The table entries cover quantitative analyses of uncertainty, characterization of unquantified uncertainty, and the potential effect of alternative modeling paradigms for costs and benefits. Additional treatment of alternative paradigms is necessary because reasonable people may disagree with our methodological choices regarding these issues, and these choices might be considered to significantly influence the results of the study.

## *Quantitative Analysis of Physical Effects and Valuation Uncertainties*

As discussed previously in this chapter, we have conducted quantitative uncertainty analysis of our benefits estimates to reflect measurement error in two key steps of the analysis: estimation of physical effects and economic valuation. We present the results of our analysis in Figure 8-1 and Table 8-1 above. The procedure used to generate these estimates is well-suited to analysis of uncertainties where the probability of alternative outcomes can be quantitatively characterized in an objective manner. For example, most studies that estimate concentration-response relationships report an estimate of the statistical uncertainty around the central estimate. Because many estimates are available for the value of statistical life, we can use the discrete distribution of the best available estimates as a basis for quantitatively characterizing the probability of alternative values. It is important to recognize, however, that this procedure reflects only a portion of the range of possible sources of uncertainty in our benefits estimates. Other, nonquantified sources of uncertainty must also be factored into conclusions about the ratio of benefits to costs.

As part of our analysis of key contributors to uncertainty in benefits estimates, we also conducted a sensitivity analysis to determine the physical effects estimation and economic valuation variables with the greatest contribution to the quantified measurement uncertainty range. We present the results of this sensitivity analysis in Figure 8-2. In this sen-

**Figure 8-2**
**Analysis of Contribution of Key Parameters to Quantified Uncertainty**

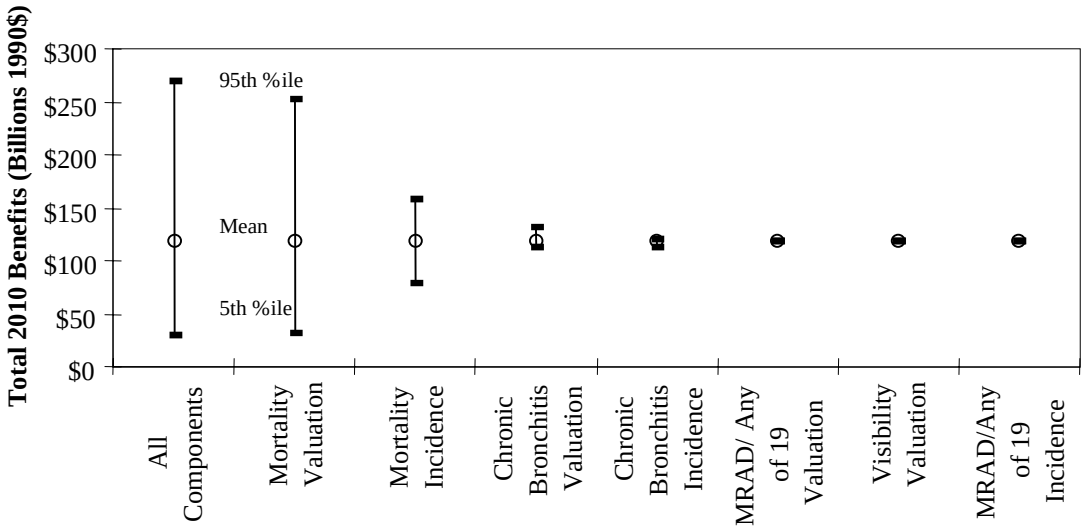


**Table 8-5**
**Summary of Key Sources of Uncertainty and Their Impact on Costs and Benefits**

| Source of Uncertainty | Description of Alternative Parameter Inputs | Impact on Annual Estimates in 2010 | |
|---|---|---|---|
| | | Costs | Benefits |
| Measurement error and uncertainty in the physical effects and economic valuation steps | Use a range of input assumptions to reflect statistical measurement uncertainty in concentration-response functions, modeling of physical effects, and estimation of economic values. Most important input parameters are value of statistical life and estimated relationship between particulate matter and premature mortality (see Chapters 5, 6, and 7). | None | For Titles I through V, effect of the use of alternative input assumptions ranges from a $84 billion decrease (5th percentile) to a $160 billion increase (95th percentile). |
| Measurement error and uncertainty in direct cost inputs | Use alternative assumptions for key input parameters for six of the highest cost provisions. Conduct sensitivity tests for each provision separately (see Chapter 3, pages 30 to 32). As discussed in Chapter 3 and in this chapter, aggregation of provision-specific results would be inappropriate. | High estimates for some provisions are $1 billion higher than primary estimate. Low estimates are as much as $2 billion below primary estimate | None |
| Value of statistical life-based estimates do not reflect age at death | Use estimates of the incremental number of life-years lost from exposure to ambient PM and a value of statistical life-year as opposed to measuring number of lives lost and a value of statistical life (see Chapters 5 and 6). | None | Decrease by $47 billion |
| Basis of estimate of avoided mortality from PM exposure | The Dockery et al. study provides an alternative estimate of the long-term relationship between chronic PM exposure and mortality (see Chapter 5). | None | Increase by $100 to $150 billion |
| Uncertainties in Title VI health benefits analysis | Major uncertainties include: estimating fatal cancer cases resulting from UV-b exposure; not accounting for future averting behavior; and not accounting for future improvements in the early detection and treatment of melanoma (see Table 5-6). | None | Not quantified, but net effect is probably that benefits estimates are too high. |
| Uncertainties in emissions and air quality steps | Major uncertainties include: underestimation of $PM_{2.5}$ emissions; omission of changes in primary and organic PM in eastern U.S.; emissions estimation uncertainties in the western U.S.; scarcity of $PM_{2.5}$ monitors; and lack of a fully integrated air quality and emissions modeling system (see Tables 2-5 and 4-7). | Uncertainties in emissions estimates affects some costs, but net effect is minor. | Not quantified, but net effect is probably that benefits estimates are too low. |
| Omission of potentially important benefits categories from primary estimate | Non-quantified categories of impacts summarized in Chapters 5 and 7. Quantified but omitted categories include household soiling, nitrogen deposition, and residential visibility (see Chapter 7). | None | Increase by at least $8 billion, (does not reflect unquantified categories) |

sitivity analysis, we hold constant all inputs to the probabilistic uncertainty analysis except one -- for example, the economic valuation of mortality. We allow that one variable to vary across the estimated range of that variable's uncertainty. The sensitivity analysis isolates the effect of this single source of uncertainty on the total measured uncertainty in estimated aggregate benefits. The first uncertainty bar represents the range associated with the total monetized benefits of the Clean Air Act, based on analysis of quantifiable components of uncertainty, as reported above. This range captures the multiple measurement uncertainties in the quantified benefits estimation. The rest of the uncertainty bars represent the quantified measurement uncertainty ranges generated by single variables. As shown in Figure 8-2, the most important contributors to aggregate quantified measurement uncertainty are mortality valuation and incidence, followed by chronic bronchitis valuation and incidence.

### Measurement Error and Uncertainty in Direct Cost Inputs

As noted in Chapter 3, explicit and implicit assumptions about changes in consumption patterns, input costs, and technological innovation are crucial to estimating the direct compliance costs of the CAAA. For many of the factors contributing to uncertainty, the degree and, in some cases, the direction of the bias are unknown or cannot be determined. Uncertainties and sensitivities can be identified, however, and in many cases the potential measurement errors can be quantitatively characterized. We designed our sensitivity analyses of key input parameters to provide a sense of the relative importance of various input parameters and assumptions necessary to generate estimates of direct costs. The sensitivity tests use ranges of input parameters that include all reasonable alternative estimates that we could identify.

The results indicate that the sensitivity of our primary central cost estimates is not uniform across provisions. Low and high estimates may vary by as much as a factor of two. Unlike our quantitative analysis of benefits, we do not assign probabilities to the likelihood of alternative input parameters. In our judgement, assignment of probabilities to these alternative outcomes would be a largely subjective task; we know of no objective means to develop these probabilities. As a result, it would be inappropriate simply to add up the array of low and the array of high estimates to arrive at an overall range of uncertainty around the central estimates, because it is unlikely that a plausible scenario could be constructed where all the estimates are concurrently either at the high or low end of their individual plausible ranges. A better interpretation of these results is that uncertainty in key input parameters can have a significant effect on the overall uncertainty of our estimates of direct compliance costs and ultimately the net benefits calculation.[7]

### PM Mortality Valuation Based on Life-Years Lost

The primary analytical results we present 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 EPA, some argue 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 rough 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 life expectancy of those who are exposed to PM.

The ability to estimate, however roughly, 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

[7] Although the analysis conducted here is a direct cost analysis, other sources of uncertainty would also need to be considered for a social cost analysis. For example, forecasts of key economic variables (e.g., interest rates), specification of production functions, and the reliability of key supply and demand elasticities are all important factors in social cost modeling that contribute to measurement uncertainty. In addition, most current social cost analyses assume that markets are currently operating under optimally efficient conditions. Emerging literature suggests that a full accounting of the social costs and efficiency impacts of environmental regulations could also include an assessment of the incremental costs that reflect existing market distortions, such as those imposed by the current tax code. Our assessment of uncertainties in direct cost estimates do not reflect these considerations.

of life-years saved.[8] As stated in our retrospective analysis, we have on occasion performed sensitivity calculations that adjust mortality values for those over age 65. Nonetheless, as discussed in Appendix H, the current state of knowledge and available analytical tools do not conclusively support using a life-years lost approach or any other approach which assigns different risk reduction values to people of different ages or circumstances. While we prefer an approach which makes no valuation distinctions based on age or other characteristics of the affected population, we present alternative results based on a VSLY approach below. The method used to develop life years lost estimates is described briefly in Chapter 5 and Appendix D. The method used to develop VSLY estimates is described in Appendix H.

The fourth row of Table 8-5 summarizes the effect of using a VSLY approach on results for 2010. The results indicate that the choice of valuation methodology significantly affects the estimate of the monetized value of 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 conclusion of this study, since the central estimate of monetized benefits of the CAAA still substantially exceeds the costs of compliance.

We emphasize that the results of the VSLY approach to valuing avoided mortality benefits represent a crude estimate of the value of changes in age-specific life expectancy. These results should be interpreted cautiously, due to the several significant assumptions required to generate a monetized estimate of life years lost from the relative risks reported in the Pope et al., 1995 study and the available economic literature. These assumptions include, but are not limited to: extrapolation of the age distribution of the U.S. population in future years; assumptions about the age-specificity of the relative risk reported by Pope et al., 1995; assumptions about the life expectancy of different age groups, adjustment of the life years lost estimates by an appropriate lag period (if any); assumptions about the age-specificity of the lag period (if any); derivation of VSLY estimates from VSL estimates; assumptions about the variation in VSLY with age; and selection of an appropriate rate at which to discount the lagged estimates of life years lost. Changes in any of these assumptions could significantly affect the VSLY benefit estimate. For example, if we were to assume no lag period for PM-related mortality effects instead of the five-year lag structure described in Chapter 5, VSLY benefit estimates would increase from $53 billion to $61 billion. The specific assumptions we used in generating these results are discussed in Appendix H.

### PM Mortality Incidence Using the Dockery Study

As described in Chapter 5, we chose to use the results of the Pope et al. (1995) study to estimate the magnitude of the effect of ambient PM exposure on the incidence of premature mortality. Alternative estimates do exist in the literature, however. Although we chose the Pope study because of its coverage of the largest number of cities and other technical advantages, the Dockery et al. (1993) study provides a credible and reasonable alternative to the Pope study. The Dockery study used a smaller sample of individuals in fewer U.S. cities than the Pope study, but it features improved exposure estimates, a slightly broader study population (including adults aged 25 to 30), and a follow-up period nearly twice as long as that used in the Pope study.

Use of the Dockery study in place of the Pope study would substantially increase the benefits estimate. As shown in the fifth row of Table 8-5, we estimate that using the Dockery study estimates would increase the annual central benefits estimate by $100 to $150 billion, more than doubling the total annual benefits for Titles I through V and, in turn, doubling the estimated benefit-cost ratio.

### Uncertainties in Title VI Health Benefits Analysis

As discussed in Chapter 5 and Appendix G, health benefits such as avoided mortality from melanoma and non-melanoma skin cancers constitute the majority of monetized benefits resulting from Title

[8] This issue was extensively discussed during the Science Advisory Board Council review of drafts of the retrospective 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. Consistent with SAB Council review advice for the present study, we apply the same approach in this analysis.

VI regulations on stratospheric ozone-depleting chemicals. Estimates of avoided mortality from skin cancer due to reduced UV-b exposure between 1990 and 2165 represent over 90 percent of the total health benefits of Title VI. As a result, uncertainties related to avoided mortality estimation under Title VI represent key uncertainties for our overall CAAA benefits estimate. Three main areas of uncertainty are important for our avoided mortality estimates for Title VI: dose-response relationships; predicting averting behavior; and predicting future medical advancements.

Because the literature on the relationship between exposure to ultraviolet rays and melanoma and non-melanoma mortality is not as well developed as that for other health effects, the dose-response functions for both of these endpoints are characterized by significant uncertainty. The association of UV-b exposure with melanoma is controversial, although studies suggest that sunlight exposure is a major environmental risk factor for melanoma. If one assumes that a causal relationship exists between UV-b rays and melanoma, uncertainty still remains about three aspects of the nature of the dose-response relationship. Specifically, the relative contribution of different wavelengths of light to melanoma development, the critical exposure period (e.g., acute, intermittent, or chronic), and the existence (and length) of a latency period between UV exposure and disease are all unclear. The effect of the first two uncertainties on our results cannot be determined from available information. If a significant latency period exists, then the third uncertainty may indicate that our analysis, which does not include a latency period, overestimates avoided melanoma mortality benefits. Because limited data on non-melanoma mortality precluded the development of a dose-response function for this endpoint in the current analysis, our estimate of non-melanoma skin cancer mortality resulting from UV-b exposure is calculated indirectly, by assuming the mortality rate is a fixed percentage of non-melanoma incidence. New data on the death rate for non-melanoma skin cancer may significantly influence this mortality estimate.

Our analysis of avoided mortality also does not incorporate adjustments for future increases in averting behavior (i.e., efforts by individuals to protect themselves from UV-b radiation ). Our estimates rely on epidemiological studies that incorporate averting behavior as currently practiced. However, if people would react to increased skin cancer risk in the future by applying sun screen more frequently, spending more time indoors or otherwise reducing their UV-b exposure, then our estimate of avoided mortality would significantly overestimate Title VI benefits. It is not certain, though, that individuals will pursue such behavior, and studies show that those engaging in averting behavior may also alter their behavior in ways that may increase exposure or risk, counteracting the benefits of averting behavior. For example, a recent study of young Europeans by Autier et al. (1999) found that the use of high sun protection factor (SPF) sun screen is associated with increased frequency and duration of sun exposure.

Finally, our analysis does not adjust estimates of future mortality for possible advances in medical technology that could lead to earlier detection and more effective treatment of melanomas. Such advancements could significantly reduce the expected future melanoma mortality, and by not adjusting for such developments, we may be overestimating avoided melanoma mortality. However, future research may also identify additional adverse human health outcomes associated with UV exposure that we have not considered in this analysis, resulting in an underestimate of Title VI benefits.

### *Uncertainties in Emissions and Air Quality Steps*

The emissions estimates presented in this analysis are a critical component of the overall analysis. As the starting point for both costs and benefits, they provide a consistent basis for evaluating the economic efficiency of the CAAA. Characterizing emissions can be very difficult, however, particularly for those source categories where emissions monitoring data are sparse or nonexistent. In general, all our emissions estimates are affected by three major sources of uncertainty: estimation of the base-year inventory, prediction of the growth in pollution-generating activity, and assumptions about future-year controls.

Base-year emissions were estimated using emissions factors that express the relationship between a particular human/industrial activity and the level of

emissions. The accuracy of base-year emissions estimates varies from pollutant to pollutant, depending largely on how directly the selected activity and emissions correlate. We likely estimated 1990 $SO_2$ emissions with the greatest precision. Sulfur dioxide emissions are generated during combustion of sulfur-containing fuel and are directly related to fuel sulfur content. In addition, we were able to verify these estimates through comparison with Continuous Emission Monitoring (CEM) data. As a result, we were able to accurately estimate $SO_2$ emissions using emissions factors based on data on fuel usage and fuel sulfur content. Nitrogen oxides are also a product of fuel combustion, allowing us to estimate emissions of this pollutant using the same general technique used to estimate $SO_2$ emissions. However, the processes involved in the formation of $NO_x$ during combustion are more complicated than those involved in the formation of $SO_2$; thus, our $NO_x$ emissions estimates are more variable and less certain than $SO_2$ estimates.

Volatile organic compounds, like $SO_2$ and $NO_x$, are products of fuel combustion; however, these compounds are also a product of evaporation. To estimate evaporative emissions of this pollutant we used emissions factors that relate changes in emissions to changes in temperature. Because future meteorological conditions are difficult to predict, the uncertainty associated with forecasting temperature influences the uncertainty in our VOC emissions estimates. The likely significance of this uncertainty, in terms of its impact on the overall monetary benefit present in this analysis, is probably minor.

Of particular importance, however, are uncertainties that affect the estimation of future year emissions of particulate matter and secondarily formed PM precursors. In this analysis we estimated primary $PM_{2.5}$ emissions based on unit emissions that may not accurately reflect the composition and mobility of particles. The ratio of crustal to carbonaceous particulate material, for example, likely is high as a result of overestimation of the fraction of crustal material, primarily composed of fugitive dust, and underestimation of the fraction of carbonaceous material. Because the CAAA have a greater impact on emissions sources that generate carbonaceous particles (mobile sources) than on sources that mainly emit crustal material (area sources), we likely underestimate the impact of the CAAA on reducing $PM_{2.5}$, thereby reducing monetary benefits estimates. The uncertainty associated with estimating the partition of $PM_{2.5}$ emissions components could conceivably have a major impact on the net benefit estimate. Compared to secondary $PM_{2.5}$ precursor emissions, however, changes in primary $PM_{2.5}$ emissions have a relatively small impact on $PM_{2.5}$ related benefits.

Our future-year control assumptions are also a source of uncertainty. Despite our efforts to minimize this uncertainty, whether each of the Post-CAAA controls will be adopted, whether Post-CAAA control programs will be more or less effective than estimated, and whether unanticipated technological shifts will reduce future-year emissions are all unknown. For example, the Post-CAAA scenario includes implementation of a region-wide $NO_x$ control strategy designed to regulate the regional transport of ozone. However, the control program assumed under the Post-CAAA scenario may not reflect the $NO_x$ controls that are actually implemented in a regional ozone transport rule.

In addition to potential inaccuracies in the emissions inventories used as air quality modeling inputs, there are at least three sources of air quality modeling uncertainty that may have a major effect on the precision and accuracy of our projected changes in air quality. First, we estimate changes in PM concentrations in the eastern U.S. based exclusively on changes in the concentrations of sulfate and nitrate particles. By not accounting for changes in organic and primary particulate fractions, we likely underestimate the impact of the CAAA on PM concentrations. Second, by using separate air quality models for individual pollutants and different geographic regions, as opposed to a single integrated model, we were unable to fully capture the interaction among air pollutants or reflect transport of pollutants or precursors across the boundaries of the models covering the western and eastern states. Third, the lack of a well-developed modeling network for $PM_{2.5}$ means we must estimate monitored concentrations of this pollutant based on $PM_{10}$ monitor estimates. The direction and magnitude of bias these limitations impose on net benefits estimate presented in this analysis can not be determined based on current information.

Some model-related uncertainties, however, may be mitigated because this analysis uses the air qual-

ity modeling results in a relative, not absolute, sense. We focus on the change in air quality between the Pre- and Post-CAAA scenarios and not on the ambient concentrations projected by the individual models themselves. Therefore, uncertainties that affect a model's ability to accurately predict the relative change in concentration of a pollutant from one scenario to another are more important in the context of this study than those that affect only the absolute model results. In addition, as summarized in the previous chapters, most of the uncertainties in emissions estimation and air quality modeling contribute to a conservative bias in our benefits results. When faced with alternative approaches to emissions and air quality modeling, we made explicit attempts to choose parameters, assumptions and modeling strategies that would tend to understate benefits.

## Omission of Potentially Important Benefits Categories

As described in Chapters 5 through 7 above, and in more detail in Appendix H, the primary estimate reflects application of a strict set of criteria for inclusion of monetized benefits categories. For example, estimates of the value of improved visibility in U.S. residential areas indicate a positive value for this service flow, but the best available residential visibility estimates rely on an unpublished study of values in the eastern U.S. Although our physical effects analysis indicates significant visibility improvements in all regions of the U.S., our application of the results of the economic valuation literature reflect a conservative approach to valuation of improved visibility in the U.S. While we believe our conservative inclusion criteria for the primary benefits reflects the greater uncertainty in measuring some economic values, we also believe that the statutory language of section 812 clearly warns against the practice of assuming a default value of zero for demonstrated categories of benefits. Therefore, the last row of Table 8-5 presents the effect of using a somewhat more inclusive set of criteria for accepting benefits transfer-based economic values. In this alternative case, we included estimates for improved residential visibility, displaced costs from reduced airborne nitrogen loadings to estuaries, and reduced expenditures for household soiling (which are not included in any form in the primary estimate).

In addition to these quantified but omitted categories of benefits, there is a wide range of benefits of the CAAA that we can identify but cannot quantify. We present summaries of unquantified health effects in Chapter 5 (Tables 5-1 and 5-5) and unquantified ecological and welfare effects in Chapter 7 (Tables 7-5 and 7-9). Two of the most important omissions, in our judgement, are the lack of any quantified estimates for the health benefits of air toxics control and the omission of the systemic and long-term ecological effects of mercury and other persistent air pollutants. The importance of these two categories of effects are discussed in Chapters 5 and 7, respectively.

## Alternative Discount Rates

In some instances, the choice of discount rate can have an important effect on the results of a benefit-cost analysis; for example, when the distribution of costs and benefits throughout the time period are very different from one another. In this assessment, the discount rate affects annualized costs (i.e., amortized capital expenditures), and the discounting of all costs and benefits to 1990. Table 8-6 summarizes the effect of alternative discount rates on the Primary Central estimate results of this analysis. The estimates we present show that altering the discount rate has only a small effect on annual cost and benefit estimates. In part, this is due to limitations in our ability to conclusively identify costs as annualized capital expenditures or annual operating costs in the underlying estimates. As described in Chapter 3, about $3 billion (or roughly 10 percent) of the 2010 estimate is annualized capital costs. Varying the discount rate, which we also use to represent the cost of capital, affects only this component of costs. The benefits estimates that employ a discount rate include the mortality estimate, where it is used as part of our valuation of the lag effect of PM mortality, and the chronic asthma value, where we use a discount rate to develop a lump-sum value for avoidance of incidence from an annual payment value in the underlying literature.

Not surprisingly, the effect of discount rates on the net present value benefit calculations is greater. Nonetheless, the estimates we present in Table 8-6 show that varying the discount rate assumption also does not change our overall conclusion that the benefits of the CAAA exceed its costs.

**Table 8-6**

**Effect of Alternative Discount Rates on Primary Central Estimates (Estimates in million 1990$)**

| | Discount Rate Assumption | | |
|---|---|---|---|
| | **3%** | **5%** | **7%** |
| ***Annual Costs in 2010:*** | | | |
| Titles I through V | $26,600 | $26,800 | $26,900 |
| ***Annual Benefits:*** | | | |
| Titles I through V | $110,000 | $110,000 | $107,000 |
| ***Present Value of Costs:*** | | | |
| Titles I through V | $230,000 | $180,000 | $140,000 |
| Title VI | $43,000 | $27,000 | $20,000 |
| ***Present Value of Benefits:*** | | | |
| Titles I through V | $890,000 | $690,000 | $520,000 |
| Title VI | $1,900,000 | $530,000 | $240,000 |
| ***Cumulative Net Benefits:*** | | | |
| Titles I through V | $650,000 | $510,000 | $380,000 |
| Title VI | $1,860,000 | $500,000 | $220,000 |
| ***Benefit/Cost Ratio:*** | | | |
| Titles I through V | 4/1 | 4/1 | 4/1 |
| Title VI | 44/1 | 20/1 | 12/1 |

# Emissions Analysis

Appendix A

The prospective analysis examines the emissions of eight air pollutants: volatile organic compound (VOC), nitrogen oxides ($NO_x$), sulfur dioxide ($SO_2$), carbon monoxide (CO), particulate matter of ten microns or less ($PM_{10}$), particulate matter with an aerodynamic diameter of 2.5 microns or less ($PM_{2.5}$), ammonia ($NH_3$), and mercury (Hg). Changes in emissions of these pollutants were projected based on two emissions control scenarios: a) pre-1990 Clean Air Act Amendments ("Pre-CAAA") scenario assuming that no additional controls would be implemented beyond those that were in place when the CAAA were passed; and b) "Post-CAAA" scenario incorporating the effects of controls authorized by the 1990 Amendments. Comparison of the resulting projections revealed the predicted impact of the CAAA on emissions. In addition, these estimates provided the basis for the subsequent cost estimation and air quality modeling steps of the prospective analysis.

This appendix summarizes the Pre- and Post-CAAA emissions estimates for each of the major sources of VOC, $NO_x$, $SO_2$, CO, $PM_{10}$, $PM_{2.5}$, $NH_3$, and Hg and describes the methodology for the Agency's projections. EPA based its Pre- and Post-CAAA emissions estimates on projections from 1990 base year emissions estimates. For all of the pollutants, except particulate matter and mercury,[1] the Agency selected emissions levels taken from Version 3 of the National Particulates Inventory (NPI) to serve as the baseline. For both $PM_{10}$ and $PM_{2.5}$, however, EPA updated NPI estimates to reflect the emissions from the National Emission Trends (NET) inventory. Once EPA finalized the base year levels, the Agency projected 1990 emissions to 2000 and 2010 under both the Pre- and Post- CAAA scenarios. At the time the emissions data base selection was made, the NPI was the most comprehensive source of emission estimates of all criteria pollutant emissions. Other available data sets, such as that available from the Ozone Transport Assessment Group (OTAG), were considered, but not selected because they were limited to ozone precursor emissions.

EPA estimated future emissions for all major source categories: industrial point sources, utilities, nonroad engines/vehicles, motor vehicles, and area sources. To make these projections, for all but utility sources, the Agency relied on emissions analysis that incorporated growth forecasts and future year control assumptions about rule effectiveness and control efficiency. In this analysis EPA projected growth largely based on anticipated changes in economic activity, and treated the rule effectiveness and the rate of control efficiency as the key differences between the Pre- and Post-CAAA scenarios.

EPA used the Integrated Planning Model (IPM) to estimate utility emissions. With this optimization model EPA forecasts emissions for the 48 contiguous States and the District of Columbia. All existing electric power generation units are covered in the model, as well as independent power producers and other cogeneration facilities that sell wholesale power, if they were included in the North American Electric Reliability Council (NERC) data base for reliability planning. The model considers future capacity additions by both utilities and independent power producers. In addition, this model is capable of producing baseline air emission forecasts and estimates of air emissions levels under various control scenarios at the national, and NERC region and subregional, level. A full explanation of the IPM model and the assumptions EPA used for this prospective analysis may be found in Analyzing Electric Power Generation under the CAAA, EPA, July 1996.[2]

This appendix first provides an overview of Pre- and Post-CAAA scenario development. It discusses

[1] A separate methodology was used to estimate mercury emissions. Mercury emissions are discussed independently beginning on page A-48 of this appendix.

[2] This document was updated in March 1998 to describe model refinements made for IPM Version 7.1 and the latest base case forecasts. (EPA, Office of Air and Radiation, Analyzing Electric Power Generation under the CAAA, March 1998.)

key factors influencing the 2000 and 2010 estimates, such as the timing of modeling decisions with respect to control programs and available data. In the following section, there is a comparison of the prospective analysis base year inventory and emissions projections with other existing data. Subsequent sections address the influence of Title 1 progress requirements and the major sources of uncertainty, and provide detailed breakdowns of emissions projections for the five major source categories responsible for air pollution: industrial point, utility, nonroad, motor vehicle, and area sources. For each of these source categories this appendix provides: (1) an overview of the approach used to make the emissions estimates; (2) a discussion of how base year emissions levels were determined; (3) an explanation of how growth projections were determined; (4) an outline of the assumptions made for the control scenarios; and (5) a summary of the emissions estimates for both the Pre- and Post-CAAA scenarios.

## Scenario Development

EPA projected emissions by adjusting 1990 base year emissions to reflect projected economic activity levels in 2000 and 2010, and applying future year control assumptions. The resulting estimates depended largely upon three factors: how the base year inventory was selected, what indicators were used to forecast growth, and what specific regulatory programs were incorporated in the Pre- and Post-CAAA scenarios. These three factors are addressed in Tables A-1 through A-3. Table A-1 highlights the approach EPA used to establish the base year inventory. The indicators the Agency relied on to forecast growth and predict future activity levels, along with the analytical approach EPA used to project emissions, are shown in Table A-2. The Pre- and Post-CAAA regulatory scenarios are summarized in Table A-3.

Of the factors that influence EPA's emissions projections for 2000 and 2010, the most significant is the suite of air pollution regulations and programs the Agency incorporated in the Pre- and Post-CAAA scenarios. For the Pre-CAAA scenario, air pollution controls are frozen at their 1990 levels; only standards and initiatives implemented prior to the CAAAs are included. The Post-CAAA scenario, in addition to the measures contained in the Pre-CAAA scenario, incorporates emission controls associated with the 1990 Amendments. Due to the necessity of developing emissions scenarios early in the prospective analysis process, the exact provisions of some regulatory programs could not be foreseen. For example, decisions about how to translate the OTAG recommendations into regional $NO_x$ control requirements had not been made, so estimates were made on affected sources, geographic coverage, and control levels.

EPA included in the Post-CAAA scenario:

- Title I VOC and $NO_x$ reasonably available control technology (RACT) and reasonable further progress (RFP) requirements for ozone nonattainment areas (NAAs);
- Title II motor vehicle and nonroad engine/vehicle provisions;
- Title III 2- and 4-year maximum achievable control technology (MACT) standards;
- Title IV $SO_2$ and $NO_x$ emissions programs for utilities;
- Title V permitting system for primary sources of air pollution; and
- Title VI emissions limits for chemicals that deplete stratospheric ozone.

This scenario also assumes the implementation of a region-wide $NO_x$ cap and trade system for the entire OTAG domain[3] and a similarly designed trading program for the Ozone Transport Region (OTR) that is consistent with Phase II of the Ozone Transport Commission (OTC) $NO_x$ Memorandum of Understanding (MOU). For motor vehicles, emission reductions associated with a 49-State low emission vehicle (LEV) program were also included in the Post-CAAA scenario. A more detailed outline of the controls included in both the Pre- and Post-CAAA scenarios is provided in Table A-3.

[3]The $NO_x$ control program incorporated in the Post-CAAA scenario may not reflect the $NO_x$ controls that are actually implemented in a regional ozone transport rule.

**Table A-1**
**Base Year Inventory - Summary of Approach**

| Sector | Analysis Approach/Data Sources |
|---|---|
| Industrial Point Sources | 1985 National Acid Precipitation Assessment Program (NAPAP) emissions inventory grown to 1990 based on historical Bureau of Economic Analysis (BEA) earnings data.<br>$PM_{10}$ emissions based on total suspended particulate (TSP) emissions and particle-size multipliers. |
| Utilities | The 1990 utility emission estimates are from the 1990 NPI. |
| Nonroad | Nonroad Engines/Vehicles (VOC, $NO_x$, CO, $PM_{10}$): 1991 Office of Mobile Sources (OMS) Nonroad Inventory.<br>Nonroad Engines/Vehicles ($SO_2$) and Aircraft, Commercial Marine Vessels, Railroads: 1985 NAPAP grown to 1990 based on historical BEA earnings data. |
| Motor Vehicles | Federal Highway Administration (FHWA) travel data, MOBIL5a/PART5 emission factors. |
| Area Sources | 1985 NAPAP inventory grown to 1990 based on historical BEA earnings data and State Energy Data Systems (SEDS) fuel use data; emission factor changes for selected categories. |

**Table A-2**
**Analysis Approach by Major Sector**

| Sector | Growth Forecast | Analysis Approach |
|---|---|---|
| Industrial Point | 1995 BEA Gross State Product (GSP) Projections by State/Industry. | VOC, $NO_x$ — Emission Reduction and Cost Analysis Model (ERCAM): applies BEA growth projection to base year emissions and applies future year controls as selected by the user.<br>$PM_{10}$, $SO_2$, CO — While no formal model exists, the same basic approach applied in ERCAM was used for these pollutants. |
| Utilities | Projections of heat input by unit based on National Electric Reliability Council (NERC) data, price and demand forecasts, and technology assumptions. | $SO_2$, $NO_x$ — Integrated Planning Model (IPM).<br>VOC, $PM_{10}$, CO — Base year emissions rates or AP-42 emission factors applied to IPM projected heat input by unit. |
| Nonroad | 1995 BEA GSP and Population Projections by State/Industry. | VOC, $NO_x$ — ERCAM.<br>$PM_{10}$, $SO_2$, CO — ERCAM approach (no formal model). |
| Motor Vehicles | MOBILE Fuel Consumption Model (FCM) National Vehicle Miles Traveled (VMT) Projection Scaled to Metropolitan/REST-of-State Areas by Population. | $NO_x$, VOC, CO — ERCAM: applies MOBIL5a emission factors to projected VMT by month and county/vehicle type/roadway classification.<br>$PM_{10}$, $SO_2$ — PART5 emission factors applied to projected VMT. |
| Area | 1995 BEA GSP and Population Projections by State/Industry, and USDA Agricultural Projections. | VOC, $NO_x$ — ERCAM.<br>$PM_{10}$, $SO_2$, CO — ERCAM approach (no formal model). |

**Table A-3**
**Projection Scenario Summary by Major Sector**

| Sector | Pre-CAAA | Post-CAAA* |
|---|---|---|
| Industrial Point | RACT held at 1990 levels. | VOC and $NO_x$ RACT for all NAAs (except $NO_x$ waivers).<br>New control technique guidelines (CTGs) (VOC).<br>0.15 pounds per million British thermal unit (lbs/MMBtu) OTAG-wide NOx cap on fuel combustors of 250 MMBtu per hour or above($NO_x$).<br>OTAG Level 2 $NO_x$ controls across OTAG States ($NO_x$).<br>2- and 4- year MACT standards (VOC).<br>Ozone Rate-of-Progress (3 percent per year) requirements (further reductions in VOC). |
| Utilities | 250 ton prevention of Significant Deterioration (PSD) and New Source Performance Standards (NSPS) held at 1990 levels.<br>RACT and New Source Review (NSR) held at 1990 levels. | Title IV $SO_2$ emission allowance program ($SO_2$).<br>Title IV Phase I and Phase II emission limits for all boiler types ($NO_x$).<br>250 ton PSD and NSPS.<br>RACT and NSR for all non-waived ($NO_x$ waiver) NAAs ($NO_x$).<br>Phase II of the Ozone Transport Commission (OTC) NOx memorandum of understanding (MOU) ($NO_x$).<br>0.15 lbs/MMBtu OTAG-wide seasonal $NO_x$ cap with banking/trading ($NO_x$). |
| Nonroad | Controls (engine standards) held at 1990 levels. | Federal Phase I and II compression ignition (CI) engine standards ($NO_x$, PM).<br>Federal Phase I and II spark ignition (SI) engine standards (VOC, CO, $NO_x$).<br>Federal locomotive standards ($NO_x$, PM).<br>Federal commercial marine vessel standards ($NO_x$).<br>Federal recreational marine vessel standards (VOC, $NO_x$). |
| Motor Vehicles | Federal Motor Vehicle Control Program (FMVCP) — engine standards set prior to 1990.<br>Phase 1 Reid vapor pressure (RVP) limits.<br>I/M programs in place by 1990. | Tier 1 tailpipe standards (Title II) (VOC, $NO_x$).<br>49-State LEV program (Title I) (VOC, $NO_x$, CO).<br>Phase 2 RVP limits (Title II) (VOC).<br>I/M programs for ozone and CO NAAs (Title I) (VOC, $NO_x$, CO).<br>Federal reformulated gasoline for ozone NAAs (Title I) (VOC, $NO_x$, CO).<br>California LEV (California only) (Title I) (VOC, $NO_x$, CO).<br>California reformulated gasoline (California only) (Title I) (VOC, $NO_x$, CO).<br>Diesel fuel sulfur content limits (Title II) ($SO_2$, PM).<br>Oxygenated fuel in CO NAAs (Title I) (CO). |

**Table A-3**
**Projection Scenario Summary by Major Sector**

| Sector | Pre-CAAA | Post-CAAA* |
|---|---|---|
| Area | Controls held at 1990 levels. | VOC and $NO_x$ RACT requirements.<br>New CTGs (VOC).<br>2- and 4- year MACT standards (VOC).<br>Ozone Rate-of-Progress (3 percent per year) requirements (further reductions in VOC).<br>PM NAA controls (PM).<br>Onboard vapor recovery (vehicle refueling) (VOC).<br>Stage II vapor recovery systems (VOC). |

*Also includes all Pre-CAAA measures.

Decisions about which control programs to include in the Post-CAAA scenario and how their emission reductions were modeled, were made during the summer of 1996. While some adjustments to the emission projection methods were made since that time in response to review comments, opportunities were not available to revise the emission projections once air quality modeling was initiated using these data sets. The result is that there are some differences between the emissions modeling practices being used currently in EPA regulatory analyses and those reflected in the Section 812 Prospective. As examples, the VOC emission reductions of 7 and 10 year MACT standards are not included in the Prospective, nor are CO, $NO_x$, $SO_2$, and PM emission benefits of any MACT standards. Similarly, the $NO_x$ State Implementation Plan (SIP) Call affects 22 States, not 37, and OTAG Level 2 controls are not applied to all non-electrical generating units (EGUs) $NO_x$ sources, only to cement kilns and internal combustion engines.

## Comparison of the Base Year Inventory and Emissions Projections with Other Existing Data

Comparison of the emissions estimates in the prospective analysis to historical emissions estimates drawn from EPA's National Air Pollutant and Emissions Trends reports (hereafter referred to as *Trends*) can provide a check on the reasonableness of our emissions inventories. In addition, comparison of emissions projections from the prospective analysis with those of the Grand Canyon Visibility Transport Commission (GCVTC) study of western regional haze (Radian, 1995; Science & Policy Associates, 1995; Argonne, 1995) provides an initial test of the sensitivity of emissions projections to baseline inventories and growth assumptions. Analysis of PM emissions and comparison of estimated PM data with observed PM data presented in the 1997 National Air Quality and Emissions Trends Report (EPA, 1998a) also helps evaluate the prospective study's emissions estimation methods.

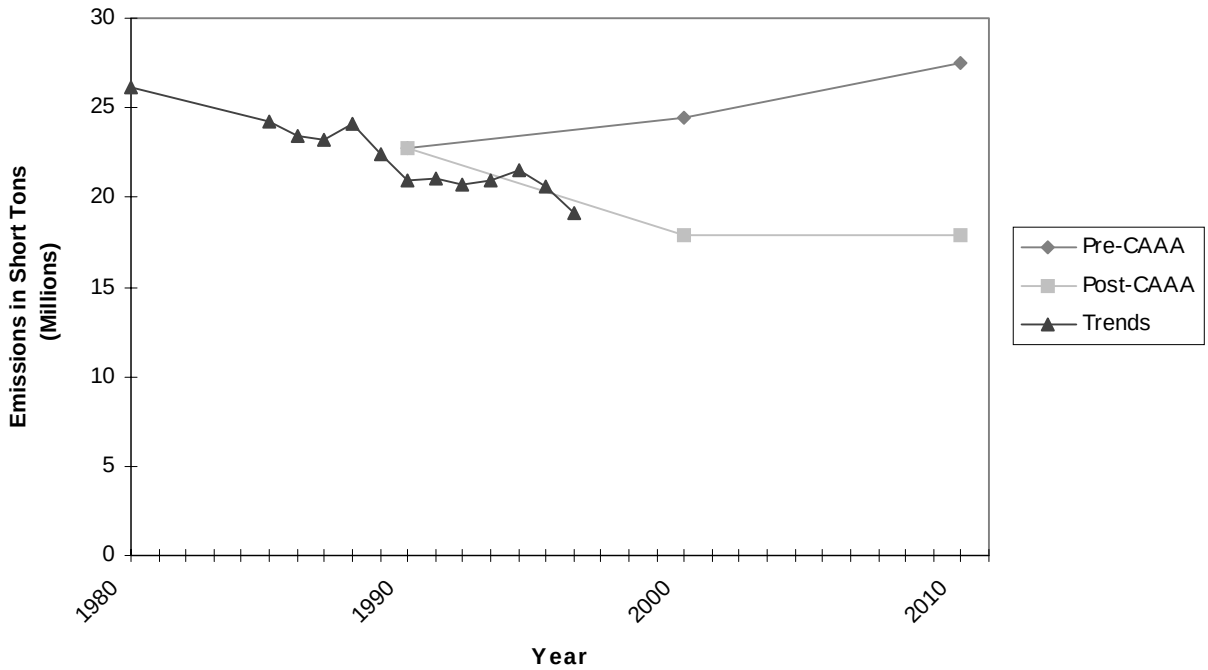
### *Post-CAAA Emissions Estimates and EPA* Trends *Data*

EPA publishes annual National Air Pollutant Emission Trends reports that contain estimates of historical trends in emissions of VOC, $NO_x$, $SO_2$, CO, $PM_{10}$, and lead (Pb). Comparison of the *Trends* (EPA, 1997a) and Post-CAAA estimates reveals that from 1990 to 1995, VOC, $NO_x$, $SO_2$, CO, and $PM_{10}$ emissions figures from both are similar.

Figures A-1 through A-5 display the Pre-CAAA, Post-CAAA, and *Trends*, emissions estimates for VOC, $NO_x$, $SO_2$, CO, and $PM_{10}$ respectively. Although the Post-CAAA and *Trends* emissions trends are comparable for all five of these pollutants, there are several instances where there are differences between the estimates from these two different sources. VOC emissions are highly variable from year to year. To illustrate this fluctuation, annual *Trends* estimates, and five year *Trends* increments, are provided in Figure A-1 for the years 1985 to 1996. Although the 1990 *Trends* estimate is lower than the prospective analysis' base year inventory, the general emissions trend projected under the Post-CAAA scenario is similar to that represented by the historical EPA estimates .[4]

[4]VOC emissions estimates not only fluctuate from year to year, but also from *Trends* report to *Trends* report. Had the *Trends* report published in October of 1996, a year earlier, been used as the data source for Figure A-1, the 1990 *Trends* VOC estimate would have been higher, not lower, than the prospective analysis' base year inventory.

**Figure A-1. Comparison of Pre-CAAA, Post-CAAA, and *Trends* VOC Emissions Estimates**




As shown in Figure A-2, the 1995 *Trends* estimate for $NO_x$ emissions exceeds the Post-CAAA $NO_x$ projection. The primary influence on $NO_x$ emissions in the mid-1990s was the requirement to install RACT on major stationary source $NO_x$ emitters in certain ozone nonattainment areas, and the Northeast OTR. Because the EPA *Trends* report is still in the process of incorporating the State's 1996 periodic emission inventories in the NET data base, it is believed that the *Trends* values in Figure A-2 do not capture all of the $NO_x$ emission reductions that have occurred to date. When these State data are incorporated in *Trends*, it is expected that the 1990 to 1996 $NO_x$ emissions trend line will more closely parallel the Post-CAAA estimates.

Comparison of $SO_2$ Post-CAAA and *Trends* estimates based upon the profiles plotted in Figure A-3 shows that the 1995 *Trends* estimates are somewhat lower than the corresponding prospective projection line. This is because the *Trends* profile reflects the sudden reduction in $SO_2$ emissions that resulted from the implementation of the acid rain $SO_2$ trading program in the early 1990's. While this reduction is incorporated in the Post-CAAA scenario, it is not captured until the year 2000 emissions projections; plotting the corresponding trend line has the effect of distributing these early reductions over the entire decade in constant annual increments. Between 1995 and 2000 the actual rate of $SO_2$ reduction will almost certainly slow so that by the year 2000 Post-CAAA and *Trends* estimates will be much more comparable than the 1995 levels depicted in Figure A-3.

Post-CAAA and *Trends* emissions estimates for CO (Fig. A-4) and $PM_{10}$ (Fig. A-5) respectively are similar for the years 1990 and 1995. For $PM_{10}$, the values used to develop the Pre- and Post-CAAA, and *Trends* profiles were adjusted to eliminate the influence of wind erosion, a natural source of $PM_{10}$ that can cause significant fluctuation in emission estimates from year to year. Even though this source is

controlled for, there is still significant variability in the yearly historical $PM_{10}$ emissions estimates. To capture this variability in the *Trends* estimates, instead of providing estimates at five year increments, annual *Trends* emissions levels are displayed in Figure A-5. For the years since the adoption of the 1990 CAAA, both the *Trends* estimates and the Post-CAAA scenario projections show $PM_{10}$ emissions remaining at the same relative level. The drastic drop in the *Trends* line from 1989 to 1990 is the result of a change in methodology used to calculate $PM_{10}$ emissions.

Figures A-1 through A-5 contain *Trends* estimates for 1980 and 1985 in addition to the 1990 and 1995 values primarily discussed above. This information is included to provide a broader picture of actual emissions levels over the last 15 years and to show how the general historical trends in emissions compare to the projected future trends under both the Pre- and Post-CAAA scenarios. Close comparison of pre-1990 *Trends* estimates with 1990 to present *Trends* estimates and prospective analysis projections, however, has the potential to be misleading. Beginning in 1990 there was a significant change in the methodology used to estimate *Trends* emissions. The 1980 and 1985 figures presented here are intended only for general comparison.

**Figure A-2. Comparison of Pre-CAAA, Post-CAAA, and *Trends* $NO_x$ Emissions Estimates**

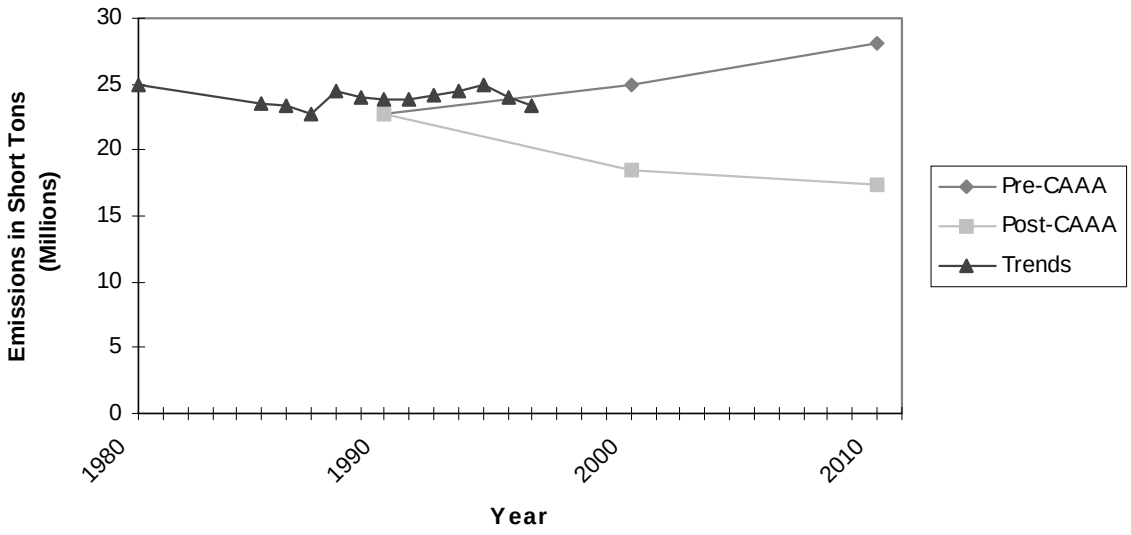



**Figure A-3. Comparison of Pre-CAAA, Post-CAAA, and *Trends* SO Emissions Estimates**

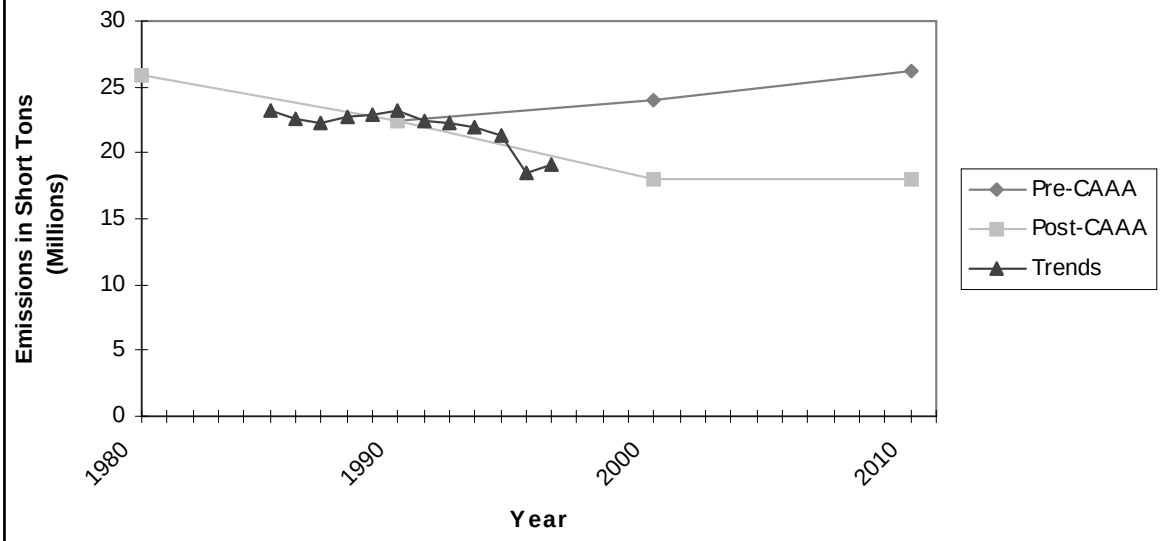


**Figure A-4. Comparison of Pre-CAAA, Post-CAAA, and *Trends* CO Emissions Estimates**

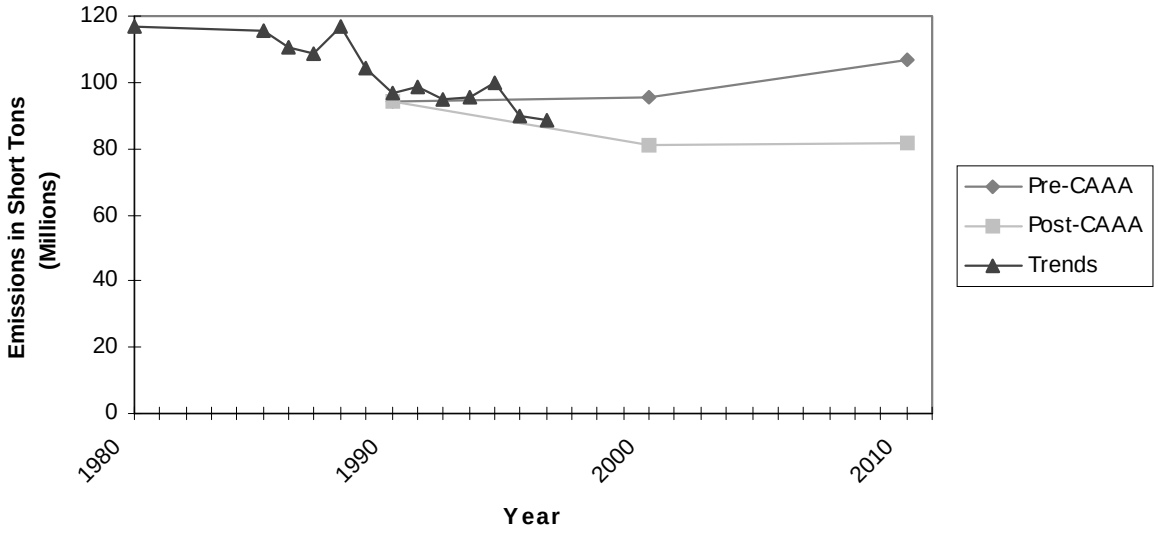
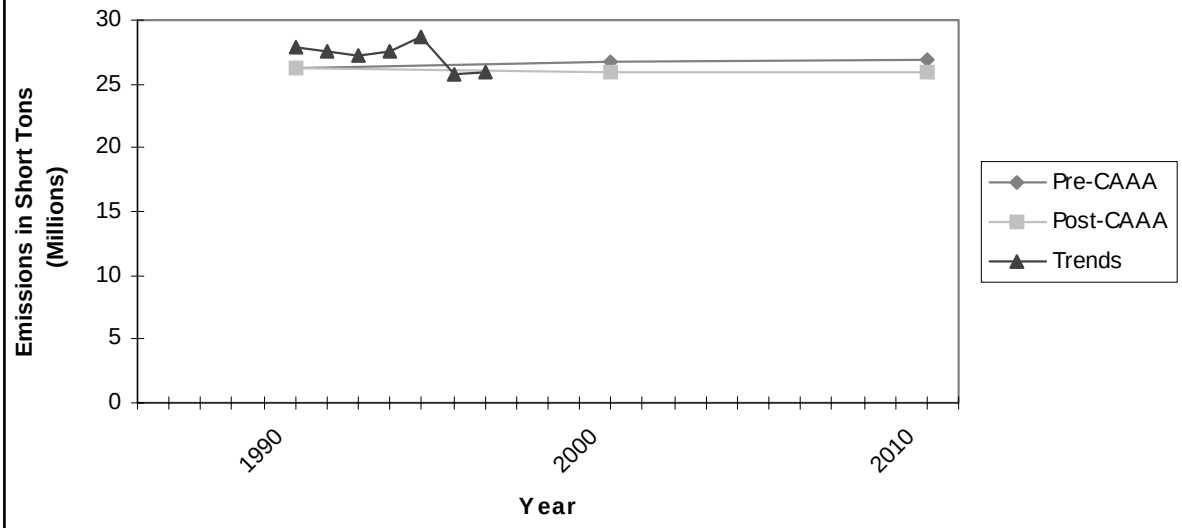



**Figure A-5. Comparison of Pre-CAAA, Post-CAAA, and *Trends* $PM_{10}$ Emissions Estimates**



## Prospective Analysis and GCVTC Emissions Estimates

The GCVTC conducted, for the Western States, an air pollution analysis that projected emissions for selected pollutants from 1990 base year levels for the year 2000 and every tenth subsequent year up to 2040. Comparison of the 2000 and 2010 GCVTC study and prospective analysis Post-CAAA scenario projections for the Western States indicate that although their estimates for $NO_x$, $SO_2$, and $PM_{2.5}$ are somewhat different, these differences arise from the use of different baseline inventories in the two studies, or from specific regional reductions not incorporated in the prospective study scenarios (Table A-4).

$NO_x$ and $SO_2$ base year figures in the prospective analysis are approximately 10-15 percent higher than the corresponding estimates in the GCVTC study. The difference is most likely the result of the separate inventories that are relied upon by the two analyses to develop their respective base year emissions levels. Version 3 of the NPI, the primary source of base year emissions data for the prospective analysis, is largely derived from 1985 emissions figures that are adjusted to 1990 levels using BEA growth projections. This inventory does not capture the effect of new controls and technology change on emissions between 1985 and 1990. The GCVTC base year estimates for $NO_x$ and $SO_2$, based primarily on State provided point source emissions figures from one of the years 1990 to 1992, however, incorporate these effects. As a result, 1990 emissions estimates in the GCVTC study are lower than those in the prospective analysis.

Due to the difference in the two studies' base year $NO_x$ estimates, their projected absolute levels of $NO_x$ emissions also differ. Both studies, however, estimate that $NO_x$ pollution will decrease at a similar rate from 1990 levels. The prospective analysis Post-CAAA scenario shows a 16 percent drop by the year 2010, while the GCVTC estimates a 17 percent reduction.

$SO_2$ projections for the two studies are not characterized by similar percentage changes in emissions. In fact, under the Post-CAAA scenario the prospective analysis estimates an increase in $SO_2$ emission from 1990 to 2010 of about 15 percent, while the GCVTC study shows roughly an 11 percent decrease over this same time period. The reason for this disparity is that only the GCVTC emissions forecasts take into account specific modernization plans for the Kennecott-Utah Copper Corporation which are predicted to lower future $SO_2$ emissions in the West by approximately 30 thousand tons per year (tpy), as well as, a regional electric utility cut of roughly 80 thousand tpy. Together, these anticipated reductions account for the bulk of the difference between the two studies future year estimates.

Emissions figures for $PM_{2.5}$ are the source of the largest disparity between the prospective analysis and the GCVTC study. In general, emissions estimates are roughly 40 percent lower in the former than in the latter. This is due to a difference in the inventories used to develop the 1990 base year $PM_{2.5}$ estimates. While the more recent prospective analysis relied on NPI data that was updated to incorporate NET estimates that reflected revisions to $PM_{2.5}$ emissions factors for fugitive dust, the GCVTC study was conducted prior to the lowering of these factors. As a result, the $PM_{2.5}$ estimates in the GCVTC study are considerably higher than those in the prospective analysis. The percent change in $PM_{2.5}$ emissions from 1990 to 2010, however, is similar in the two studies (approximately 10 percent in prospective analysis and approximately 13 percent in GCVTC study).

**Table A-4**
**Comparison of Emissions: Prospective Analysis and GCVTC Stud**

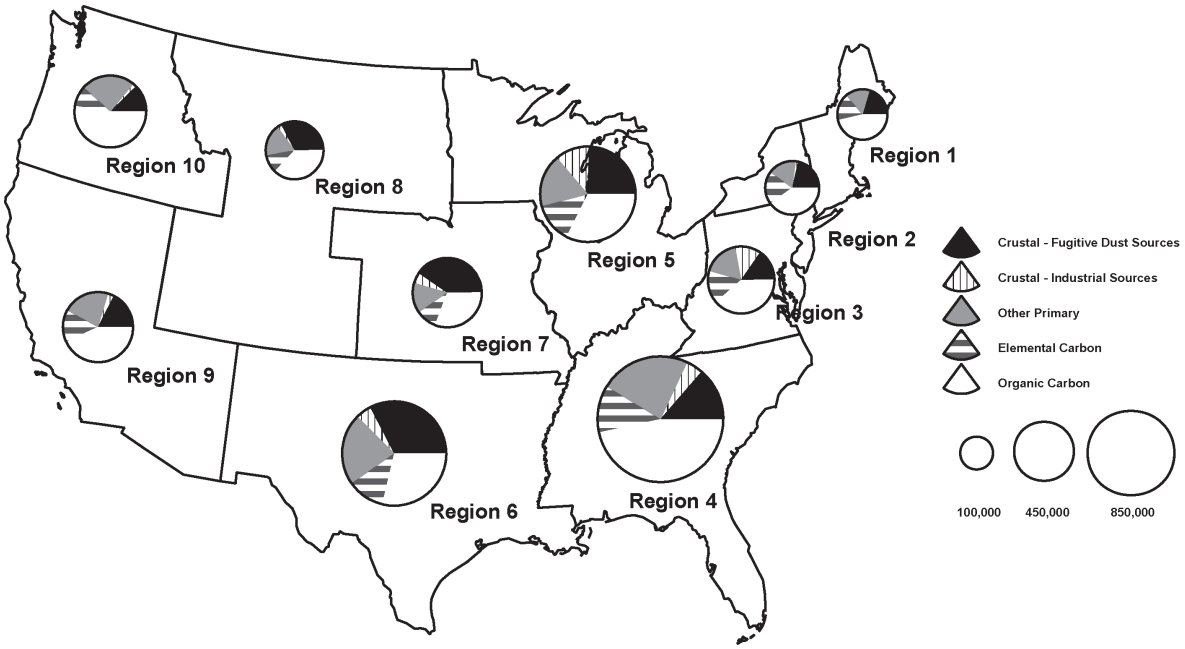
| | 1990 | | 2000 | | 2010 | |
|---|---|---|---|---|---|---|
| | Post-CAA | GCVTC | Post-CAA | GCVTC | Post-CAA | GCVTC |
| $NO_x$ | 3,303,536 | 3,058,221 | 2,938,833 | 2,596,409 | 2,784,580 | 2,532,855 |
| $SO_2$ | 1,245,439 | 1,094,928 | 1,326,546 | 944,689 | 1,434,470 | 970,762 |
| $PM_{2.5}$ | 1,701,869 | 2,412,177 | 1,759,434 | 2,535,829 | 1,864,656 | 2,730,304 |

NOTE: The figures in this table represent the total annual emissions (tons per year) for the Western States: Arizona, California, Colorado, Idaho, Nevada, New Mexico, Oregon, Utah, and Wyoming.

## *Prospective Analysis $PM_{2.5}$ Emissions Estimates and Observed Data*

The 1997 National Air Quality and Emissions Trends Report provides a summary of $PM_{2.5}$ concentration speciation data. This report shows the relative contribution of the major PM emissions source components (crustal material, carbonaceous particles, nitrate, and sulfate) to ambient $PM_{2.5}$ concentrations in urban and nonurban areas throughout the U.S. Comparison of primary $PM_{2.5}$ emissions estimates generated for this analysis with the observed concentration data presented in the 1997 report indicates that the ratio in the prospective study of crustal material to primary carbonaceous particles is high. At least part of this apparent overestimation

**Figure A-6**
**1990 Primary PM2.5 Emissions by EPA Region (tons/year)**



of crustal material and underestimation of carbonaceous particulates, however, is due to the fact that much of the emitted crustal material quickly settles and does not have a quantifiable impact on ambient air quality. In this analysis, we apply a factor of 0.2 to crustal emissions to estimate the fraction of crustal $PM_{2.5}$ that makes its way into the "mixed layer" of the atmosphere and influences pollutant concentrations. Figure A-6 displays the breakout of primary $PM_{2.5}$ into its adjusted crustal and carbonaceous (elemental carbon (EC) and organic carbon (OC)) components. The figure divides crustal material into two subcategories based on the source of the material (fugitive dust or industrial sources) and also shows the portion of primary $PM_{2.5}$ that is neither crustal nor carbonaceous. The ratios, one for each EPA Region, of adjusted crustal material to primary carbonaceous particles presented in Figure A-6 are in line with the observed $PM_{2.5}$ concentration data presented in the 1997 report.

## Industrial Point Sources

This section addresses industrial sector emitters – boilers and processes – that are large enough to be included in the 1990 emissions data base as individual point sources. In most cases, these are facilities that emit more than 100 tons per year of at least one criteria air pollutant. For industrial point source VOC and $NO_x$ emitters, trend analysis using the Emission Reduction and Cost Analysis Model (ERCAM) was conducted to project emissions for the years 2000 and 2010 (Pechan, 1994a; 1994b). The same procedures employed in the VOC and $NO_x$ projections were also used in developing CO, $SO_2$, and $PM_{10}$ estimates.

### *Overview of Approach*

In order to estimate the combined effects of activity growth and CAA controls on industrial point sources, the base year 1990 point source inventory was projected to 2000 and 2010 using growth factors from the Bureau of Economic Analysis (BEA), and CAA control assumptions. In its guidance for projecting emissions by combining growth and control effects, EPA identifies the following two options:

1. Aggregating all base year emissions and control information at the county level and performing all projections on that basis; or

2. Retaining source-specific information in the base year inventory and performing projections on a source-by-source basis (EPA, 1991a).

The second of these two approaches was selected, and future year emissions were projected by multiplying source-specific base year emissions by a corresponding growth factor and control factor. The decision to follow this option was based on the need to use source-level emissions estimates as the input for the air quality modeling phase of the prospective analysis.

The growth factors used in this analysis for projecting industrial point source emissions are from 1995 BEA industry-level Gross State Product (GSP) and population projections by State (BEA, 1995). ERCAM was used to model VOC and $NO_x$ emissions under each of the control scenarios. The basic approach for projecting emissions in ERCAM is as follows:

$$EMIS_Y = EMIS_{90} * GFAC_Y * \left[\frac{1 - (RE_Y * CE_Y)}{1 - (RE_{90} * CE_{90})}\right]$$

where:

| | | |
|---|---|---|
| $EMIS_Y$ | = | Emissions in projection year y |
| $EMIS_{90}$ | = | 1990 base year emissions |
| $GFAC_Y$ | = | Growth factor for projection year y |
| $RE_Y$ | = | Future year rule effectiveness (RE) |
| $CE_Y$ | = | Future year control efficiency |
| $RE_{90}$ | = | Base year (1990) RE |
| $CE_{90}$ | = | Base year (1990) control efficiency |

In cases where the future year control level ($RE_Y$ * $CE_Y$) is less stringent than the base year control level, 1990 base year control levels are retained (i.e., $RE_Y$ and $CE_Y$ equal 1990 levels in the emission projection algorithm).

## Base Year Emissions

The base year 1990 point source emission inventory for the prospective analysis is Version 3 of the NPI, originally developed in 1994 as a component of the Office of Policy, Planning and Evaluation (OPPE)'s "National Particulate Matter Study" (Pechan, 1994c; Pechan, 1995a). The NPI is a 1990 air emissions inventory for the United States (excluding Alaska and Hawaii). This data base contains plant and process level emissions for each of the criteria pollutants examined in this analysis.

Industrial point source emissions in the NPI were estimated using emission estimates from the 1985 National Acid Precipitation Assessment Program (NAPAP) Inventory projected to 1990 using BEA industrial sector earnings. Emission estimates for 1985 were projected to 1990 based on the State-level growth in earnings by industry (2-digit Standard Industrial Classification (SIC) code). Each record in the point source inventory was matched with the BEA earnings data based on the State and the 2-digit SIC code.

The industrial sector 1990 emission estimation procedures do not account for technological improvements since 1985 that may have lowered emissions per unit of production/output, nor do they account for emission controls that were added during this period. As a result, the base year emissions estimates, if biased, may overstate industrial point source emissions for 1990. In the Western States, with the incorporation of the GCVTC estimates, the 1990 emissions baseline for this region of the country may be less biased. The reason for this is that the GCVTC inventory is based upon State-supplied emissions information covering one of the years from 1990 to 1992. These more recent State reports reflect the effects of technological improvements and emission controls of the latter half of the 1980's that the NPI baseline inventory does not capture.

The NAPAP Inventory does not contain $PM_{10}$ and $PM_{2.5}$ emissions estimates. As a result, the annual $PM_{10}$ and $PM_{2.5}$ emissions figures in the NPI were calculated from 1985 total suspended particulate (TSP) emissions. These 1985 TSP estimates were projected to 1990 using BEA data and emissions estimates from each point source in the NAPAP Inventory (excluding steam electric utilities). What portion of 1990 TSP emissions was $PM_{10}$ and what portion was $PM_{2.5}$ was then determined. In order to make this determination, however, the 1990 TSP estimates first had to be adjusted to eliminate the effect of particulate controls, because in order to estimate particle size distribution using EPA's *Compilation of Emission Factors* (EPA, 1995), uncontrolled source data were required. Once this adjustment was made, $PM_{10}$ and $PM_{2.5}$ emissions were calculated by applying a Source Classification Code (SCC)-specific particle size distribution factor to the 1990 "uncontrolled" TSP emissions estimates. Then, the effects of primary and secondary controls on the two pollutants were estimated and base year $PM_{10}$ and $PM_{2.5}$ emissions were calculated.

## Growth Projections

The base year 1990 point source emission inventory was projected to 2000 and 2010 to determine the effects of Pre-CAAA and Post-CAAA controls on future year emission levels. Point source emissions growth is based on 1995 BEA industry GSP and population projections by State (BEA, 1995). EPA guidance for projecting emissions (EPA, 1991a) lists the following economic variables (in order of preference) for projecting emissions:

- Product output;
- Value added;
- Earnings; and
- Employment.

In the absence of product output projections, EPA guidance recommends value added projections. *Value*

*added* is the difference between the value of a firm's output and the inputs it purchases from other firms. BEA GSP projections represent a measure of value added, and are a fuller measure of growth than BEA's earnings projections because earnings represents only one component of GSP. GSP measures reflect the value added to revenue from selling a product minus the amounts paid for inputs from other firms. By incorporating inputs to production, GSP reflects future changes in production processes, efficiency, and technological changes. A comparison of BEA's 1995 GSP projections and BEA's 1990 earnings projections indicates that GSP growth factors are slightly higher than the earnings data. This is more often true for capital-intensive industries (e.g., manufacturing) than for labor-intensive industries (e.g., services). Components of GSP include payments to capital. This is an important distinction to make because it implicitly reflects the effect of factor substitution in production. As discussed in EPA's projections guidance, factor substitution should be included in growth projections, making value added data preferable to earnings data for projecting emissions.

For reasons mentioned above, the 1995 BEA industry GSP and population projections by State (BEA, 1995) were selected as the best available growth factors for projecting 1990 emissions to 2000 and 2010 for the prospective analysis. BEA's GSP estimates are broken down by industry sector (2-digit SIC codes) and State. For each record in the industrial point source component of the NPI, a link was established between the State code, the SIC code field, and the BEA GSP growth factors. Then projected future year emissions for each point source record were calculated by multiplying the 1990 emissions by the corresponding BEA growth factor.

BEA GSP growth factors were used to project industrial point source emissions for the prospective analysis because BEA data provide growth by industry on a State-level in a form that provides a straightforward link to the industrial point source component of the NPI (the SIC code field). GSP growth factors also comply with EPA's guidance for projecting emissions, since they represent a measure of *value added*. In the development of the BEA GSP projections, BEA ensures consistency with national projections of population from the Bureau of the Census, of the labor force from the Bureau of Labor Statistics (BLS), of the unemployment rate from the Congressional Budget Office, and of mining output from the Department of Energy (DOE). It is important to note, however, that BEA's projections are based on the assumption that past economic relationships will continue and that no major policy changes will occur. The growth factors used in this analysis therefore do not explicitly reflect potential future changes in economic conditions or technologies except those that may be reflected in historical industry trends.

## *Control Scenarios*

The Pre-CAAA scenario represents expected point source emissions after the application of BEA GSP growth factors, with 1990 levels of control efficiency retained. The Post-CAAA scenario incorporates control efficiencies based on measures mandated by the CAAA. The control assumptions associated with each of the two scenarios are described separately below.

### Pre-CAAA Scenario

The Pre-CAAA scenario assumes the continuation of 1990 control efficiencies for all emitters. Point source emissions of VOC, $NO_x$, CO, $SO_2$, $PM_{10}$, $PM_{2.5}$, and $NH_3$ under the Pre-CAAA scenario were projected to 2000 and 2010 by applying BEA GSP growth factors to the base year 1990 point source emission inventory based on the State and SIC code data fields.

### Post-CAAA Scenario

The Post-CAAA scenario represents point source emissions after the application of BEA GSP growth factors, and the effects of controls implemented under the CAAA. CAAA provisions affecting industrial point sources include:

- Title III 2-year and 4-year MACT standards (VOC only);
- VOC and $NO_x$ RACT requirements in ozone NAAs;
- New control technique guidelines (CTGs);
- A 0.15 lbs/MMBtu $NO_x$ cap on fuel combustors of 250 MMBtu per hour or above across the OTAG 37-State region; and
- Ozone rate-of-progress (ROP) requirements.

The control assumptions used to estimate emissions from industrial point sources under the Post-CAAA scenario differ by pollutant. For each of the pollutants, the corresponding controls assumed to effect emissions in 2000 and 2010 are briefly described in Table A-5.

**Table A-5**
**Industrial Point Source Control Assumptions For The Post-CAAA Scenario**

| Pollutant | Point Source Control Measures |
|---|---|
| VOC | Point source control measures for VOC include RACT, new CTGs, and Title III MACT controls. Title III MACT controls are generally as stringent, or more stringent, than RACT controls and are thus the dominant control option for many source categories. An 80 percent RE is assumed for all control measures. |
| $NO_x$ | Industrial point source $NO_x$ controls include $NO_x$ RACT, OTAG level 2 $NO_x$ controls, and a 0.15 lbs/MMBtu cap on fuel combustors of 250 MMBtu per hour and above across the OTAG 37-State region. Major stationary source $NO_x$ emitters in marginal and above NAAs and in the northeast Ozone Transport Region (OTR) are required to install RACT-level controls under the ozone nonattainment-related provisions of Title I. RACT control levels are specified by each State and are based on an assumed rule effectiveness (RE) of 80 percent. |
| CO | No new CO controls were modeled for the Post-CAAA scenario, although some CO NAAs may have adopted controls for specific point sources within NAAs. |
| $SO_2$ | $SO_2$ nonattainment provisions of the CAAA do not specify any mandatory controls for $SO_2$ emitters, though an emission cap of 5.6 million tons of $SO_2$ per year was set by the CAAA for industrial sources. |
| $PM_{10}$ and $PM_{2.5}$ | Possible control initiatives for particulates under the CAA would result from the Title I provisions related to $PM_{10}$ nonattainment. Because the controls are specific to each area, the CAAA $PM_{10}$ emissions for industrial point sources were assumed to be equivalent to the Pre-CAAA emissions. Point source $PM_{2.5}$ emissions were also assumed to be unaffected by CAAA provisions. |
| $NH_3$ | Point source $NH_3$ emissions were assumed to be unaffected by CAAA provisions. |

## Emission Summary

National point source emission projections for 2000 and 2010 for each of the pollutants are shown in Table A-6. VOC emissions from industrial point sources in these two years are primarily affected by new National Emission Standards for Hazardous Air Pollutants (NESHAPs) under Title III and new CTGs for achieving further VOC emission reductions in ozone NAAs. Source categories with significant VOC emission reductions in this sector include chemical and allied product manufacturing, petroleum refineries, solvent utilization, and petroleum storage.

$NO_x$ emission reductions from industrial fuel combustors result mainly from the implementation of RACT for major stationary sources in ozone NAAs and addition of further $NO_x$ controls for large fuel combustors (larger than 250 MMBtu per hour) throughout the 37 OTAG States. The OTAG stationary source $NO_x$ strategy included in this analysis assumes that large fuel combustors meet a 0.15 lbs/MMBtu $NO_x$ emission limit. With these and other standards, CAA $NO_x$ emission benefits in 2010 for this sector are projected to be more than one million tons.

Industrial point source emission projections for the other criteria pollutants (CO, $SO_2$, $PM_{10}$, $PM_{2.5}$, and $NH_3$) show no appreciable effect of the CAAA on future year emissions. Stationary source CO emitters could be subjected to further control requirements as part of individual area CO State Implementation Plans (SIPs), but this is unlikely. Similarly for $PM_{10}$, there may be industrial source $PM_{10}$ emission reductions observed through application of best available control measures in some $PM_{10}$ attainment plans, but these potential reductions are not captured in this analysis.

**Table A-6**
**Industrial Point Source Emission Summaries by Pollutant For 1990, 2000, and 2010***
**(thousand tons per year)**

| Pollutant/Source Category | 1990 | 2000 Pre-CAAA | 2000 Post-CAAA | 2010 Pre-CAAA | 2010 Post-CAAA |
|---|---|---|---|---|---|
| **VOC** | | | | | |
| Fuel Comb. Industrial | 126.1 | 135.9 | 134.9 | 153.7 | 157.7 |
| Fuel Comb. Other | 10.3 | 12.3 | 12.3 | 14.4 | 14.9 |
| Chemical & Allied Product Mfg | 1,066.2 | 1,101.4 | 878.9 | 1,266.6 | 1,007.4 |
| Metals Processing | 72.5 | 85.8 | 83.1 | 90.3 | 87.2 |
| Petroleum & Related Industries | 238.2 | 243.1 | 160.2 | 269.5 | 166.7 |
| Other Industrial Processes | 327.0 | 372.5 | 365.3 | 418.9 | 417.3 |
| Solvent Utilization | 1,126.2 | 1,256.7 | 1,036.0 | 1,394.1 | 1,120.0 |
| Storage & Transport | 490.1 | 540.9 | 405.7 | 617.0 | 467.5 |
| Waste Disposal & Recycling | 9.7 | 9.9 | 9.9 | 11.1 | 11.7 |
| Miscellaneous | 0.3 | 0.4 | 0.4 | 0.6 | 0.6 |
| TOTAL | 3,466.6 | 3,758.9 | 3,086.7 | 4,236.2 | 3,450.9 |
| **$NO_x$** | | | | | |
| Fuel Comb. Industrial | 1,955.8 | 2,181.8 | 1,213.5 | 2,464.5 | 1,255.9 |
| Fuel Comb. Other | 103.9 | 122.9 | 77.4 | 141.1 | 84.1 |
| Chemical & Allied Product Mfg | 275.4 | 281.5 | 263.0 | 322.2 | 300.0 |
| Metals Processing | 81.0 | 103.0 | 98.9 | 111.3 | 104.7 |
| Petroleum & Related Industries | 99.9 | 104.1 | 104.1 | 111.1 | 108.1 |
| Other Industrial Processes | 308.0 | 350.9 | 277.6 | 394.6 | 302.8 |
| Solvent Utilization | 2.5 | 2.9 | 2.9 | 3.2 | 3.1 |
| Storage & Transport | 2.4 | 2.6 | 2.6 | 3.0 | 3.0 |
| Waste Disposal & Recycling | 20.7 | 23.5 | 20.4 | 26.9 | 22.9 |
| TOTAL | 2,849.7 | 3,173.3 | 2,060.4 | 3,577.9 | 2,184.5 |
| **CO** | | | | | |
| Fuel Comb. Industrial | 529.1 | 586.8 | 586.8 | 656.7 | 656.7 |
| Fuel Comb. Other | 96.8 | 118.4 | 118.4 | 140.1 | 140.1 |
| Chemical & Allied Product Mfg | 1,923.4 | 1,957.2 | 1,957.2 | 2,233.0 | 2,233.0 |
| Metals Processing | 2,106.3 | 2,418.1 | 2,418.1 | 2,486.1 | 2,486.1 |
| Petroleum & Related Industries | 436.3 | 475.1 | 475.1 | 545.9 | 545.9 |
| Other Industrial Processes | 754.0 | 947.1 | 947.1 | 1,134.1 | 1,134.1 |
| Solvent Utilization | 2.5 | 2.8 | 2.8 | 3.2 | 3.2 |
| Storage & Transport | 54.8 | 51.5 | 51.5 | 58.7 | 58.7 |
| Waste Disposal & Recycling | 96.7 | 106.8 | 106.8 | 118.7 | 118.7 |
| TOTAL | 5,999.7 | 6,663.9 | 6,663.9 | 7,376.6 | 7,376.6 |

| Pollutant/Source Category | 1990 | 2000 Pre-CAAA | 2000 Post-CAAA | 2010 Pre-CAAA | 2010 Post-CAAA |
|---|---|---|---|---|---|
| **$SO_2$** | | | | | |
| Fuel Comb. Industrial | 2,482.2 | 2,861.9 | 2,861.9 | 3,262.0 | 3,262.0 |
| Fuel Comb. Other | 202.4 | 243.7 | 243.7 | 282.9 | 282.9 |
| Chemical & Allied Product Mfg | 440.1 | 486.6 | 486.6 | 546.4 | 546.4 |
| Metals Processing | 664.7 | 808.9 | 808.9 | 857.1 | 857.1 |
| Petroleum & Related Industries | 434.8 | 449.5 | 449.5 | 489.3 | 489.3 |
| Other Industrial Processes | 393.6 | 456.4 | 456.4 | 522.6 | 522.6 |
| Solvent Utilization | 0.8 | 0.9 | 0.9 | 1.0 | 1.0 |
| Storage & Transport | 4.6 | 5.4 | 5.4 | 6.4 | 6.4 |
| Waste Disposal & Recycling | 21.0 | 22.8 | 22.8 | 26.1 | 26.1 |
| TOTAL | 4,644.2 | 5,336.2 | 5,336.2 | 5,993.9 | 5,993.9 |
| **$PM_{10}$** | | | | | |
| Fuel Comb. Industrial | 221.1 | 245.2 | 245.2 | 275.8 | 275.8 |
| Fuel Comb. Other | 16.6 | 19.8 | 19.8 | 23.0 | 23.0 |
| Chemical & Allied Product Mfg | 62.5 | 65.4 | 65.4 | 74.2 | 74.2 |
| Metals Processing | 137.9 | 167.2 | 167.2 | 179.4 | 179.4 |
| Petroleum & Related Industries | 28.9 | 31.7 | 31.7 | 36.0 | 36.0 |
| Other Industrial Processes | 374.3 | 427.3 | 427.3 | 485.5 | 485.5 |
| Solvent Utilization | 2.1 | 2.6 | 2.6 | 3.0 | 3.0 |
| Storage & Transport | 64.4 | 73.8 | 73.8 | 83.6 | 83.6 |
| Waste Disposal & Recycling | 8.0 | 8.7 | 8.7 | 9.7 | 9.7 |
| Miscellaneous | 10.7 | 13.7 | 13.7 | 16.9 | 16.9 |
| TOTAL | 926.4 | 1,055.3 | 1,055.3 | 1,186.9 | 1,186.9 |
| **$PM_{2.5}$** | | | | | |
| Fuel Comb. Industrial | 162.0 | 178.1 | 178.1 | 200.0 | 200.0 |
| Fuel Comb. Other | 8.2 | 9.7 | 9.7 | 11.1 | 11.1 |
| Chemical & Allied Product Mfg | 42.7 | 45.7 | 45.7 | 52.0 | 52.0 |
| Metals Processing | 96.3 | 116.7 | 116.7 | 124.4 | 124.4 |
| Petroleum & Related Industries | 19.5 | 21.3 | 21.3 | 24.1 | 24.1 |
| Other Industrial Processes | 224.3 | 257.9 | 257.9 | 294.7 | 294.7 |
| Solvent Utilization | 1.8 | 2.2 | 2.2 | 2.5 | 2.5 |
| Storage & Transport | 26.5 | 30.5 | 30.5 | 34.4 | 34.4 |
| Waste Disposal & Recycling | 6.7 | 7.2 | 7.2 | 8.0 | 8.0 |
| Miscellaneous | 1.6 | 2.1 | 2.1 | 2.6 | 2.6 |
| TOTAL | 589.5 | 671.5 | 671.5 | 754.0 | 754.0 |

| Pollutant/Source Category | 1990 | 2000 Pre-CAAA | 2000 Post-CAAA | 2010 Pre-CAAA | 2010 Post-CAAA |
|---|---|---|---|---|---|
| **$NH_3$** | | | | | |
| Fuel Comb. Industrial | 10.0 | 10.3 | 10.3 | 11.3 | 11.3 |
| Fuel Comb. Other | 0.3 | 0.3 | 0.3 | 0.3 | 0.3 |
| Chemical & Allied Product Mfg | 182.6 | 202.6 | 202.6 | 231.6 | 231.6 |
| Metals Processing | 5.9 | 6.8 | 6.8 | 7.0 | 7.0 |
| Petroleum & Related Industries | 42.8 | 48.7 | 48.7 | 55.1 | 55.1 |
| Other Industrial Processes | 2.1 | 2.1 | 2.1 | 2.1 | 2.1 |
| TOTAL | 243.6 | 270.8 | 270.8 | 307.5 | 307.5 |

* The totals reflect emissions for the 48 contiguous States, excluding Alaska and Hawaii. Totals may not add due to rounding.

## Utilities

EPA used the IPM to estimate future year heat input, $SO_2$ and $NO_x$ emissions, fuel type, and optimal control techniques for each current and planned electric utility unit.[5] The IPM modeling inputs, outputs, and key assumptions are discussed in more detail in EPA 1997b. This section focuses on the steps used to supplement these projections by adding emissions of VOC, CO, $PM_{10}$, $PM_{2.5}$, and $NH_3$, as well as adding data elements needed for air quality modeling (location and stack parameters).

### *Overview of Approach*

For the prospective analysis, EPA matched each unit in the IPM file to the 1990 NPI (Pechan, 1994c; Pechan, 1995a) based on the Office of the Regulatory Information System (ORIS) plant and boiler code. For units that were matched, stack parameters and location coordinates were taken directly from the NPI. VOC, CO, $PM_{10}$, and $PM_{2.5}$ emissions were calculated using AP-42 emission rates (standard EPA emission factors that are developed from stack tests and engineering calculations) and control efficiencies as reported in the NPI. $NH_3$ emissions were calculated for ammonia slippage where boilers were forecast to install selective catalytic reduction (SCR) as the control technique to reduce $NO_x$ emissions.

### *Base Year Emissions*

The base year emission inventory used in the air quality modeling portion of the Section 812 prospective analysis is Version 3 of the NPI. The utility portion of this inventory, which covers fossil-fuel fired steam electric generating boilers, was developed from DOE Form EIA-767 (the Steam-Electric Plant Operation and Design Report) fuel use data combined with AP-42 emission factors and emission limits from EIA-767. The NPI also includes gas turbines and internal combustion engines to the extent that these were included in the 1985 NAPAP.

The IPM uses a different modeling set as input for emission projections. This set is consistent with the NPI in that boilers included in the NPI are also in the IPM modeling set. The IPM data set, however, also includes combustion turbines (more than those included in the NPI) and non-utility generators. Some of these units may be included in the NPI as part of the industrial point source inventory or area source fuel combustion (and would thus be double-counted in the projections) while others may be missing from the NPI data set. It should be noted that in projection year 2000, under the Pre-CAAA scenario, these units account for just over 1 percent of total utility $NO_x$ emissions and only 0.1 percent of total utility $SO_2$ emissions. Therefore, the potential exclusion of the units from the 1990 base year data set and the potential double-counting in the projection year is expected to have minimal effect on air quality, benefits, and cost modeling.

[5] The IPM was constructed and is maintained by ICF, an EPA contractor.

## Control Scenarios

The Pre-CAAA and Post-CAAA scenario $NO_x$ and $SO_2$ emissions were modeled using IPM (EPA, 1997b). Heat input by unit under each projection year and scenario was also derived from IPM results. Default emission factors were applied to the unit-specific heat input to calculate VOC, CO, $PM_{10}$, and $PM_{2.5}$ emissions. For these pollutants, the Pre-CAAA and Post-CAAA emission rates were assumed to be the same. Any differences in emissions between these scenarios are due to shifts in operation between units or fuel changes (including ash content of the coals as well as switching to natural gas) predicted by IPM. Differences in $NH_3$ emissions between the scenarios is a fraction of added SCR controls to reduce $NO_x$ under the Post-CAAA scenario.

## Emission Summary

Table A-7 is a summary of utility emissions by unit and fuel type. Oil- and gas-fired units have been grouped together, because information on the division of fuel for boilers burning oil and gas was not contained in the unit-level file developed from IPM. Utility $SO_2$ and $NO_x$ emissions are affected most by the CAAA. Differences between Pre-CAAA and Post-CAAA $NO_x$ emissions result from a combination of Title IV - Acid Rain regulations, and nonattainment provisions that require $NO_x$ RACT controls for major stationary sources in ozone NAAs. The anticipated effect of the OTC MOU and a regional $NO_x$ trading program on stationary source $NO_x$ emissions in the eastern portion of the United States also influence the Post-CAAA utility $NO_x$ emissions estimates (OTC, 1994). In 2010, the difference between Pre-CAAA and Post-CAAA utility $NO_x$ is about 5 million tons.

$SO_2$ emission reductions attributable to the CAA is the result of the Title IV - Acid Rain control program. Through this program, annual emissions of $SO_2$ are to be reduced by 10 million tons from 1980 levels through a market-based allowance system. Differences between Pre-CAAA and Post-CAAA $SO_2$ estimates for coal-fired units reflect flue gas desulfurization installations and some switching to lower sulfur coal in the Post-CAAA case. $SO_2$ emissions from oil/gas units are actually lower in the Pre-CAAA scenario because a higher percentage of units would have been expected to burn coal with less stringent air pollution emission limits. The 2010 Pre-CAAA estimates show no significant oil use by utilities.

**Table A-7**
**Utility Emission Summary***
**(thousand tons per year)**

| Pollutant/Source Category | | 1990 | 2000 Pre-CAAA | 2000 Post-CAAA | 2010 Pre-CAAA | 2010 Post-CAAA |
|---|---|---|---|---|---|---|
| **VOC** | | | | | | |
| | Coal | 27.1 | 23.6 | 23.1 | 26.3 | 24.7 |
| | Gas/Oil/Other | 7.8 | 1.8 | 1.9 | 1.5 | 1.7 |
| | Internal Combustion | 1.9 | 5.6 | 6.1 | 21.2 | 23.5 |
| | TOTAL | 36.8 | 31.0 | 31.1 | 49.0 | 49.9 |
| **$NO_x$** | | | | | | |
| | Coal | 6,689.5 | 7,895.7 | 3,779.0 | 8,700.3 | 3,610.0 |
| | Gas/Oil/Other | 679.1 | 324.1 | 216.0 | 220.0 | 72.6 |
| | Internal Combustion | 57.1 | 97.3 | 82.1 | 134.4 | 83.7 |
| | TOTAL | 7,425.7 | 8,317.1 | 4,077.1 | 9,054.7 | 3,766.3 |
| **CO** | | | | | | |
| | Coal | 232.6 | 191.8 | 188.7 | 215.3 | 202.5 |
| | Gas/Oil/Other | 81.9 | 48.0 | 49.3 | 44.8 | 45.9 |
| | Internal Combustion | 14.7 | 50.7 | 55.4 | 193.6 | 214.8 |
| | TOTAL | 329.2 | 290.5 | 293.4 | 453.7 | 463.2 |
| **$SO_2$** | | | | | | |
| | Coal | 15,221.9 | 16,111.3 | 10,315.0 | 17,696.0 | 9,776.6 |
| | Gas/Oil/Other | 611.9 | 44.1 | 175.5 | 0.0 | 84.2 |
| | Internal Combustion | 30.7 | 0.0 | 0.0 | 0.0 | 0.0 |
| | TOTAL | 15,897.5 | 16,155.4 | 10,490.5 | 17,696.0 | 9,860.8 |
| **$PM_{10}$** | | | | | | |
| | Coal | 268.4 | 244.7 | 245.1 | 281.1 | 249.0 |
| | Gas/Oil/Other | 10.6 | 1.5 | 2.5 | 1.0 | 1.6 |
| | Internal Combustion | 4.1 | 6.1 | 6.6 | 23.4 | 26.0 |
| | TOTAL | 283.1 | 252.3 | 254.2 | 305.5 | 276.6 |
| **$PM_{2.5}$** | | | | | | |
| | Coal | 99.2 | 82.9 | 82.3 | 97.0 | 83.7 |
| | Gas/Oil/Other | 5.9 | 1.3 | 2.4 | 1.0 | 1.6 |
| | Internal Combustion | 3.7 | 6.0 | 6.6 | 23.3 | 25.8 |
| | TOTAL | 108.8 | 90.2 | 91.3 | 121.3 | 111.1 |
| **$NH_3$** | | | | | | |
| | Coal | 0.0 | 0.0 | 33.3 | 0.0 | 221.9 |
| | Gas/Oil/Other | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| | Internal Combustion | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| | TOTAL | 0.0 | 0.0 | 33.3 | 0.0 | 221.9 |

* The totals reflect emissions for the 48 contiguous States, excluding Alaska and Hawaii.

## Nonroad Engines/Vehicles

The nonroad engines/vehicles sector includes all transportation sources that are not counted as highway vehicles. Thus, this sector includes marine vessels, railroads, aircraft, and nonroad internal combustion engines and vehicles. Nonroad engines are significant emitters of $NO_x$, $PM_{10}$, and VOC. Diesel engines account for most of the $NO_x$ and $PM_{10}$ emissions, while gasoline engines emit most of the VOC. This section contains summaries of 1990 emissions from major source categories in the nonroad engine/vehicle sector. The growth factors and control efficiencies used to project emissions to 2000 and 2010 under the two control scenarios are also described.

### Overview of Approach

Nonroad VOC and $NO_x$ emissions were projected using ERCAM; similar modeling techniques were used for the remaining pollutants. The algorithm for projecting nonroad emissions is:

$$EMIS_Y = EMIS_{90} * GFAC_Y * \left[1 - CE_Y * PE_Y\right]$$

where:

| | | |
|---|---|---|
| $EMIS_Y$ | = | emissions in projection year y |
| $EMIS_{90}$ | = | 1990 emissions |
| GFAC | = | growth factor for projection year y |
| $CE_Y$ | = | control efficiency for projection year y |
| $PE_Y$ | = | penetration rate for projection year y |

The control efficiency is a function of the percentage reduction or decrease in emission rate expected through new engine standards and the fraction of emissions covered through fleet turnover. The penetration rate accounts for the fraction of emissions from affected engine types (generally resulting from horsepower (hp) cutoffs) in a broad engine category (e.g., construction).

Growth factors applied are based on the 1995 BEA GSP projections by State and industry and population projections. The 1990 base year emissions are from Version 3 of the NPI. Under the Pre-CAAA scenario, no changes in engine standards are modeled (future year emission rates are assumed to be equivalent to 1990 rates). Under the Post-CAAA scenario, Federal nonroad engine standards are incorporated. All modeling is at the county and SCC level to retain necessary details for cost and air quality modeling.

### Base Year Emissions

The 1990 emission estimates for nonroad vehicles are from the 1990 NPI. The emissions in the NPI from sources in the nonroad engines/vehicles sector are based on one of the following sources: (1) a nonroad emission inventory compiled by EPA's OMS (EPA, 1991b); or (2) the 1985 NAPAP Area Source Emissions Inventory. EPA's OMS inventories provided the majority of criteria pollutant emissions for the base year 1990 inventory, accounting for nearly 90 percent of VOC emissions and nearly 60 percent of $NO_x$ emissions. The remaining emissions for the nonroad engines/vehicles sector are based on the NAPAP emissions inventory.

### Growth Projections

The 1990 estimates from the NPI for the nonroad engine/vehicle sector are projected to 2000 and 2010 to estimate the impact of CAAA controls on future year emission levels. For each major nonroad engine/vehicle category a growth surrogate is identified for estimating future emissions. Growth surrogates for nonroad engine/vehicle categories include 1995 BEA projections of population for recreational and lawn and garden equipment categories, and an appropriate GSP by SIC code estimate for all other categories. SIC codes are assigned to area source categories according to an assignment made for other EPA projects, such as the ozone and PM NAAQS cost analyses. This assignment of nonroad engine/vehicle categories to BEA indicators is shown in Table A-8.

The 1995 BEA GSP and population projections by State and industry were selected as the best available growth factors for projecting 1990 emissions to 2000 and 2010 for the prospective analysis (BEA, 1995). EPA's projection guidance recommends that area source emissions be projected using surrogate growth indicators such as BEA, or using local studies/surveys (EPA, 1991a). BEA provided a consistent set of projections by SIC code that could be easily applied to the 1990 nonroad engine/vehicle sector across all geographic regions.

## *Control Scenarios*

Emissions from engines used in nonroad equipment are a significant source of $NO_x$, VOC, and PM emissions. In some areas of the country, emissions from nonroad engines represent a third of the total mobile source $NO_x$ and VOC emissions and over two-thirds of the mobile source PM emissions.

### Pre-CAAA Scenario

The Pre-CAAA scenario incorporates the growth factors described above, and assumes that future year emission rates from nonroad engines remain the same as 1990 levels.

**Table A-8**
**BEA Growth Forecasts by Major Source Category:**
**Nonroad Engines/Vehicles**

| | | Annual Growth (% per year): | |
|---|---|---|---|
| **Major Category** | **BEA Growth Category*** | **1990-2000** | **1990-2010** |
| Nonroad Internal Combustion Engines and Vehicles: | | | |
| Airport Service Equipment | Transportation by air (SIC 45) | 5.8% | 5.5% |
| Construction Equipment | Construction (SIC 15, 16, and 17) | 0.8% | 1.0% |
| Farm Equipment | Farm (SIC 01) | 2.4% | 2.4% |
| Industrial Equipment | Total Manufacturing | 1.9% | 1.9% |
| Lawn & Garden Equipment | Population | 1.1% | 1.0% |
| Light Commercial Equipment | Total Manufacturing | 1.9% | 1.9% |
| Logging Equipment | Agricultural Services, Forestry, Fisheries (SIC 07, 08, 09) | 7.8% | 7.4% |
| Recreational Marine Vessels | Population | 1.1% | 1.0% |
| Recreational Vehicles | Population | 1.1% | 1.0% |
| Aircraft: | | | |
| Military | Federal, military | -1.2% | -0.4% |
| Commercial | Transportation by air (SIC 45) | 5.8% | 5.5% |
| Civil | Transportation by air (SIC 45) | 5.8% | 5.5% |
| Railroads | Railroad Transportation (SIC 40): Earnings | -1.5% | -0.9% |
| Commercial Marine Vessels | Water Transportation (SIC 44) | -0.5% | -0.2% |

*BEA growth category refers to GSP projections for each industry, unless otherwise specified.

## Post-CAAA Scenario

The CAAA specifically directed EPA to study the contribution of nonroad engines to urban air pollution, and to regulate them, if warranted. In 1991, EPA released a study that documented higher than expected emission levels across a broad spectrum of nonroad engines and equipment (EPA, 1991b). In response, EPA initiated several regulatory programs for nonroad engines. The impact of these programs is incorporated in the Post-CAAA scenario.

### *Emission Summary*

A summary of projected emissions by engine classification is shown in Table A-9. Future year VOC and $NO_x$ emissions are the pollutants most affected by the CAAA, as most of the new engine standards focus on controlling these ozone precursors. CO, $SO_2$, and $PM_{10}$ emissions under the Pre-CAAA scenario are nearly equal to emissions under the Post-CAAA scenario since CAAA controls only affect $NO_x$ and VOC emissions from the nonroad engine/vehicle sector. Effects in 2000 are modest because the new engine standards do not affect emissions until the mid-to-late 1990s. More dramatic differences are seen in 2010.

Gasoline-powered engines are the most significant nonroad VOC emitter, so most of the VOC emissions difference in this sector is the result of small spark ignition (SI) engine standards. Lawnmowers, for example, are affected by these new standards.

$NO_x$ emission benefits shown in Table A-9 for non-road engines result principally from compression ignition (CI) (diesel engine) standards. In 2010, difference between the Post-CAAA and Pre-CAAA nonroad diesel engine $NO_x$ emissions is almost 0.5 million tons. These engines are primarily used in construction equipment. Other off-highway $NO_x$ sources with lower emissions levels under the Post-CAAA scenario include railroads (diesel locomotives) and marine vessels. In contrast, a small $NO_x$ disbenefit is associated with non-road gasoline engines in the Post-CAAA scenario; this is because the small SI engine standard for hydrocarbons (HCs) is expected to increase $NO_x$ emissions.

**Table A-9**
**Nonroad National Emission Projections by Source Category***
**(thousand tons per year)**

| Pollutant/Source Category | | 1990 | 2000 Pre-CAAA | 2000 Post-CAAA | 2010 Pre-CAAA | 2010 Post-CAAA |
|---|---|---|---|---|---|---|
| **VOC** | | | | | | |
| | Nonroad Gasoline | 1,596.8 | 1,810.8 | 1,549.3 | 2,004.0 | 1,257.4 |
| | Nonroad Diesel | 185.0 | 225.3 | 225.3 | 261.6 | 261.6 |
| | Aircraft | 191.9 | 242.6 | 242.6 | 300.3 | 300.3 |
| | Marine Vessels | 36.0 | 33.6 | 33.6 | 34.4 | 34.4 |
| | Railroads | 44.2 | 37.2 | 37.2 | 35.5 | 35.4 |
| | TOTAL | 2,053.9 | 2,349.5 | 2,088.0 | 2,635.8 | 1,889.2 |
| **$NO_x$** | | | | | | |
| | Nonroad Gasoline | 176.0 | 205.4 | 220.5 | 237.0 | 269.8 |
| | Nonroad Diesel | 1,438.4 | 1,751.4 | 1,603.0 | 2,032.5 | 1,546.9 |
| | Aircraft | 139.7 | 194.8 | 194.8 | 249.2 | 249.2 |
| | Marine Vessels | 183.7 | 169.8 | 169.8 | 173.2 | 161.5 |
| | Railroads | 898.0 | 759.4 | 759.4 | 725.9 | 513.6 |
| | TOTAL | 2,835.8 | 3,080.9 | 2,947.5 | 3,417.8 | 2,740.9 |
| **CO** | | | | | | |
| | Nonroad Gasoline | 12,047.2 | 13,973.8 | 13,417.5 | 15,735.4 | 15,020.6 |
| | Nonroad Diesel | 781.9 | 948.6 | 948.6 | 1,097.3 | 1,097.3 |
| | Aircraft | 960.9 | 1,423.6 | 1,423.7 | 1,855.9 | 1,855.9 |
| | Marine Vessels | 58.0 | 54.1 | 54.1 | 55.5 | 55.5 |
| | Railroads | 121.8 | 102.8 | 102.8 | 98.2 | 98.2 |
| | TOTAL | 13,969.8 | 16,503.0 | 15,946.6 | 18,842.3 | 18,127.5 |
| **$SO_2$** | | | | | | |
| | Nonroad Gasoline | 3.2 | 3.6 | 3.6 | 4.1 | 4.1 |
| | Nonroad Diesel | 16.7 | 19.0 | 19.0 | 22.4 | 22.4 |
| | Aircraft | 8.0 | 10.9 | 10.9 | 13.8 | 13.8 |
| | Marine Vessels | 147.5 | 139.8 | 139.8 | 142.3 | 142.3 |
| | Railroads | 66.6 | 56.3 | 56.3 | 53.8 | 53.8 |
| | TOTAL | 242.1 | 229.6 | 229.6 | 236.5 | 236.5 |
| **$PM_{10}$** | | | | | | |
| | Nonroad Gasoline | 42.1 | 47.0 | 47.0 | 51.6 | 51.6 |
| | Nonroad Diesel | 185.6 | 227.3 | 227.3 | 262.6 | 187.1 |
| | Aircraft | 40.4 | 38.1 | 38.1 | 41.3 | 41.3 |
| | Marine Vessels | 24.2 | 22.7 | 22.7 | 23.3 | 23.3 |
| | Railroads | 44.0 | 37.3 | 37.3 | 35.7 | 33.1 |
| | TOTAL | 336.3 | 372.5 | 372.5 | 414.4 | 336.3 |

| Pollutant/Source Category | 1990 | 2000 Pre-CAAA | 2000 Post-CAAA | 2010 Pre-CAAA | 2010 Post-CAAA |
|---|---|---|---|---|---|
| **$PM_{2.5}$** | | | | | |
| Nonroad Gasoline | 35.0 | 39.2 | 39.2 | 42.9 | 42.9 |
| Nonroad Diesel | 170.8 | 209.1 | 209.1 | 241.6 | 172.1 |
| Aircraft | 28.5 | 26.9 | 26.9 | 29.1 | 29.1 |
| Marine Vessels | 17.8 | 16.6 | 16.6 | 17.0 | 17.0 |
| Railroads | 40.5 | 34.3 | 34.3 | 32.8 | 30.4 |
| TOTAL | 292.6 | 326.1 | 326.1 | 363.5 | 291.7 |
| **$NH_3$** | | | | | |
| Nonroad Gasoline | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Nonroad Diesel | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Aircraft | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Marine Vessels | 1.1 | 1.1 | 1.1 | 1.1 | 1.1 |
| Railroads | 1.8 | 1.5 | 1.5 | 1.4 | 1.4 |
| TOTAL | 2.9 | 2.6 | 2.6 | 2.5 | 2.5 |

* The totals reflect emissions for the 48 contiguous States, excluding Alaska and Hawaii.

## Motor Vehicles

Motor vehicles are a significant contributor of VOC, $NO_x$, and CO emissions. In 1990, motor vehicles contributed 30 percent of total VOC, 33 percent of total $NO_x$, and 66 percent of total CO emissions. The CAA includes provisions to reduce motor vehicles emissions in both Title I and Title II.

### *Overview of Approach*

The general procedure for calculating historic and projection year motor vehicle emissions is to multiply activity, in the form of VMT by pollutant specific emission factor estimates. ERCAM (Pechan, 1996) was used to project motor vehicle emissions for VOC, $NO_x$, and CO. Emission factors for these pollutants were generated using the EPA's motor vehicle emission factor model MOBILE5a (EPA, 1993a). $PM_{10}$, $PM_{2.5}$, and $SO_2$ emission factors were generated using another EPA motor vehicle emission factor model, PART5 (EPA, 1994). Emission factors for all pollutants are modeled using common assumptions about ambient temperatures and vehicle speeds at the State level. Control programs (I/M, reformulated gasoline) are specified at the county level. Temporally, emissions are calculated by month and summed to develop annual emission estimates.

### *Base Year Emissions*

Base year emissions are from Version 3 of the NPI. The NPI VMT, by county/SCC (i.e., vehicle type/functional roadway class), are based on data from the Federal Highway Administration (FHWA) Highway Performance Monitoring System (HPMS). The HPMS area wide data base contains State-level VMT estimates for rural and small urban areas, as well as separate VMT estimates for each large urban area within the State. VMT estimates for each of these categories are by functional roadway class. Two procedures were performed to convert this VMT data into a county/SCC level format. First, each State's rural, small urban, and large urban VMT by functional roadway class were distributed to the county level based on population data. Second, the resulting county/functional roadway class VMT were allocated to the vehicle type level based on HPMS and other FHWA data. The resulting VMT estimates are county-level estimates segregated by vehicle type and roadway class.

The 1990 emission estimates were calculated by applying 1990 control-specific emission factors to the

VMT estimates. The 1990 emission factors were generated using historical temperatures, gasoline volatility Reid vapor pressure (RVP) data, and inspection and maintenance (I/M) information. Emissions estimates are calculated at the county/vehicle type/roadway type level, allowing for county differences in I/M programs.

## Growth Projections

### Vehicle Miles Traveled

The general procedure used to project motor vehicle emissions was to grow 1990 activity (VMT) to the future year (2000 or 2010) and then to apply future year emission factors. Estimates of national growth in VMT from the MOBILE4.1 Fuel Consumption Model (FCM) (EPA, 1991c; Wolcott and Kahlbaum, 1991) were used as the basis for VMT projections. Primary MOBILE4.1 FCM inputs were vehicle registrations, VMT, and fuel economy for each vehicle class. MOBILE4.1 FCM outputs included estimates of fleet fuel consumption, VMT, on-road fuel economy, and vehicle registrations. All are national values. Historical vehicle stock information is available from R.L. Polk and Department of Transportation (DOT). The MOBILE4.1 FCM relies primarily on the R.L. Polk data to estimate historical stocks of cars and light trucks in 1990, and uses DOT and American Association of Automobile Manufacturers statistics to estimate truck stocks by weight class.

### Modeled Motor Vehicle Emission Rates

The tunnel study portion of the South Coast Air Quality Study (SCAQS) (Ingalls et al., 1989) showed that there were wide discrepancies between measured and modeled motor vehicle emissions in an experiment performed in 1987 at a tunnel near Los Angeles. Running VOC emission factors were from 1.4 to 6.9 times the emission factors calculated from the California Air Resources Board (CARB) computer program output, measured CO emission rates were from 1.1 to 3.6 times modeled emission factors, and measured $NO_x$ emission rates were 0.6 to 1.4 times the modeled values. Since that time there have been many research studies performed to attempt to identify the reasons for the observed discrepancies and to modify the two models developed by regulatory agencies (EPA's MOBILE emission factor model and CARB's emission factors model (EMFAC)) to perform better in estimating real world emission rates.

It is difficult to estimate how present uncertainties in estimating motor vehicle emissions might affect the estimated difference between Pre-CAAA and Post-CAAA emissions. The difference between the scenarios will widen if the *excess* emissions are successfully reduced by Post-CAAA measures. However, if the *excess* emissions are irreducible, or not influenced by new Post-CAAA initiatives, then the relative emissions difference between the cases would be expected to remain the same as is estimated in this analysis.

## Control Scenarios

This section describes the control assumptions for the Pre-CAAA and Post-CAAA scenarios. Table A-10 summarizes the geographic applicability of all controls modeled.

### Pre-CAAA Scenario

The Pre-CAAA scenario applies estimated increases in activity levels with emission factors reflecting control programs in place prior to the passage of the 1990 Amendments. The motor vehicle controls applied under this scenario include the Federal Vehicle Motor Control Program (FMVCP) (tailpipe standards), Phase I gasoline volatility (RVP limits), and current 1990 I/M programs.

$PM_{10}$ emission factors representing gasoline vehicles do not vary between the Pre-CAAA and Post-CAAA control scenarios. Pre-CAAA $PM_{10}$ emission factors for diesel vehicles were generated by freezing emission rates at 1993 levels, since the PART5 model does not calculate emission factors without application of CAA tailpipe standards. A composite diesel emission factor by diesel vehicle type was then calculated by applying by-model-year emission rates by yearly travel fractions.

## Post-CAAA Scenario

The Post-CAAA control scenario incorporates the likely effects of controls mandated under the CAAA. Motor vehicle controls applied under this scenario include CAA tailpipe standards and evaporative standards, Phase II RVP limits, reformulated gasoline, oxygenated fuel, I/M (none, basic, low enhanced, OTR low enhanced, and high enhanced), and low emission vehicles. Of the above mentioned controls, only I/M and reformulated gasoline affect particulate and $SO_2$ emission factors. Each of the CAA controls and their applicability are summarized in Table A-10.

### *Emission Summary*

Table A-11 summarizes national emissions by vehicle type. Comparison of Pre- and Post-CAAA scenarios shows motor vehicle VOC emissions reductions of 28 percent in 2000 and 46 percent in 2010 as a larger fraction of the vehicle fleet meets low emission vehicle (LEV) program emission standards. CAA tailpipe standards, reformulated gasoline, and I/M requirements also contribute to declines in motor vehicle VOC emissions.

For motor vehicle-emitted $NO_x$, again the differences between Post-CAAA and Pre-CAAA scenarios are most pronounced in 2010 (38 percent, compared with a 14 percent difference in 2000) as the 49 State LEV program becomes more effective with fleet turnover.

The Post-CAAA scenario also shows that there are expected to be significant CO benefits achieved through the Nonattainment (Title I) and Motor Vehicle Provisions (Title II) of the 1990 Amendments. The most important new provisions and programs expected to be providing these benefits, in order of estimated importance, include: enhanced vehicle emission inspections, wintertime oxygenated fuel use, and LEV program adoption.

$SO_2$ motor vehicle emissions decrease under the Post-CAAA scenario as a result of sulfur limits for diesel fuel. Section 211 of the CAAA limited the sulfur content of motor vehicle diesel fuel to 0.05 percent (by weight) beginning October 1, 1993.

Motor vehicle $PM_{10}$ and $PM_{2.5}$ emission changes result from CAA tailpipe standards. Almost all of the motor vehicle-emitted PM changes occur because of PM exhaust emission standards for heavy-duty diesel trucks (HDDTs).

**Table A-10**
**Applicability of Mobile Source Control Programs**

| Control Measure | Applicability |
|---|---|
| **Pre-CAAA Scenario** | |
| FMVCP | National |
| Phase I RVP | National (standard varies by region) |
| I/M | Programs in place in 1990 |
| **Post-CAAA Scenario** | |
| Phase II RVP | National (standard varies by region) |
| CAA Tailpipe Standards | National |
| CAA Evaporative Controls | National |
| Heavy Duty $NO_x$ Standard | National |
| Federal Reformulated Gasoline | Nine areas required to adopt this program under the CAA plus areas |
| Oxygenated Fuel | All CO NAAs |
| Basic I/M | All moderate ozone NAAs, moderate CO NAAs, and areas with I/M in |
| Low Enhanced I/M | All areas previously required to implement high enhanced I/M who are |
| High Enhanced I/M | Serious and above ozone NAAs, in metropolitan areas in the OTR with |
| National LEV | Nationally, with the exception of California |
| California LEV | California |

**Table A-11**
**National Highway Vehicle Emissions by Vehicle Type***
**(thousand tons)**

| | Vehicle Type | 1990 | 2000 Pre-CAAA | 2000 Post-CAAA | 2010 Pre-CAAA | 2010 Post-CAAA |
|---|---|---|---|---|---|---|
| **VOC** | | | | | | |
| | LDGV | 4,207.2 | 3,823.6 | 2,692.9 | 4,311.9 | 2,263.6 |
| | LDGT1 | 954.0 | 1,089.0 | 793.5 | 1,341.0 | 760.6 |
| | LDGT2 | 803.5 | 711.4 | 550.4 | 867.1 | 583.0 |
| | HDGV | 466.9 | 291.1 | 228.8 | 279.8 | 142.2 |
| | LDDV | 11.6 | 1.4 | 1.4 | <0.1 | <0.1 |
| | LDDT | 2.8 | 1.0 | 1.0 | 0.4 | 0.4 |
| | HDDV | 313.9 | 367.5 | 227.2 | 469.6 | 151.8 |
| | MC | 50.5 | 42.5 | 37.8 | 49.9 | 44.3 |
| | **Total** | 6,810.5 | 6,327.6 | 4,533.1 | 7,319.7 | 3,945.9 |
| **$NO_x$** | | | | | | |
| | LDGV | 3,406.1 | 3,633.6 | 2,873.8 | 4,161.6 | 2,402.1 |
| | LDGT1 | 775.2 | 1,045.5 | 815.8 | 1,326.1 | 783.8 |
| | LDGT2 | 557.8 | 646.6 | 565.1 | 821.6 | 631.6 |
| | HDGV | 333.1 | 333.2 | 324.3 | 392.6 | 296.3 |
| | LDDV | 35.9 | 4.0 | 4.0 | <0.1 | <0.1 |
| | LDDT | 7.4 | 2.5 | 2.5 | 1.0 | 1.0 |
| | HDDV | 2,318.4 | 2,135.1 | 2,051.7 | 2,359.1 | 1,443.0 |
| | MC | 11.6 | 14.1 | 14.1 | 16.5 | 16.5 |
| | **Total** | 7,445.6 | 7,814.6 | 6,651.3 | 9,078.6 | 5,574.4 |
| **CO** | | | | | | |
| | LDGV | 40,073.4 | 36,759.0 | 26,920.2 | 40,304.6 | 23,711.9 |
| | LDGT1 | 8,458.6 | 10,566.9 | 7,947.5 | 13,054.8 | 7,986.4 |
| | LDGT2 | 6,533.4 | 6,867.5 | 5,349.4 | 8,457.5 | 6,102.3 |
| | HDGV | 5,895.0 | 2,921.9 | 2,766.0 | 1,807.1 | 1,639.3 |
| | LDDV | 29.1 | 3.8 | 3.8 | <0.1 | <0.1 |
| | LDDT | 5.7 | 2.3 | 2.3 | 1.0 | 1.0 |
| | HDDV | 1,300.1 | 1,827.2 | 1,826.8 | 2,397.1 | 2,208.3 |
| | MC | 185.6 | 220.8 | 209.5 | 259.3 | 245.9 |
| | **Total** | 62,480.9 | 59,169.6 | 45,025.5 | 66,281.6 | 41,895.1 |
| **$SO_2$** | | | | | | |
| | LDGV | 143.0 | 151.6 | 151.6 | 178.0 | 178.0 |
| | LDGT1 | 37.6 | 50.1 | 50.1 | 64.0 | 64.0 |
| | LDGT2 | 20.5 | 25.6 | 25.6 | 32.6 | 32.6 |
| | HDGV | 10.8 | 11.7 | 11.7 | 14.1 | 14.1 |
| | LDDV | 12.7 | 1.1 | 0.3 | <0.1 | <0.1 |
| | LDDT | 2.8 | 0.8 | 0.2 | 0.3 | 0.1 |
| | HDDV | 340.1 | 390.3 | 97.6 | 480.3 | 120.1 |
| | MC | 0.3 | 0.4 | 0.4 | 0.5 | 0.5 |
| | **Total** | 567.7 | 631.6 | 337.4 | 769.6 | 409.2 |

| | Vehicle Type | 1990 | 2000 Pre-CAAA | 2000 Post-CAAA | 2010 Pre-CAAA | 2010 Post-CAAA |
|---|---|---|---|---|---|---|
| **$PM_{10}$** | | | | | | |
| | LDGV | 63.1 | 66.1 | 66.1 | 75.3 | 75.3 |
| | LDGT1 | 15.2 | 18.4 | 18.4 | 23.0 | 23.0 |
| | LDGT2 | 16.9 | 10.7 | 10.7 | 11.5 | 11.5 |
| | HDGV | 10.6 | 8.0 | 8.0 | 6.6 | 6.6 |
| | LDDV | 8.8 | 0.6 | 0.6 | <0.1 | <0.1 |
| | LDDT | 1.7 | 0.6 | 0.4 | 0.2 | 0.1 |
| | HDDV | 238.2 | 186.9 | 152.9 | 179.8 | 88.4 |
| | MC | 0.4 | 0.4 | 0.4 | 0.5 | 0.5 |
| | **Total** | 354.7 | 291.7 | 257.6 | 297.0 | 205.4 |
| **$PM_{2.5}$** | | | | | | |
| | LDGV | 38.1 | 36.9 | 36.9 | 43.4 | 43.4 |
| | LDGT1 | 9.8 | 11.2 | 11.2 | 13.8 | 13.8 |
| | LDGT2 | 11.1 | 6.7 | 6.7 | 7.0 | 7.0 |
| | HDGV | 7.0 | 5.3 | 5.3 | 4.3 | 4.3 |
| | LDDV | 7.8 | 0.5 | 0.5 | <0.1 | <0.1 |
| | LDDT | 1.5 | 0.5 | 0.3 | 0.2 | 0.1 |
| | HDDV | 215.7 | 164.4 | 134.4 | 164.7 | 73.9 |
| | MC | 0.2 | 0.2 | 0.2 | 0.3 | 0.2 |
| | **Total** | 291.0 | 225.7 | 195.6 | 233.7 | 142.7 |
| **$NH_3$** | | | | | | |
| | LDGV | 165.6 | 262.8 | 262.8 | 314.0 | 314.0 |
| | LDGT1 | 23.6 | 58.5 | 58.5 | 80.6 | 80.6 |
| | LDGT2 | 8.6 | 25.8 | 25.8 | 36.8 | 36.8 |
| | HDGV | 0.4 | 1.6 | 1.6 | 2.9 | 2.9 |
| | LDDV | <0.1 | <0.1 | <0.1 | <0.1 | <0.1 |
| | LDDT | <0.1 | <0.1 | <0.1 | <0.1 | <0.1 |
| | HDDV | 0.2 | 0.4 | 0.4 | 0.5 | 0.5 |
| | MC | <0.1 | <0.1 | <0.1 | 0.1 | 0.1 |
| | **Total** | 198.5 | 349.2 | 349.2 | 434.9 | 434.9 |

*The totals reflect emissions for the 48 contiguous States, excluding Alaska and Hawaii. Totals may not add due to rounding.

## Area Sources

This section discusses the base year 1990 area source inventory, and the development of the future year emission estimates for area sources. Area sources include small stationary sources not included in the point source data base (e.g., dry cleaners, graphic arts, industrial fuel combustion, gasoline marketing) and solvent use (e.g., consumer solvents, architectural coatings). The growth factors and control efficiencies used to project the base year 1990 area source inventory to 2000 and 2010 under the two control scenarios are also described, and alternative growth indicators for area sources are identified. ERCAM was used to project area source VOC and $NO_x$ emissions under the two control scenarios. The approach used in ERCAM was also used to project controlled area source CO, $SO_2$, $PM_{2.5}$, and $PM_{10}$ emissions.

### Overview of Approach

The base year 1990 area source inventory was projected to 2000 and 2010 to estimate the combined effects of growth and CAA controls on area sources. In order to project emissions, a surrogate activity indicator (e.g., population, gasoline consumption) was identified for each area source category. In its guidance for projecting emissions for area sources, EPA identifies preferred growth indicators for each area source category (EPA, 1991a). Pechan chose a growth indicator for each area source category based on EPA's guidance, the availability of projection data in the relevant years of analysis, and the appropriateness of the measure for projecting emissions. Emissions were then projected using growth factors calculated based on projections for each activity indicator. The growth rates represent an increase or decrease in the basic activity that causes emissions.

Area source emissions for VOC and $NO_x$ under each control scenario were projected using ERCAM; similar modeling techniques were used for the other criteria pollutants. The algorithm for projecting area source emissions is:

$$EMIS_Y = EMIS_{90} * GFAC_Y * \left[\frac{1 - (CE_Y * RE_Y)}{1 - (CE_{90} * RE_{90})}\right]$$

where:

| | | |
|---|---|---|
| $EMIS_Y$ | = | emissions in projection year y |
| $EMIS_{90}$ | = | 1990 emissions |
| $GFAC_Y$ | = | growth factor for projection year y |
| $CE_Y$ | = | control efficiency in projection year y |
| $RE_Y$ | = | rule effectiveness (RE) for the control in projection year y |
| $CE_{90}$ | = | 1990 control efficiency |
| $RE_{90}$ | = | 1990 RE |

In cases where the control level for the projection year control strategy is less than the control level in 1990, 1990 control levels are retained in the projection year. All computations and reporting are at the county/SCC level for air quality and cost modeling.

Under the Pre-CAAA scenario, future year control levels are assumed to be equivalent to 1990 levels. The Post-CAAA scenario applies control levels to model the effects of the Title I nonattainment provisions, Federal rules, and, in the case of VOC, Title III MACT standards.

### Base Year Emissions

The base year 1990 area source emission inventory for the prospective analysis is Version 3 of the NPI (Pechan, 1995a; Pechan, 1995b). This inventory contains county/SCC level emissions for area source categories. Most non-fugitive dust area source emissions estimates in the NPI originate with the 1985 NAPAP Area Source Emission Inventory. Exceptions to this are solvent emissions, prescribed burning, forest wildfires, fugitive dust, and residential wood combustion.

The general method for estimating 1990 area source emissions using the 1985 NAPAP Inventory was to apply growth factors to the NAPAP Inventory values. BEA historical earnings data, population, fuel

use from State Energy Data System (SEDS) (DOE, 1991), and other category-specific indicators were used to project the 1985 NAPAP to 1990. SEDS data were used as an indicator of emissions growth for the area source fuel combustion categories and for the gasoline marketing categories (EPA, 1993b). Particle size multipliers were applied to estimate $PM_{10}$ emissions from TSP estimates (EPA, 1995).

*Solvent* emissions were estimated from a national solvent material balance using solvent data from various marketing surveys (EPA, 1993b). Emissions are allocated to the county-level based on employment and population data.

*Prescribed burning* emission estimates were based on a 1989 United States Department of Agriculture (USDA) Forest Service Inventory of PM and air toxics (USDA, 1989). This inventory of prescribed burning contained State-level emissions, which were allocated to the county level using the State-to-county distribution of emissions in the 1985 NAPAP Inventory.

*Wildfire* emissions were taken from estimates developed for the GCVTC for the 11 GCVTC States (Western U.S.) (Radian, 1995). The wildfire data in the GCVTC Inventory represent a detailed survey of forest fires in the study area. For non-GCVTC States, emissions are based on the 1985 NAPAP Inventory values.

$PM_{10}$ emissions for *fugitive dust* sources were taken from the Trends inventory (EPA, 1997a) for agricultural tilling, agricultural burning, construction activity, paved roads, unpaved roads, prescribed burning, and wind erosion. Emissions from beef cattle feedlots were developed for the NPI. In general, the Trends Inventory emission estimates are available at the State level, with the exception of construction activity emission estimates, which are at the EPA-region level. These were disaggregated to the county level based on Census of Agriculture data, land use, and construction earnings data. Paved and unpaved road emissions are estimated using the EPA's OMS PART5 emission factor model combined with paved and unpaved road VMT estimates based on FHWA data. PART 5 reentrained road dust emission factors depend on the average weight, speed, and number of wheels of the vehicles traveling on paved and unpaved roadways, the silt content of roadway surface material, and precipitation data. The activity factor for calculating reentrained road dust emissions is VMT.

*Residential wood combustion* emissions estimated for EPA's NET effort were used in Version 3 of the NPI (EPA, 1993c). Residential wood combustion emissions include those from traditional masonry fireplaces, freestanding fireplaces, wood stoves, and furnaces.

For the States of California and Oregon, 1990 criteria pollutant emissions from the GCVTC inventory were incorporated for all area source categories. The data for these two States are based on State-compiled inventories that are presumably based on more recent and detailed data than the emissions in the NAPAP Inventory.

## Growth Projections

The base year 1990 area source inventory was projected to 2000 and 2010 to determine the effects of CAA controls on future emission levels. Growth in pollution generating activity to future years was estimated using the BEA industry GSP and population projections for most area source categories. Exceptions include activity indicators for agricultural tilling and burning and managed (or prescribed) burning. For example, the USDA has developed baseline projections of farm acres planted (USDA, 1998). These data, combined with historical data back to 1990, for eight major crop types shows an average annual growth of only 0.38 percent per year from 1990 to 2007. The BEA GSP projections for *farm* result in an annual average growth of 2.0 percent per year. Projections of acres planted represent better predictors of future activity than GSP for agricultural tilling, so they were used in this projection.

EPA's projection guidance states that area source projections can be made using local studies or surveys,

or through surrogate growth indicators such as BEA. Because this is a national projection, BEA was chosen as a consistent data set which could be used across all regions. Emissions must be allocated to the grid cell in order to perform air quality modeling; these projections will not reflect changes in the spatial patterns of emissions between counties or grid cells, since State-level growth surrogates are used. Essentially, BEA GSP projections incorporate inputs to production, and therefore, reflect future changes in technology, processes, and efficiency. Ideally, projections from States and Metropolitan Planning Organizations would be a more reliable estimate of growth in population, land use, and employment, but these were not available in a consistent format for the entire contiguous United States.

## Control Scenarios

The Pre-CAAA scenario for area source emissions assumes that future year control levels are equal to those in 1990 with the exception of applying the new source performance standards (NSPS) for residential woodstoves. The residential woodstove NSPS affects emissions of both PM and VOC in all areas. The Post-CAAA scenario applies future year controls to model the impact of the 1990 Amendments on projected emissions.

Changes in agricultural practices are likely to influence future fugitive dust emissions from activities like agricultural tilling. In recent years, agricultural practices such as conservation tillage have been instituted to provide protection against surface soil erosion, primarily from water and runoff losses. These practices have also affected wind erosion losses. The primary attributes of conservation tillage practices are: (1) reducing the number of passes by farm vehicles; and (2) maintaining a higher amount of crop residue in the soil. With respect to particle emissions from tilling operations, the reduced number of vehicle passes through the field is the most important parameter. The emission rate (using current EPA estimation methods) is primarily related to the acres tilled (and therefore, the number of vehicle passes) and the soil silt content. Assuming the soil silt content remains the same, reducing the number of vehicle passes produces a proportional reduction in emissions. The increased crop residue provided by conservation tillage acts to help shelter the soil particles from wind erosion, which reduces soil depletion and reduces vertical fluxes of particles to the atmosphere. The increased residue has little, if any, effect on emissions.

Projections of conservation tillage practices are that the amount of conservation tillage in 2000 will be 26 percent of total acres tilled. The 26 percent figure is the level achieved in 1990. The 2010 projection assumes that conservation tillage increases to 50 percent by 2010. Because the trend toward conservation tillage appears to result from the 1985 Farm Bill conservation compliance program, economic influences, and improved efficiency, the same assumptions are used for estimating Pre- and Post-CAAA $PM_{10}$ and $PM_{2.5}$ emissions for this category.

Under the Post-CAAA scenario, controls are implemented in PM NAAs. The controls modeled depend on the severity of PM nonattainment and the level of emissions from source categories for which controls are available. The Post-CAAA projection for $NO_x$ incorporates controls for industrial fuel combustion emissions to model the effects of lowering the RACT source size cutoff in ozone NAAs. Low $NO_x$ burners were selected as the representative $NO_x$ control. CAA controls affecting VOC include controls for Title I (RACT, new CTGs, stage II vapor recovery, and Federal consumer solvent controls), Title III MACT standards, and onboard vapor recovery systems. The same control level is applied in 2000 and 2010. Future year control levels for $SO_2$ and CO area source emitters (generally fuel combustion and fires) were assumed to be equivalent to 1990 levels under both the Pre-CAAA and Post-CAAA scenario.

## Emission Summary

Table A-12 is a summary of emission projections by year and scenario at the Tier 2 source category level.

The VOC emission differences between Post-CAAA and Pre-CAAA scenarios shown in Table A-12 for 2000 and 2010 are largely expected to result from Federal measures and ozone NAA-specific requirements to reduce ozone precursor emissions. Area source categories with the biggest differences between Post-CAAA and Pre-CAAA VOC emissions include commercial and consumer solvent use, surface coating (paints), small graphic arts shops, and hazardous waste treatment, storage, and disposal facilities (TSDFs). Note that while many hazardous waste TSDFs are large enough emitters to be classified as point sources, they are represented in the NPI as area sources. Service station VOC emissions are reduced in 2000 primarily by Stage II vapor recovery systems installed in NAAs, with further reductions in refueling emissions expected by 2010 as onboard vapor recovery systems are installed in new cars and light trucks.

Only modest $NO_x$ emission benefits are expected from CAA mandates for area sources, as most CAA initiatives focus on major stationary sources. Some $NO_x$ reducing measures, however, do affect the area source category; some controls are required in serious, severe, and extreme ozone NAAs emitting 25 tons per year, or less. In addition, some sources emitting 25 to 100 tons of $NO_x$ per year are represented in the area source emissions file. Area source $PM_{10}$ emitters with differences between the Post-CAAA and Pre-CAAA scenarios are fugitive dust sources.

**Table A-12**
**Area Source Emission Summary by Pollutant For 1990, 2000, and 2010***
**(thousand tons)**

| Pollutant/Source Category | 1990 | 2000 Pre-CAAA | 2000 Post-CAAA | 2010 Pre-CAAA | 2010 Post-CAAA |
|---|---|---|---|---|---|
| **VOC** | | | | | |
| Fuel Comb. Industrial | 17.8 | 22.4 | 22.4 | 26.6 | 26.6 |
| Fuel Comb. Other | 686.0 | 623.6 | 623.6 | 518.2 | 518.2 |
| Chemical & Allied Product Mfg | 449.2 | 517.0 | 366.3 | 578.5 | 408.5 |
| Petroleum & Related Industries | 450.2 | 454.8 | 198.5 | 494.5 | 207.7 |
| Other Industrial Processes | 84.4 | 94.8 | 93.1 | 108.0 | 106.2 |
| Solvent Utilization | 4,701.0 | 5,459.2 | 4,290.7 | 6,146.6 | 4,780.6 |
| Storage & Transport | 1,220.3 | 1,537.8 | 1,328.8 | 1,744.6 | 1,298.1 |
| Waste Disposal & Recycling | 2,154.6 | 2,596.5 | 471.3 | 3,030.2 | 524.2 |
| Natural Sources | 13.8 | 13.8 | 13.8 | 13.8 | 13.8 |
| Miscellaneous | 568.6 | 621.6 | 584.9 | 656.8 | 656.8 |
| TOTAL | 10,345.9 | 11,941.7 | 7,993.5 | 13,317.7 | 8,540.6 |
| **$NO_x$** | | | | | |
| Fuel Comb. Industrial | 1,269.7 | 1,615.6 | 1,600.2 | 1,917.8 | 1,900.1 |
| Fuel Comb. Other | 611.2 | 702.7 | 702.7 | 797.7 | 797.7 |
| Petroleum & Related Industries | 19.4 | 15.8 | 15.8 | 15.0 | 15.0 |
| Other Industrial Processes | 4.3 | 5.2 | 5.2 | 6.0 | 6.0 |
| Waste Disposal & Recycling | 60.0 | 67.3 | 67.3 | 74.5 | 74.5 |
| Miscellaneous | 224.1 | 228.1 | 228.1 | 230.7 | 230.7 |
| TOTAL | 2,188.8 | 2,634.8 | 2,619.4 | 3,041.7 | 3,023.9 |
| **CO** | | | | | |
| Fuel Comb. Industrial | 192.6 | 244.3 | 244.3 | 289.5 | 289.5 |
| Fuel Comb. Other | 3,759.1 | 4,674.4 | 4,674.4 | 5,485.9 | 5,485.9 |
| Petroleum & Related Industries | 3.9 | 3.2 | 3.2 | 3.0 | 3.0 |
| Other Industrial Processes | 2.0 | 2.7 | 2.7 | 3.1 | 3.1 |
| Waste Disposal & Recycling | 1,401.9 | 1,542.9 | 1,542.9 | 1,672.8 | 1,672.8 |
| Miscellaneous | 6,246.0 | 6,477.6 | 6,477.6 | 6,625.6 | 6,625.6 |
| TOTAL | 11,605.5 | 12,945.0 | 12,945.0 | 14,080.0 | 14,080.0 |
| **$SO_2$** | | | | | |
| Fuel Comb. Industrial | 626.9 | 803.6 | 803.6 | 948.4 | 948.4 |
| Fuel Comb. Other | 390.6 | 463.8 | 463.8 | 541.5 | 541.5 |
| Petroleum & Related Industries | 1.4 | 1.2 | 1.2 | 1.1 | 1.1 |
| Other Industrial Processes | 1.7 | 2.2 | 2.2 | 2.5 | 2.5 |
| Waste Disposal & Recycling | 14.8 | 17.1 | 17.1 | 19.6 | 19.6 |
| Miscellaneous | 6.2 | 6.3 | 6.3 | 6.3 | 6.3 |
| TOTAL | 1,041.5 | 1,294.0 | 1,294.0 | 1,519.4 | 1,519.4 |

| Pollutant/Source Category | 1990 | 2000 Pre-CAAA | 2000 Post-CAAA | 2010 Pre-CAAA | 2010 Post-CAAA |
|---|---|---|---|---|---|
| **$PM_{10}$** | | | | | |
| Fuel Comb. Industrial | 29.1 | 37.1 | 37.1 | 43.8 | 43.8 |
| Fuel Comb. Other | 510.7 | 470.1 | 469.6 | 398.8 | 398.3 |
| Metals Processing | <0.1 | <0.1 | <0.1 | <0.1 | <0.1 |
| Petroleum & Related Industries | 1.6 | 1.3 | 1.3 | 1.2 | 1.2 |
| Other Industrial Processes | 34.6 | 37.9 | 37.9 | 44.2 | 44.2 |
| Waste Disposal & Recycling | 218.2 | 240.7 | 240.7 | 261.6 | 261.6 |
| Natural Sources | 2,092.4 | 2,092.4 | 2,092.4 | 2,092.4 | 2,092.4 |
| Miscellaneous | 23,501.7 | 23,915.2 | 23,262.0 | 23,947.9 | 23,189.6 |
| TOTAL | 26,388.4 | 26,794.7 | 26,141.0 | 26,790.0 | 26,031.3 |
| **$PM_{2.5}$** | | | | | |
| Fuel Comb. Industrial | 14.8 | 18.8 | 18.8 | 22.2 | 22.2 |
| Fuel Comb. Other | 495.8 | 453.0 | 452.2 | 379.3 | 378.3 |
| Metals Processing | <0.1 | <0.1 | <0.1 | <0.1 | <0.1 |
| Petroleum & Related Industries | 1.6 | 1.3 | 1.3 | 1.2 | 1.2 |
| Other Industrial Processes | 22.6 | 25.1 | 25.1 | 29.3 | 29.3 |
| Waste Disposal & Recycling | 190.7 | 210.1 | 210.1 | 228.1 | 228.1 |
| Natural Sources | 313.9 | 313.9 | 313.9 | 313.9 | 313.9 |
| Miscellaneous | 4,769.0 | 5,016.9 | 4,911.3 | 5,296.1 | 5,173.0 |
| TOTAL | 5,808.0 | 6,038.7 | 5,932.7 | 6,270.2 | 6,146.0 |
| **$NH_3$** | | | | | |
| Fuel Comb. Industrial | 7.3 | 9.1 | 9.1 | 10.7 | 10.7 |
| Fuel Comb. Other | 7.7 | 8.8 | 8.8 | 10.0 | 10.0 |
| Other Industrial Processes | 35.5 | 42.5 | 42.5 | 49.7 | 49.7 |
| Waste Disposal & Recycling | 81.8 | 100.7 | 100.7 | 119.2 | 119.2 |
| Miscellaneous | 3,593.8 | 4,650.6 | 4,650.6 | 5,542.2 | 5,542.2 |
| TOTAL | 3,726.1 | 4,811.6 | 4,811.6 | 5,731.8 | 5,731.8 |

* The totals reflect emissions for the 48 contiguous States, excluding Alaska and Hawaii. Totals may not add due to rounding.

## Reasonable Further Progress Requirements

The Post-CAAA scenario incorporates the effect that Title I ozone controls will have on the emissions of VOC and $NO_x$, both ozone precursors. The ozone nonattainment provisions in this section of the 1990 CAAA state that areas not in compliance with the ozone national ambient air quality standard (NAAQS) must reduce precursor emissions. NAAs classified as moderate, serious, severe, or extreme all are directed to meet the required rate of progress (ROP), and by 1996, cut annual VOC emissions by 15 percent from 1990 levels. In addition, serious, severe, and extreme ozone NAAs must continue reducing precursor emissions after the 1996 ROP deadline. Areas falling into these three categories are required to make reasonable further progress (RFP) towards attainment. To satisfy this regulation NAAs must cut VOC levels by 3 percent annually until they comply with the ozone NAAQS.[6]

For areas to comply with ROP requirements VOC reductions are mandated. For those NAAs that must make additional cuts and satisfy RFP regulations it is possible for $NO_x$ reductions to be substituted for VOC cuts. This trading of one ozone precursor for another is acceptable as long as: 1) a NAA has not been given a $NO_x$ waiver, and 2) the substitution of $NO_x$ for VOC does not result in a greater reduction of NOx than is necessary for an area to comply with the ozone NAAQS.

The ROP and RFP requirements are designed to establish a minimum standard for reducing ozone precursor emissions. In many cases, nonattainment areas satisfy these two regulations simply by complying with other ozone provisions of the CAAA. Reduction of VOC and $NO_x$, below 1990 baseline levels, made in order to meet other standards, are credited towards ROP/RFP requirements. These credited reductions, although captured by the Post-CAAA scenario, are not a direct result of ROP/RFP standards. To accurately capture the influence of ROP/RFP requirements in the Post-CAAA scenario it was necessary to predict which NAAs would have to make emissions cuts solely for the purpose of satisfying these progress requirements, which precursor(s) would be cut, what the size of the cuts would be, and which sources would be forced to make these cuts.

For the purpose of the prospective analysis it was assumed that NAAs working to satisfy ROP and RFP requirements would, whenever possible, first take credit for all available $NO_x$ reductions and then for all available VOC reductions; any remaining shortfall, corresponding to cuts that NAAs would have to make specifically to meet ROP or RFP requirements, would be made up through additional VOC emission reductions. The size of the total shortfall for all NAAs, thus, is a measure of the impact of these Title I progress requirements on the emission of ozone precursors. This shortfall is captured by the Post-CAAA scenario.

To estimate the VOC shortfall, a separate daily VOC target and daily $NO_x$ target was calculated for the years 2000 and 2010 for every NAA subject to Title I progress requirements. These target figures represent the daily maximum allowable emissions levels that NAAs cannot exceed if they are to comply with ROP/RFP standards. The $NO_x$ target was set according to how much $NO_x$ credit it was assumed will be counted towards ROP/RFP requirements, and the VOC target was set based on the assumption that the remainder of the emissions cuts needed to satisfy ROP/RFP requirements will come through reductions in VOC. The difference between the VOC target and the expected level of daily VOC emissions in the absence of ROP/RFP requirements, represented by ozone season daily (OSD) emissions estimates, equals the shortfall.

Table A-13 shows, for each NAA subject to ROP/RFP requirements, both the OSD and target VOC and $NO_x$ emissions levels for 2000. Table A-14

[6] The prospective analysis does not model for attainment. For the purposes of this analysis NAAs are assumed to make reasonable further progress until their respective attainment deadlines, as outlined in the CAAA, are reached.

contains the same information for projection year 2010. In addition, these tables indicate whether or not an area has a $NO_x$ waiver (these areas cannot be given $NO_x$ credit towards RFP), and reveal how RFP reductions were divided between VOC and $NO_x$ in order to calculate the target values for both ozone precursors. For example, looking at Table A-13 shows that for Philadelphia in the year 2000, $NO_x$ reductions of 18 percent from 1990 base year levels are credited towards satisfying RFP provisions. After accounting for this credit, the maximum daily VOC emissions that is allowable for Philadelphia, if this area is to comply with progress requirements, is 1,376.55 tons. Since this VOC target value is greater than the predicted level of daily emissions (VOC OSD) of 1,090.49 tons, there is no shortfall. For Philadelphia in the year 2010 (Table A-14), however, an additional 9 percent reduction from 1990 VOC emission levels is necessary in order to satisfy RFP requirements. Once again there is no shortfall.

After calculating the shortfall for each NAA, EPA estimated how these additional VOC reductions would be achieved. The Agency compiled a list of the available area and point source controls and assumed that each NAA would adopt the most cost-effective measure, followed by the second most cost-effective option, and so on until the area satisfied its ROP/RFP requirements. Table A-15 displays the control option identified by EPA and lists them in the approximate order the Agency believed they would be selected by NAAs attempting to eliminate shortfall emissions. In general, it is assumed that nonroad controls would be the last to be selected, along with AIM coatings. Furthermore, since most areas in the analysis already have reformulated gasoline and I/M programs in the baseline, these were not considered as potential discretionary measures. It should be noted that episodic bans are not creditable towards ROP shortfalls. These measures were chosen to be illustrative of what individual areas might select to meet ROP shortfalls, however, individual areas may select measures that differ from those modeled here. Some of the measures may be politically infeasible for some areas, however, the cost thresholds used to estimate the costs of these measures are consistent with those used in the ozone NAAQS analysis.

Shortfalls are the greatest and most difficult to eliminate in severe ozone NAAs with $NO_x$ waivers. These areas have to meet significant ozone precursor reduction requirements solely by cutting VOC emissions. In Chicago and Milwaukee-Racine, both wavier areas, the VOC shortfall is so large that it cannot be eliminated even if all of the identified controls are implemented. As a result, for these two NAAs it is assumed that unidentified controls are adopted to reduce the remaining shortfall. In all other areas, however, the required reduction can be achieved with the identified controls.

**Table A-13**
**2000 Rate of Progress Analysis**

| Att. Class. | Att. Date | Ozone Nonattainment Area | 2000 OSD (tons per day)[1] $NO_x$ | VOC | Target Selection[2] | $NO_x$ Target | VOC Target | Shortfall |
|---|---|---|---|---|---|---|---|---|
| Ser. | 1999 | Atlanta | 420.93 | 518.94 | 1%$NO_x$/8%VOC | 426.28 | 476.35 | 42.59 |
| Mod. | 1996 | Atlantic City | 46.11 | 37.97 | ROP - 15%VOC | 0.00 | 45.71 | 0.00 |
| Sev. | 2005 | Baltimore | 335.42 | 318.33 | 18%$NO_x$ | 381.53 | 376.47 | 0.00 |
| Ser. | 1999 | Baton Rouge | 449.70 | 203.77 | $NO_x$ waiver | 0.00 | 415.65 | 0.00 |
| Ser. | 1999 | Beaumont-Port Arthur | 245.20 | 340.66 | $NO_x$ waiver | 0.00 | 450.66 | 0.00 |
| Ser. | 1999 | Boston-Lawrence-Worcester-E.MA | 560.20 | 822.65 | 9% $NO_x$ | 602.06 | 918.01 | 0.00 |
| Sev. | 2007 | Chicago-Gary-Lake County | 1,056.70 | 1,240.89 | $NO_x$ waiver | 0.00 | 1,202.25 | 38.64 |
| Mod. | 1996 | Cincinnati-Hamilton | 378.70 | 305.34 | ROP - 15%VOC | 0.00 | 341.18 | 0.00 |
| Mod. | 1996 | Cleveland-Akron-Lorain | 369.12 | 521.14 | ROP - 15%VOC | 0.00 | 573.07 | 0.00 |
| Mod. | 1996 | Dallas-Fort Worth | 528.83 | 694.53 | ROP - 15%VOC | 0.00 | 673.97 | 20.56 |
| Ser. | 1999 | El Paso | 113.35 | 85.38 | 0%$NO_x$/9%VOC | 0.00 | 69.02 | 16.36 |
| Mod. | 1996 | Grand Rapids | 129.61 | 182.72 | ROP - 15%VOC | 0.00 | 175.66 | 7.06 |
| Ser. | 1999 | Greater Connecticut | 211.67 | 316.27 | 9%$NO_x$ | 224.49 | 370.11 | 0.00 |
| Sev. | 2007 | Houston-Galveston-Brazoria | 1,094.58 | 1,426.65 | $NO_x$ waiver | 0.00 | 2,268.31 | 0.00 |
| Mod. | 1996 | Kewaunee Co Wi | 3.23 | 4.86 | ROP - 15 VOC | 0.00 | 4.56 | 0.30 |
| Mod. | 1996 | Knox & Lincoln Cos ME | 9.26 | 9.95 | ROP - 15%VOC | 0.00 | 10.37 | 0.00 |
| Mod. | 1996 | Lewiston-Auburn ME | 23.56 | 34.08 | ROP - 15%VOC | 0.00 | 35.98 | 0.00 |
| Ext. | 2010 | Los Angeles-South Coast | 1,010.32 | 972.91 | 1%$NO_x$/17%VOC | 1,019.06 | 939.08 | 33.83 |
| Mod. | 1996 | Louisville | 264.00 | 219.66 | ROP - 15%VOC | 0.00 | 215.97 | 3.69 |
| Mod. | 1996 | Manitowoc Co WI | 13.69 | 19.72 | ROP - 15%VOC | 0.00 | 17.20 | 2.52 |
| Sev. | 2007 | Milwaukee-Racine | 272.22 | 327.09 | $NO_x$ waiver | 0.00 | 293.24 | 33.85 |
| Mod. | 1996 | Monterey Bay | 78.60 | 64.16 | ROP - 15%VOC | 0.00 | 79.63 | 0.00 |
| Mod. | 1996 | Muskegon | 34.25 | 46.67 | ROP - 15%VOC | 0.00 | 44.32 | 2.35 |
| Mod. | 1996 | Nashville | 167.12 | 231.71 | ROP - 15%VOC | 0.00 | 205.60 | 26.11 |
| Sev. | 2007 | New York-N New Jersey-Long Is | 1,280.26 | 1,994.96 | 18%$NO_x$ | 1,553.41 | 2,407.97 | 0.00 |
| Sev. | 2005 | Philadelphia-Wilmingtn-Trenton | 678.53 | 1,090.49 | 18%$NO_x$ | 690.79 | 1,376.55 | 0.00 |
| Mod. | 1996 | Phoenix | 404.47 | 377.43 | ROP - 15%VOC | 0.00 | 347.91 | 29.52 |
| Mod. | 1996 | Pittsburgh-Beaver Valley | 534.53 | 407.05 | ROP - 15%VOC | 0.00 | 399.80 | 7.25 |
| Mod. | 1996 | Portland ME | 53.72 | 70.05 | ROP - 15%VOC | 0.00 | 73.33 | 0.00 |
| Ser. | 1999 | Portsmouth-Dover-Rochester | 37.55 | 53.54 | 9%$NO_x$ | 44.26 | 58.70 | 0.00 |
| Ser. | 1999 | Providence | 92.51 | 173.78 | 5%$NO_x$/4%VOC | 91.77 | 180.51 | 0.00 |
| Mod. | 1996 | Reading PA | 48.66 | 60.53 | ROP - 15%VOC | 0.00 | 61.14 | 0.00 |

| Att. Class. | Att. Date | Ozone Nonattainment Area | 2000 OSD (tons per day)[1] $NO_x$ | VOC | Target Selection[2] | $NO_x$ Target | VOC Target | Shortfall |
|---|---|---|---|---|---|---|---|---|
| Mod. | 1996 | Richmond-Petersburg | 141.62 | 179.97 | ROP - 15%VOC | 0.00 | 201.70 | 0.00 |
| Sev. | 2005 | Sacramento Metro | 164.60 | 158.01 | 0%$NO_x$/18%VOC | 0.00 | 155.08 | 2.93 |
| Mod. | 1996 | Salt Lake City | 178.25 | 182.75 | ROP - 15%VOC | 0.00 | 150.80 | 31.95 |
| Ser. | 1999 | San Diego | 228.55 | 192.90 | 0%$NO_x$/9%VOC | 0.00 | 189.71 | 3.19 |
| Ser. | 1999 | San Joaquin Valley | 499.25 | 470.50 | 5%$NO_x$/4%VOC | 505.63 | 532.41 | 0.00 |
| Mod. | 1996 | Santa Barbara-Santa Maria-Lomp | 63.77 | 82.75 | ROP - 15%VOC | 0.00 | 83.10 | 0.00 |
| Mod. | 1996 | Sheyboygan | 38.02 | 24.49 | ROP - 15%VOC | 0.00 | 22.44 | 2.05 |
| Sev. | 2007 | Southeast Desert Modified | 355.88 | 227.71 | 3%$NO_x$/15%VOC | 358.76 | 219.32 | 8.39 |
| Ser. | 1999 | Springfield/Pittsfield-W. MA | 112.38 | 155.51 | 0%$NO_x$/9%VOC | 0.00 | 152.42 | 3.09 |
| Mod. | 1996 | St. Louis | 476.98 | 465.64 | ROP - 15%VOC | 0.00 | 549.14 | 0.00 |
| Sev. | 2005 | Ventura Co CA | 80.45 | 65.69 | 13%$NO_x$/5%VOC | 79.55 | 70.52 | 0.00 |
| Ser. | 1999 | Washington DC | 449.80 | 402.76 | 9%$NO_x$ | 499.12 | 477.03 | 0.00 |

NOTES:
[1]OSD = ozone season daily
[2]The target selection column indicates the percentage reduction of $NO_x$ NAA's are credited towards RFP and what percentage reduction in VOC is then needed to satisfy Title I progress requirements.

**Table A-14**
**2010 Rate of Progress Analysis**

| Att. Class. | Att. Date | Ozone Nonattainment Area | 2010 OSD (tons per day)[1] $NO_x$ | VOC | Target Selection[2] | $NO_x$ Target | VOC Target | Shortfall |
|---|---|---|---|---|---|---|---|---|
| Ser. | 1999 | Atlanta | 336.10 | 492.40 | 9%$NO_x$ | 391.84 | 541.99 | 0.00 |
| Mod. | 1996 | Atlantic City | 39.46 | 33.21 | ROP - 15%VOC | 0.00 | 45.71 | 0.00 |
| Sev. | 2005 | Baltimore | 278.64 | 293.56 | 27%$NO_x$ | 311.86 | 376.47 | 0.00 |
| Ser. | 1999 | Baton Rouge | 407.49 | 206.65 | $NO_x$ waiver | 0.00 | 415.65 | 0.00 |
| Ser. | 1999 | Beaumont-Port Arthur | 232.32 | 377.63 | $NO_x$ waiver | 0.00 | 450.66 | 0.00 |
| Ser. | 1999 | Boston-Lawrence-Worcester-E.MA | 478.53 | 775.66 | 9%$NO_x$ | 602.06 | 918.01 | 0.00 |
| Sev. | 2007 | Chicago-Gary-Lake County | 990.61 | 1,236.73 | $NO_x$ waiver | 0.00 | 840.15 | 396.58 |
| Mod. | 1996 | Cincinnati-Hamilton | 288.20 | 283.74 | ROP - 15%VOC | 0.00 | 341.18 | 0.00 |
| Mod. | 1996 | Cleveland-Akron-Lorain | 306.32 | 485.90 | ROP - 15%VOC | 0.00 | 573.07 | 0.00 |
| Mod. | 1996 | Dallas-Fort Worth | 472.66 | 687.15 | ROP - 15%VOC | 0.00 | 673.97 | 13.18 |
| Ser. | 1999 | El Paso | 113.85 | 84.33 | 0%$NO_x$/9%VOC | 0.00 | 69.02 | 15.31 |
| Mod. | 1996 | Grand Rapids | 92.07 | 183.11 | ROP - 15%VOC | 0.00 | 175.66 | 7.45 |
| Ser. | 1999 | Greater Connecticut | 192.62 | 292.53 | 9%$NO_x$ | 224.49 | 370.11 | 0.00 |
| Sev. | 2007 | Houston-Galveston-Brazoria | 1,002.13 | 1,530.07 | $NO_x$ waiver | 0.00 | 1,606.75 | 0.00 |
| Mod. | 1996 | Kewaunee Co Wi | 2.62 | 4.77 | ROP - 15%VOC | 0.00 | 4.56 | 0.21 |
| Mod. | 1996 | Knox & Lincoln Cos ME | 8.51 | 8.98 | ROP - 15%VOC | 0.00 | 10.37 | 0.00 |
| Mod. | 1996 | Lewiston-Auburn ME | 22.20 | 32.03 | ROP - 15%VOC | 0.00 | 35.98 | 0.00 |
| Ext. | 2010 | Los Angeles-South Coast | 950.39 | 847.66 | 5%$NO_x$/31%VOC | 964.60 | 670.95 | 176.71 |
| Mod. | 1996 | Louisville | 273.40 | 216.86 | ROP - 15%VOC | 0.00 | 215.97 | 0.89 |
| Mod. | 1996 | Manitowoc Co WI | 12.10 | 19.37 | ROP - 15%VOC | 0.00 | 17.20 | 2.17 |
| Sev. | 2007 | Milwaukee-Racine | 246.74 | 321.89 | $NO_x$ waiver | 0.00 | 204.72 | 117.17 |
| Mod. | 1996 | Monterey Bay | 71.93 | 61.76 | ROP - 15%VOC | 0.00 | 79.63 | 0.00 |
| Mod. | 1996 | Muskegon | 26.99 | 46.86 | ROP - 15%VOC | 0.00 | 44.32 | 2.54 |
| Mod. | 1996 | Nashville | 143.58 | 230.00 | ROP - 15%VOC | 0.00 | 205.60 | 24.40 |
| Sev. | 2007 | New York-N New Jersey-Long Is | 1,148.95 | 1,842.53 | 36%$NO_x$ | 1,166.20 | 2,407.97 | 0.00 |
| Sev. | 2005 | Philadelphia-Wilmingtn-Trenton | 631.39 | 1,070.05 | 18%$NO_x$/9%VOC | 632.97 | 1,194.41 | 0.00 |

| Att. Class. | Att. Date | Ozone Nonattainment Area | 2010 OSD (tons per day)[1] $NO_x$ | VOC | Target Selection[2] | $NO_x$ Target | VOC Target | Shortfall |
|---|---|---|---|---|---|---|---|---|
| Mod. | 1996 | Phoenix | 395.64 | 347.52 | ROP - 15%VOC | 0.00 | 347.91 | 0.00 |
| Mod. | 1996 | Pittsburgh-Beaver Valley | 368.47 | 358.67 | ROP - 15%VOC | 0.00 | 399.80 | 0.00 |
| Mod. | 1996 | Portland ME | 51.54 | 66.88 | ROP - 15%VOC | 0.00 | 73.33 | 0.00 |
| Ser. | 1999 | Portsmouth-Dover-Rochester | 33.08 | 52.49 | 9%$NO_x$ | 44.26 | 58.70 | 0.00 |
| Ser. | 1999 | Providence | 75.73 | 166.61 | 9%$NO_x$ | 87.91 | 193.15 | 0.00 |
| Mod. | 1996 | Reading PA | 46.61 | 55.33 | ROP - 15%VOC | 0.00 | 61.14 | 0.00 |
| Mod. | 1996 | Richmond-Petersburg | 137.12 | 179.35 | ROP - 15%VOC | 0.00 | 201.70 | 0.00 |
| Sev. | 2005 | Sacramento Metro | 152.59 | 135.99 | 1%$NO_x$/26%VOC | 152.62 | 120.24 | 15.75 |
| Mod. | 1996 | Salt Lake City | 179.22 | 189.83 | ROP - 15%VOC | 0.00 | 150.80 | 39.03 |
| Ser. | 1999 | San Diego | 213.59 | 174.04 | 5%$NO_x$/4%VOC | 213.42 | 202.24 | 0.00 |
| Ser. | 1999 | San Joaquin Valley | 466.55 | 448.37 | 9%$NO_x$ | 484.34 | 566.96 | 0.00 |
| Mod. | 1996 | Santa Barbara-Santa Maria-Lomp | 59.33 | 81.53 | ROP - 15%VOC | 0.00 | 83.10 | 0.00 |
| Mod. | 1996 | Sheyboygan | 34.84 | 24.69 | ROP - 15%VOC | 0.00 | 22.44 | 2.25 |
| Sev. | 2007 | Southeast Desert Modified | 329.75 | 213.87 | 9%$NO_x$/27%VOC | 332.55 | 172.34 | 41.53 |
| Ser. | 1999 | Springfield/Pittsfield-W. MA | 97.63 | 147.45 | 7%$NO_x$/2%VOC | 97.87 | 166.51 | 0.00 |
| Mod. | 1996 | St. Louis | 381.48 | 439.47 | ROP - 15%VOC | 0.00 | 549.14 | 0.00 |
| Sev. | 2005 | Ventura Co CA | 75.89 | 62.33 | 13%$NO_x$/14%VO | 76.34 | 63.11 | 0.00 |
| Ser. | 1999 | Washington DC | 393.57 | 355.35 | 9%$NO_x$ | 499.12 | 477.03 | 0.00 |

Notes:
[1]OSD = ozone season daily
[2]The target selection column indicates the percentage reduction of $NO_x$ NAA's are credited towards RFP and what percentage reduction in VOC is then needed to satisfy Title I progress requirements.

**Table A-15**
**Discretionary Control Measures Modeled For ROP/RFP**

| Source Category | Measure | Reduction |
|---|---|---|
| **Area Source** | | |
| Adhesives - industrial | Reformulation | 63% |
| Metal product surface coating | VOC content limits & improved transfer efficiency | 30% (50% in 2010 for difficult areas) |
| Cutback asphalt | Switch to emulsified asphalts (100% RE) | 100% |
| Wood product surface coating | Reformulation | 43% |
| Wood furniture surface coating | Reformulation | 43% |
| Degreasing | Solvent Limits | 63% (80% in difficult areas) |
| Open burning | Seasonal ban | 80% |
| Automobile refinishing | CARB Best Available Retrofit Control Technology (BARCT) limits | 47% |
| Bulk Terminals | Leak Detection and Repair (LDAR) | 90% (80% RE) |
| POTWs | Covers/Adsorption | 50% |
| Bakeries | Afterburner | 33% |
| Petroleum dry cleaning | Recovery dryers | 44% |
| Perchloroethylene dry cleaning | Recovery dryers | 70% (80%RE) |
| Livestock | Recovery system | 50% |
| Miscellaneous surface coating | Reformulation | 30% |
| Aerosols | South Coast Air Quality Management District (SCAQMD) Standards - Reformulation | 50% (60% in 2010) |
| Incineration | Seasonal ban | 80% |
| Synthetic fiber manufacture | Adsorber | 78% (80%RE) |
| Misc. industrial processes | Process change/incineration | 50% |
| Consumer solvents | Additional reformulation | 40% (50% in 2010) |
| AIM coatings | Additional reformulation | 40% (50% in 2010) |
| Lawn & garden | Episodic Ban | 50% |
| Recreational vehicles | Episodic Ban | 50% |
| Industrial equipment | Episodic Ban | 50% |
| Recreational marine | Episodic Ban | 50% |
| **Point Source** | | |
| Open burning | Seasonal Ban | 80% |
| Industrial surface coating | Add-on Control Levels | 90% |
| Metal product surface coating | Reformulation | 88% |
| Wood product surface coating | Reformulation | 85% |
| Point sources | Rule effectiveness improvements (80% RE to 90% RE) | -- |

*"Episodic" and "seasonal ban" controls are not creditable toward the ROP shortfall.

## Mercury Emission Estimates

EPA, as part of this analysis, also estimates the effect of CAAA provisions on airborne mercury (Hg) emissions for five separate Hg emissions sources: medical waste incinerators (MWI), municipal waste combustors (MWCs), electric utility plants, hazardous waste combustors, and chlor-alkali plants.[7] While the Integrated Planning Model (IPM) was used to generate Pre- and Post-CAAA electric utility Hg emissions estimates, data from previously conducted analyses were relied on to estimate Hg emissions from the other sources. The following section provides a description of the methods and data sources used to develop 1990 base-year Hg emissions and 2000 and 2010 mercury emission projections for the Pre- and Post-CAAA scenarios.

### *Medical Waste Incinerators (MWI)*

During the Maximum Achievable Control Technology (MACT) development process for MWI, 1990 emission estimates for hazardous air pollutants (HAPs), including mercury (Hg), were developed by back-casting 1995 emission estimates (EPA, 1996b). The back-casting was performed by adding to the 1995 database MWIs that had shut down during the 1990-1995 time period. These MWI were shut down as a result of economic considerations prompted by the adoption of strict regulations in six states (California, New Mexico, New York, Oregon, Washington, and Wisconsin). The number of MWIs in the remaining states were assumed to be the same in 1990 as in 1995.

Based on the back-casting analysis described above, EPA estimated Hg emissions of 16 tons per year (tpy) in 1995 and 50 tpy in 1990 (Cocca, 1997). Therefore, 50 tpy is selected as the base year estimate for this analysis. To estimate the Pre-CAAA scenario forecasts, it is assumed that no other states would have adopted MWI regulations between 1995 and 2010. The annual growth rates estimated by the Bureau of Economic Analysis (BEA) for health services are 2.3 percent from 1990 to 2000 and also 2.3 percent from 1990 to 2010 (Pechan, 1998). The Pre-CAAA forecasts were estimated by multiplying 16 tpy in 1995 by a 1.12 growth factor for 2000 and a 1.41 growth factor for 2010. This yielded 17.9 tpy for 2000 and 22.6 tpy for 2010 (see Table A-16).

EPA estimated a 93 to 95 percent reduction of Hg nationally for existing units in the Emission Guidelines (which incorporate the MACT standards for MWI; EPA, 1996c). A 45 to 74 percent reduction was estimated to occur within 5 years of New Source Performance Standard (NSPS) promulgation for new sources (these sources were estimated to produce 0.2 tpy without the standard by 2002). The combined effects of the NSPS/EG are approximately a 93 percent reduction (from 1995) in the years 2000 and 2010. This emission reduction estimate was used to calculate the Post-CAAA emissions for MWI (see Table A-16). No other CAAA regulatory efforts are known that would further impact emissions from MWI.

### *Municipal Waste Combustors (MWCs)*

Methods and sources of information used to estimate Hg emissions for MWC are given in a supporting document for the NSPS/EG for MWC promulgated in 1995 (EPA, 1996c). EPA estimated that there was 54 tpy emitted by MWC in 1990. Between 1990 and 1995, several factors contributed to a decline in Hg emissions from MWC: addition of air pollution controls on existing facilities; retirement of some units; a decrease in the Hg content of municipal waste being burned. EPA estimated that there were 29 tpy of Hg emitted by MWC in 1995 (EPA, 1996c). For the purposes of this analysis, it is assumed that these reductions occurred due to influences unrelated to the CAAA. It is further assumed that no additional reductions would have occurred between 1995 and 2000 (or 2010) without promulgation of the NSPS/EG.

[7] Together, these sources account for 75 to 80 percent of national anthropogenic airborne Hg emissions.

EPA further estimated that following full implementation of the NSPS/EG in 2000, national Hg emissions would be reduced to 4.4 tpy. However, the NSPS/EG was amended in 1997. As part of the amendments, MWC units with capacities less than 250 tons/day and cement kilns burning municipal waste were exempted from the NSPS/EG. Due to this exemption, EPA estimated that 87 percent of the national MWC capacity was now covered by the NSPS/EG (small units were to be covered in a later rule-making; EPA 1997c). Therefore, to estimate Post-CAAA 2000 emissions for MWC, the original 4.4 tpy estimate was divided by 0.87 to account for the exempted units. After adjusting for growth, the 5.5 tpy shown in Table A-16 was estimated for 2000.

Between 1995 and 2000, EPA (1997d) estimated that municipal solid waste combustion would increase by 7.5 percent. Therefore, the Pre-CAAA 2000 Hg emissions were estimated to be 31.2 tpy (29 tpy x 1.075). Between 2000 and 2010, growth in municipal waste combustion is estimated to be an additional 8.3 percent (EPA, 1996f). This growth factor was applied to both Pre- and Post-CAAA 2000 emission estimates to yield the year 2010 estimates.

### Electric Utility Generation

In a report to Congress which details anthropogenic mercury emissions, the EPA estimated that total 1990 mercury emissions from utility boilers was 51.3 tpy (EPA, 1996g). The Integrated Planning Model (IPM) was used to estimate emissions resulting from electric power generation in 2000 and 2010 using different control scenarios to reflect the Pre and Post-CAAA scenarios. The results generated by the IPM are presented in Table A-16 (EPA, 1996g). The scenarios modeled did not include any assumptions about the effects of any MACT standard to be promulgated by EPA in the future. Any differences in emissions between the control scenarios are due to shifts in operation between units or fuel changes predicted by IPM (including ash content of the coals as well as switching to natural gas). The modeled emission estimates are based on information available at the time, and if the same analysis were performed today, with currently available inputs, the results could be different.

### Hazardous Waste Combustion

Pechan-Avanti received preliminary draft data from Industrial Economics on the benefits of the MACT Standard for hazardous waste combustors (Yates, 1999). No information was available on growth for this source category, although the analyses conducted to date indicate that there might be a slight contraction (e.g., five percent) of the category between 1990 and 2000 (or 2010; Yates, 1999). Therefore, it was assumed that there would not be any growth from the 1990 baseline. Also, it was assumed that emission reductions would not occur until after 2000, since the standard will not be fully-implemented until 2002.

### Chlor-alkali Plants

EPA documents estimate mercury emissions from mercury-cell chlor-alkali plants to be 9.8 tpy (EPA, 1998b; EPA, 1998c) in 1990, and 7.1 tpy in 1994 (EPA, 1996h; EPA, 1996i). The number of facilities which use the mercury-cell process has been declining since reaching a peak of 35 in 1970. By 1995, there were 14 facilities, one of which has converted to a membrane process (no mercury emissions), and another of which has ceased operation, leaving 12 facilities in 1999. In the past, closures or process changes have been due to economics rather than regulations. There are no existing CAAA programs which influence mercury emissions from this source. A MACT standard is currently in the development process, and will not be promulgated for a year or more (Rosario, 1999; Dungan, 1999).

The MACT standard, when issued, will focus on both point source and fugitive emissions. There are generally three point sources at mercury-cell chlor-alkali plants which emit mercury: hydrogen vents, end-box vents, and mercury recovery vents. Point source controls being considered for the MACT standard are a combination of a cooling device and either a molecular sieve, or a carbon adsorption system. The level of control is yet to be determined, pending

further investigation into the typical concentrations of mercury found at the vents. The MACT standard for fugitive mercury emissions from the cellhouse has not been identified at this point, and investigations and meetings are planned for the near future in order to quantify typical fugitive emissions (Rosario, 1999).

Assuming that no additional plants will close or convert between the time of this writing and the end of 2000, and that mercury emissions from the 12 remaining sources have remained constant since 1994, and will continue to remain constant, 2000 emissions for both the Pre and Post-CAAA scenario are estimated to be 6.0 tpy. This is based on the 7.1 tpy reported for 1994, minus the 1994 emissions for the two facilities which no longer have the potential to emit mercury (Georgia-Pacific in Bellingham, WA which ceased operations in 1999 and is reported to have emitted 0.65 tpy in 1994, and LCP Chemicals in Reigelwood, NC which converted in 1999 and is reported to have emitted 0.55 tpy).

Future mercury emissions from mercury-cell chlor-alkali plants are assumed to continue to decline, following the trend of plant closing and conversions. Assuming that the decline in the number of mercury-cell facilities has been linear since 1970, and will remain linear in the future, a linear regression was used to estimate the number of facilities in 2010. Assuming that there will be 12 facilities in 2000, the linear regression ($r^2$ = 0.98) indicates a decline of approximately 0.75 facilities per year, which, when extrapolated to future years, results in an estimate that there will be approximately four mercury-cell facilities still in operation in 2010. Assuming that emissions will decline at a rate proportional to that of the number of facilities, emissions are estimated to decline from 6.0 tpy in 2000 to 2.0 tpy in 2010. This estimate was used as the Pre-CAAA scenario estimate for 2010 (see Table A-16).

In order to account for the possible effect of an un-promulgated MACT standard on Hg-cell chlor-alkali plants operating in 2010, the limited information available was reviewed. This information is principally made up of a brief summary of test data submitted by the operating facilities. At some point in the future after more testing has been performed, the MACT standard and estimates of the resulting reductions will be made publicly available (Rosario, 1999).

Currently, there are facilities which control mercury emissions, some of which control the emissions to near or below what the MACT limit may eventually be. Emissions from these sources are unlikely to be reduced as a result of the MACT standard being promulgated. However, emissions from the plants required to upgrade or add control systems as a result of the MACT standard being promulgated, will be reduced in future years. Based on the limited data available, and assuming that the test data available are typical of what would be reported by the facilities for which no valid test data are available, the overall reduction is estimated to be approximately 35 percent. This estimate is reflected in the Post-CAAA emission estimate for 2010 in Table A-16. The difference between 2010 emissions with and without the CAAA will be greater if the number of mercury-cell chlor-alkali plants remains steady at present levels rather than declining at a linear rate, as is assumed here.

**Table A-16**
**Airborne Mercury Emission Estimates**

| Source Category | 1990 Emissions (tons) | 2000 Emissions (tons) Pre-CAAA | 2000 Emissions (tons) Post-CAAA | 2000 Emissions (tons) Diff. | 2010 Emissions (tons) Pre-CAAA | 2010 Emissions (tons) Post-CAAA | 2010 Emissions (tons) Diff. |
|---|---|---|---|---|---|---|---|
| Medical Waste Incin. | 50 | 17.9 | 1.3 | 16.6 | 22.6 | 1.6 | 21.0 |
| Municipal Waste Comb. | 54 | 31.2 | 5.5 | 25.7 | 33.8 | 6.0 | 27.8 |
| Electric Utility Generation | 51.3 | 63.0 | 61.1 | 1.9 | 68.5 | 65.4 | 3.1 |
| Hazardous Waste Comb. | 6.6 | 6.6 | 6.6 | 0 | 6.6 | 3.0 | 3.6 |
| Chlor-Alkali Plants | 9.8 | 6.0 | 6.0 | 0 | 2.0 | 1.3 | 0.7 |
| **Total CAAA Benefits (Reductions)** | | | | **44.2** | | | **56.2** |

## Uncertainties in the Emission Estimates

This discussion is organized according to the three major sources of uncertainty in the emissions inventory and emission projections: the base year emission estimates, economic growth forecasts, and future year control assumptions.

### *Base Year Emission Estimates*

Of the pollutants covered in this analysis, the most certain emission estimates are those for $SO_2$. $SO_2$ is generated during combustion of any sulfur-containing fuel and is emitted by industrial processes that consume sulfur-containing raw materials. Because sulfur emissions are directly related to the fuel sulfur content, as long as fuel usage and fuel sulfur content are measured, $SO_2$ emissions, prior to the imposition of controls, can be precisely estimated within a narrow range. Electric utilities emit about 70 percent of the $SO_2$ in the United States. Under existing utility industry regulations, fuel consumption and sulfur content of fuels are regularly reported to DOE. Recent comparisons of Continuous Emission Monitoring (CEM) data for $SO_2$ with estimates based on $SO_2$ emission factors and fuel consumption for a sample of plants showed that the two techniques produced emission estimates within an average of 8 percent at a State level. The difference is due, in part, to higher fuel consumption numbers reported by CEM systems, as a result of the missing data substitution requirements of the acid rain program (Schott, 1996).

As part of the GCVTC emission inventory (for 11 Western States), uncertainty estimates were developed for key source sectors, representing over 70 percent of the emissions (Balentine and Dickson, 1995). $SO_2$ sources examined included copper smelters and motor vehicles. The uncertainty estimate calculated for $SO_2$ emissions from copper smelting is $\pm$ 50 percent. Diesel and gasoline vehicle emissions have uncertainty estimates of a factor of $\pm$ 1.5. Most of this uncertainty is due to the variability in the sulfur content of the fuels.

After $SO_2$, the next most certain emission estimates are probably the $NO_x$ values. Like $SO_2$, $NO_x$ is a product of fuel combustion. However, there are two $NO_x$ sources in fossil-fuel combustion (Seinfeld, 1986). The first is the oxidation of atmospheric molecular nitrogen at the high temperatures of combustion. $NO_x$ formed by this route is referred to as thermal $NO_x$. The second source is the oxidation of nitrogen-containing compounds in the fuel. $NO_x$ formed by this path is called fuel $NO_x$. Since $NO_x$ formation is somewhat more complicated than $SO_2$, emission estimates are more variable, and uncertain, as well.

A comparison of $NO_x$ emissions based on CEM data and $NO_x$ emissions based on AP-42 emission

factors for a sample of utilities in Louisiana resulted in a difference of 22 percent between the two methods. The difference is attributable to improved emission factors resulting from the use of CEM data, rather than one-time stack tests or AP-42 emission factors (Schott, 1996).

The level of uncertainty in primary $PM_{10}$ emission estimates varies widely by source category. The largest component of the 1990 $PM_{10}$ emission estimates is fugitive dust sources, which include paved and unpaved roads, construction activity, agricultural tilling, and windblown dust. The GCVTC study estimated the uncertainty for unpaved road emissions to be a factor of $\pm$ 4.0. The estimated uncertainty for $PM_{2.5}$ emissions from paved road dust is a factor of $\pm$ 1.8.

$PM_{10}$ emission estimates for large point sources such as utility boilers would be expected to be less uncertain than the fugitive dust source estimates, because these stacks are typically controlled using baghouses or electrostatic precipitators, with frequent stack tests to ensure compliance with regulations.

VOC emissions estimates are uncertain because organics are emitted both as a product of fuel combustion and through evaporation. Evaporative emissions are difficult to quantify because of the associated measurement problems. The GCVTC study estimated the VOC emissions uncertainty for motor vehicles to be a factor of $\pm$ 1.5.

Estimates of emissions from solvents and other evaporative VOC sources are probably even more uncertain than the motor vehicle VOC emission estimates. Emission estimates for such sources typically assume that all of the organic content of solvent ultimately evaporates. However, usage patterns determine what time of year these solvents are released to the atmosphere, and emissions that occur outside the ozone season may not influence ozone levels. Solvent emission estimates used in this study are based on a national material balance. Solvent emission estimates made by State and local air pollution control agencies for SIPs typically use per capita emission factors to estimate solvent emissions. This will produce different emission estimates than used in this study.

## Growth Forecasts

The 2000 and 2010 emission estimates in this analysis are influenced by the projected changes in pollution-generating activity. Inherent uncertainties and data inadequacies/limitations exist in forecasting growth for any future period. As a result, it was necessary in this analysis to use indicators of growth that may not directly correlate with changes in the factors that influence emissions. In the previous chapters of this report, alternative growth forecasts were presented for major sectors, and the implications of these alternative forecasts were noted.

The best indicator of pollution-generating activity is fuel use or some other measure of input/output that most directly relates to emissions. The key BEA indicator used in this analysis, GSP, is closely linked with the pollution-generating activity associated with many manufacturing industry processes (iron and steel, petroleum refining, etc.). However, a good portion of industrial sector emissions are from boilers and furnaces, whose activity is related to production, but not as closely as product output. Activities such as fuel switching may produce different emission patterns than those reflected in the results of this study. The modeling methods applied in this study would only capture such effects for electric utilities, but not for the industrial sector.

While it is expected that there will be energy efficiency improvements in the 1990 to 2010 forecast horizon, potential energy efficiency improvements have not been incorporated in the growth factors. The U.S. Department of Energy currently estimates that energy intensity – the amount of energy used for each dollar of output in the economy – will decline by 1 percent per year through the time horizon of this study. If these potential energy efficiency improvements had been incorporated in the 2000 and 2010 emission projections, then both the Pre-CAAA and Post-CAAA emission estimates would be lower than those presented in this report.

In general, emissions from the point, area, and nonroad engine/vehicle sectors are projected to 2000 and 2010 in this analysis based on BEA GSP by State and industry, and population projections by State. Source categories were matched with surrogate activity indicators that represent proxies for emission growth. The uncertainty of the growth forecasts used in this analysis is attributable to two factors: the uncertainty of the projections data used, and the use of surrogate activity levels to estimate future emission levels.

Throughout this analysis, efforts were made to identify potential sources of growth surrogates and to evaluate the impacts of alternative growth factors on emission projections. The impact of alternative growth factors on the emission projection results of this study vary by source category and pollutant. For example, point source emissions from chemical manufacturing would increase at an average annual rate from 0.9 (BEA GSP) to 2.6 (E-GAS/WEFA) percent per year between 1990 and 2010 depending on the activity factor used as a surrogate for emissions growth. In the nonroad vehicle sector, emissions from aircraft are projected to grow from 2 to 5 percent per year for the 1990-2010 time period, depending on whether GSP or landing and takeoff operations (LTO) data are used as the surrogate growth indicator. Growth projections for the railroad industry can range from 0.3 percent to 4.4 percent depending on whether the growth variable is ton-miles, fuel use, GSP, or earnings. In this analysis, BEA earnings data were used to represent growth in emissions for this industry because it was possible to differentiate growth rates at the State level, and because the data were available for the relevant years of this analysis. In future years, industry analysts predict lower prices per ton-mile in response to increased competition for rail traffic. To the extent that future predictions of lower rail transport prices occur as railroad transport increases, miles traveled may be a more accurate activity level surrogate for emissions than earnings. The outlook for the railroad industry is uncertain, and emissions may be over- or understated for the 2000 and 2010 scenarios depending on future industry conditions.

Each of the available variables for projecting emissions has advantages and disadvantages with respect to this analysis. The Agency chose growth surrogates for this analysis based on EPA guidance; the availability of data for 1990, 2000, and 2010; geographic detail of projections data; coverage relative to the detail of the base year inventory; and the appropriateness of using the variable as a measure of emissions growth. For this analysis, BEA provided a consistent data set that could be applied across source categories and across States.

## Future Year Control Assumptions

The uncertainties associated with future year control assumptions can be grouped in three types: (1) will the control programs be adopted; (2) will control programs be as effective as estimated in this analysis; and (3) will technological shifts produce enough changes in emission patterns to affect future year results?

On the first and second issues, there have been eight years of progress in implementing the CAAA provisions, and emission trends estimates have shown that significant emission reductions have occurred in this period (EPA, 1997a). Relative to expectations when the CAA was passed, $SO_2$ emission reductions have occurred at a faster rate than originally anticipated, while some of the VOC and $NO_x$ emissions have been less than originally anticipated, as many vehicle emission inspection programs have been delayed. By 2000, though, any short run perturbations may have a negligible effect on overall emission benefits. Future implementation depends on decisions that EPA makes about Federal rules, such as commercial/consumer solvent rules and MACT standards.

Also on the second issue, concerns about the ability of regulations to achieve expected reductions as implemented have resulted in some new programs and techniques for assuring that new programs are effective. Rule effectiveness discounting is applied to stationary source controls (other than those for $SO_2$) in this analysis to account for control equipment malfunctions and downtime, unrecognized control

responsibility, and gross noncompliance. In addition, much more continuous emission monitoring is now required for major $SO_2$ and $NO_x$ sources to ensure that emission limits are met, so many point source emission reductions should be verifiable. Verifying area source emission reductions is much more difficult. Experiments using remote sensing, tracers, and other real-world measurement tools are being performed to better assess the effectiveness of motor vehicle, nonroad engine, and solvent emission control initiatives.

On the third issue, any major technological improvements to create lower-polluting systems by 2010 could influence the emission forecasts, and would be expected to produce more emission benefits than have been estimated in this study. In-depth analyses of two sectors, petroleum refining and motor vehicles, as part of the Section 812 prospective analysis found no major technology changes that would significantly alter emission estimates in these two important sectors to be likely before 2010. For the motor vehicle industry, research has been focused on battery-powered electric vehicles since the California LEV program requirement for zero emission vehicles (ZEVs) was announced. However, these electric vehicles will be unlikely to capture more than 10 percent of LDV sales by 2010.

The refinery sector study identified the significant post-1990 technological trends in this industry to be (1) continued investment in downstream processing of petroleum; (2) increasing refinery capacity utilization; (3) continued improvement in refining process control and catalyst use; and (4) steady capital investment in stationary source controls. None of these trends is expected to significantly change refinery emission rates in 2010 apart from the further investment in stationary source controls (to meet MACT standards, for instance) and these are accounted for in the post-CAAA scenario emission estimates.

Air pollution control regulations will be technology forcing in that many VOC containing solvents will be re-formulated to low-VOC solvents or replaced with water-based substitutes. The most significant programs associated with the CAAA that impact VOC emissions from the solvent cleaning source category are being implemented through local regulations as part of ozone attainment plans. Chief among these is the revised SCAQMD Rule 1171 (SCAQMD, 1996). In the revision of this rule, SCAQMD requires the use of low-VOC solvents (e.g., aqueous) for all regulated sources (e.g., those who are not regulated under Rule 1122, which has equipment instead of solvent requirements). SCAQMD is still working on issues surrounding applicability of Rules 1171 and 1122, but has initially estimated VOC emission reductions of 46 percent associated with the revisions to Rule 1171 (SCAQMD, 1996).

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# Direct Costs

Appendix B

## Introduction

In this appendix, we present the estimation of direct compliance costs associated with the Clean Air Act Amendment programs under Title I through V that control the following criteria pollutants:

- Volatile organic compounds (VOCs)
- Oxides of Nitrogen ($NO_x$)
- Carbon monoxide (CO)
- Sulfur dioxide ($SO_2$)
- Particulate matter with an aerodynamic diameter of 10 microns or less ($PM_{10}$)
- Particulate matter with an aerodynamic diameter of 2.5 microns or less ($PM_{2.5}$)

The first section of the appendix provides a general overview of our methodology for estimating direct compliance costs and the models used in the analysis.[1] The following section presents costs first by emission sources and then by CAAA title. Cost by emission source reviews the specific costing approach (i.e., source-specific cost equations or operating cost estimates), sources of data, and emission control scenarios applied to five regulated sectors and ozone nonattainment areas. Costs are also presented by CAAA title, where the cost components (i.e., the emission sources and provision) are identified for Titles I through V. In the following section, we discusses several additional issues related to fully accounting for the broader economic consequences of reallocating resources to the production and use of pollution abatement equipment (i.e., estimating social costs versus direct compliance costs). We conclude with a discussion of analytic limitations and characterizations of the potential impact of several key uncertainties of cost estimates.

[1] This appendix is a condensed version of more detailed reports completed under EPA's direction. For more details see Pechan, 1998.

## Summary of Methods

We use two modeling approaches to calculate cost estimates under Post-CAAA control scenarios in the projection years, 2000 and 2010. The control assumptions (i.e. emissions scenarios) used as inputs in the models are consistent with the assumptions used in the analysis of both emissions projections and benefits. The cost data used as parameters in these models includes results and information from EPA regulatory impact assessments (RIAs), background information documents (BIDs), regulatory support documents, and Federal Register notices.

### *ERCAM Model*

We use ERCAM to estimate the costs associated with regulating particulate matter (PM), volatile organic compounds (VOCs), and non-utility oxides of nitrogen ($NO_x$).[2] The model is essentially a cost-accounting tool that provides a structure for modifying and updating changes in inputs while maintaining consistency with the emission and cost analyses. Cost scenarios and assumptions are developed for source categories (e.g., point, area, nonroad, and motor vehicle sources) and in response to specific provisions and emission targets. The model estimates costs based on inputs such as cost per ton, source-specific cost equations, incremental production, and operating cost estimates. For this analysis, we collected data and inputs from information presented in regulatory impact assessments (RIAs), background information documents (BIDs), regulatory support documents, and Federal Register notices.

[2] This model was developed by E. H. Pechan & Associates, Inc. to facilitate EPA's analysis of emissions control.

### IPM Model

We rely on a utility planning model, Integrated Planning Model (IPM), to estimate the costs of $NO_x$ and $SO_2$ controls for electric utilities. IPM is a linear program/optimization model that can estimate costs and emissions based on key constraints and parameters. One of the significant advantages to this model is that it provides the analysis with flexibility in the level of detail for characterizing constraints and economic assumptions. In this analysis, the model estimates compliance costs based on assessing the optimal mix of pollution control strategies subject to a series of specified constraints. Key inputs to the model include targeted emissions reductions (on a seasonal and annual basis), characteristics of control technology, and economic parameters. The characteristics of control technology examines operational costs and constraints associated with the performance of existing and new utility generating units. Examples of inputs for existing units include plant capacity, fuel usage rates, fixed and variable O&M costs. For new utility generating units, inputs are generally associated with unit characteristics such as capacity and costs of capital. Economic assumptions include the projected electric industry growth, changes in seasonal and regional demand, and forecasts of fuel prices.

### Additional Methods

We estimate non-utility $SO_2$ emission control costs for point sources by applying source-specific cost equations for flue gas desulfurization (FGD)/scrubber technology to affected sources in 2000 and 2010. While we do not explicitly model CO attainment costs, we include in the analysis the costs of programs designed to reduce CO emissions, such as oxygenated fuels and a cold temperature CO motor vehicle emission standard.

### Annualization of Costs

The costs presented in this analysis are total annualized costs (TAC) in 2000 and 2010. Annualized costs include both capital costs, such as costs of control equipment, and operation and maintenance (O&M) costs.[3] They do not represent actual cash flow in a given year, but rather are an estimate of average annual burden over the period during which firms will incur costs (i.e., equipment life). In annualizing costs, we convert total capital investment, plus O&M and other re-occurring costs, to a uniform series of per-year expenditures over a given time period. The discounted sum of these annual expenditures is equal to the net present value of total costs incurred over the time period of this analysis.[4]

## CAAA Costs

We estimate costs of implementing the Clean Air Act Amendments under two Post-CAAA scenarios, 2000 and 2010. The estimates, therefore, represent differences in costs between pre- and post-scenarios in each of the two years. The cost estimates for implementing Titles I through V of the Clean Air Act Amendments are $19 billion under the Post-CAAA 2000 scenario and $27 billion under the Post-CAAA 2010 scenario. All costs are in 1990 dollars. This appendix presents the costs first by source and then by title.

This section summarizes our costing methods and results for the following CAAA regulated sectors:

- Industrial point sources
- Electric utilities
- Nonroad engines and vehicles
- Motor vehicles
- Area sources
- Ozone nonattainment areas

Compliance with the CAAA provisions for motor vehicles is the single largest cost component: $9 billion for the Post-CAAA 2000 scenario, and $12 billion for Post-CAAA 2010. The costs of compliance

[3] For a few VOC source categories, we estimate that capital investment will not be necessary; for these sources, compliance costs reflect O&M costs only.

[4] We re-calculate the control cost estimates from regulatory documents that use a seven or ten percent discount rate so that the costs will be consistent with the five percent discount rate assumption used in this analysis. We also calculate cost using three percent and seven percent discount rates, as sensitivity tests: for detail see the discussion of uncertainty later in this appendix.

for industrial point sources, utilities, and area sources are somewhat smaller; they range from $3 to $5 billion dollars each. Table B-1 summarizes the cost estimates by year and emissions source.

**Table B-1**
**Summary of Cost Estimates by Emissions Source**

| Sector/Pollutant | Annual Cost (million 1990 dollars) Post-CAAA 2000 | Post-CAAA 2010 |
|---|---|---|
| **Total Non-utility Point** | **$ 2,900** | **$ 3,400** |
| Non-utility Point/VOC | 900 | 960 |
| Non-utility Point/$NO_x$ | 1,700 | 2,100 |
| Non-utility Point/Non-VOC MACT[1] | 310 | 320 |
| **Utility/$SO_2$ and $NO_x$** | **$ 3,100** | **$ 4,600** |
| **Non-Road Engines/Vehicles** | **$ 100** | **$ 400** |
| **Motor Vehicles** | **$ 9,100** | **$ 12,300** |
| **Total Area Sources** | **$ 2,900** | **$ 3,300** |
| Area/VOC | 920 | 1,000 |
| Area/$NO_x$ | 16 | 18 |
| Area/PM | 1,900 | 2,200 |
| **Progress Requirements** | **$ 1,200** | **$ 2,500** |
| **Permits[2]** | **$ 300** | **$ 300** |
| **TOTAL** | **$ 19,400** | **$ 26,800** |

Notes:
1 Costs reflect estimates of annualized costs from final rules. Source categories are not modeled in ERCAM-VOC because the National Emission Standards for Hazardous Air Pollutants (NESHAPs) are associated with non-VOC HAP emission reductions, and are therefore not included in the Post-CAAA 2000 and 2010 inventories.
2 These costs include costs only for State-implemented permitting programs. We exclude the costs of Federally-implemented programs since all Title V permit programs will be State-run in 2005.

## *Industrial Point Sources*

Industrial point sources are non-utility sources that are large enough to be included in the 1990 emissions data base as individual sources of emissions. To determine the level of air pollution controls necessary for reducing emissions under the 2000 and 2010 Post-CAAA scenarios, we apply the following CAAA controls to point source emission inventory:

- Title III 2-year and 4-year MACT standards for VOCs
- Title I CTGs for controlling VOCs
- Title I VOC and $NO_x$ RACT requirements in ozone NAAs
- A 0.15 lbs/MMBtu $NO_x$ cap on fuel combustors of 250 MMBtu per hour or above in the OTAG 37-State region
- Ozone NAA rate-of-progress requirements

To estimate the quantity and type of VOC controls, we apply point source Title I RACT and

CTGs requirements in areas according to ozone nonattainment classification. The Clean Air Act requires VOC controls in moderate and above ozone nonattainment areas (NAA) and throughout the ozone transport region (OTR). Existing controls are taken into consideration in our determination of which CAA-mandated controls are necessary to limit projected emissions. We use a threshold of ten percent efficiency for this determination. We calculate costs for new control if the existing control is less efficient than the model control by more than ten percent (i.e. emissions changes of less than ten percent are assumed to be *de minimus* and are not included in the cost estimate).

To estimate the quantity and type of $NO_x$ controls, we apply these controls to the point source inventory on a year-round basis. The ozone nonattainment provisions of Title I require installation of RACT-level controls for major stationary sources of $NO_x$ located in marginal and above NAAs and the northeast OTR. We determine affected source sizes according to ozone nonattainment classifications. The analysis applies the 0.15 lbs/MMBtu $NO_x$ limit to industrial boilers at or above 250 MMBtu per hour in the Ozone Transport Assessment Group (OTAG) region to approximate the effects of $NO_x$ initiatives under consideration. We also account for Title I requirements that include the application of Level 2 controls in the OTAG region.

## Cost Approach

We use ERCAM-VOC and ERCAM-$NO_x$ models for generating cost estimates. Model inputs include costs per ton and incremental cost estimates derived from RIAs and from control measure information provided by EPA, States, industry, and other agencies.[5] Using the projected 2000 and 2010 emission inventories, we also estimate costs by applying cost equations to the following individual source categories:

- Adipic and nitric acid manufacturing plants
- Cement manufacturing
- Gas turbines
- Glass manufacturing
- Industrial boilers
- Internal combustion engines
- Iron and steel mills
- Medical waste incinerators (MWIs)
- Municipal waste combustors (MWCs)
- Process heaters

For some source categories, capital and O&M cost estimates are available in the literature for two or more source sizes typical to that category. For these cases, we apply size-specific cost equations. Operating characteristics and source size, both of which influence the ease of retrofit, reduction performance, and control costs, are major factors in determining costs of controls. Although site specific characteristics can affect the overall cost, this type of information is not available in the emission inventory. Therefore we model costs based on a "typical" set of controls.

For source categories with insufficient data, we estimate annual costs for controls using average cost per ton values from the ACTs, instead of size specific cost equations. These values do not account for economies of scale or variations in capacity factor, which generally impact the cost per ton of pollutant reduced.

## Recovery Factor

ERCAM-VOC and ERCAM-$NO_x$ cost equations use a five percent discount rate and a 15-year equipment life, or a capital recovery factor (CRF) of 0.096. To calculate the capital recovery factor for converting capital charges to equivalent annual costs, we use the following formula:

$$CRF = [i * (1 + i)^n] / [(1 + i)^n - 1]$$

where i = pre-tax marginal annual rate of return (discount rate), and
n = equipment economic life (in years).

[5] The Agency bases cost effectiveness values for rules that have not yet been proposed on engineering judgement and technology transfer from other categories.

To obtain annual costs, we use the following algorithm:

$$CRC = CRF * Capital\ Costs,$$

where CRC = capital recovery cost (or annualized capital cost).

## Cost Results

Table B-2 summarizes estimated point source VOC control costs. we estimate costs to be approximately $901 million in 2000; of that total, $421 million will be Title I VOC controls costs and $480 million will result from the Title III MACT Standards. In 2010, the total annual cost of point source VOC controls is approximately $962 million: $440 million in Title I controls and $521 million in Title III controls.

Table B-3 summarizes the point source $NO_x$ control costs under the 2000 and 2010 Post-CAAA scenarios. OTAG region costs under the 2000 Post-CAAA scenario total $1.6 billion, increasing to $2.1 billion by 2010. Point source $NO_x$ control costs in the rest of the nation are $21 million under the 2000 Post-CAAA scenario and $22 million under the 2010 Post-CAAA scenario. Nationwide, ICI boilers bear the majority of point source $NO_x$ control costs, which account for seventy-nine percent of the total costs in 2010.

**Table B-2**
**Point Source VOC Cost Summary**

| | Annual Costs (million 1990 dollars)[1] | |
|---|---|---|
| **Source Category** | **Post-CAAA 2000** | **Post-CAAA 2010** |
| **National Rules** | | |
| Marine vessel loading: petroleum liquids | $ 20 | $ 30 |
| TSDFs | Less than 0.1 | Less than 0.1 |
| **New CTGs (moderate)** | | |
| Printing - lithographic | (0.7) | (0.7) |
| SOCMI distillation | 0.1 | 0.1 |
| SOCMI reactor | 1.9 | 2.2 |
| **Non-CTG and Group III CTG RACT (moderate and above)** | | |
| Automobile surface coating | 210 | 220 |
| Bakeries | 0.9 | 1.1 |
| Beverage can surface coating | 47 | 47 |
| Carbon black manufacture | 1.2 | 1.3 |
| Charcoal manufacturing | 0.0 | 0.0 |
| Cold cleaning | 17 | 18 |
| Fabric printing | 22 | 23 |
| Flatwood surface coating | 20 | 21 |
| Leather products | 1 | 1.1 |
| Metal surface coating | 51 | 57 |
| Organic acids manufacture | 1.7 | 2.0 |
| Paint and varnish manufacture | 2.5 | 2.8 |
| Paper surface coating | 5.5 | 5.5 |
| Plastic parts surface coating | 5.1 | 5.3 |
| Rubber tire manufacture | 1.4 | 1.4 |
| SOCMI processes - pharmaceutical | 3.7 | 4.1 |
| Whiskey fermentation - aging | 0.2 | 0.2 |

| Source Category | Annual Costs (million 1990 dollars)[1] Post-CAAA 2000 | Post-CAAA 2010 |
|---|---|---|
| **CTG RACT (marginal and above)** | | |
| Cellulose acetate manufacture | 1.5 | 1.6 |
| Dry cleaning - Stoddard solvants | 0.1 | 0.1 |
| In-line degreasing | (0.3) | (0.3) |
| Open top degreasing | (1.0) | (1.2) |
| Printing - letterpress | 0.5 | 0.5 |
| Terephthalic acid manufacture | 2.3 | 2.5 |
| Vegetable oil manufacture | Less than 0.1 | Less than 0.1 |
| **Total Title I Costs** | $ 420 | $ 440 |
| **NESHAP** | | |
| **Benzene NESHAP** | $ 0.2 | $ 0.2 |
| **2-Year MACT (national):** | | |
| **Dry Cleaning - PCE** | 2.2 | 2.7 |
| **SOCMI HON:** | | |
| Chemical manufacture | 12.0 | 13.0 |
| SOCMI - process vents | 2.1 | 2.4 |
| SOCMI fugitives | (3.9) | (4.5) |
| SOCMI processes | 22 | 26 |
| VOL storage | 1.5 | 1.7 |
| **4-Year MACT (national)** | | |
| Aerospace industry | 3.5 | 4.7 |
| Coke Oven Batteries | 21 | 21 |
| Gasoline distribution - Stage I | 12 | 13 |
| Halogenated solvent cleaning | (8.5) | (9.1) |
| Marine vessel loading: petroleum liquids[2] | 17 | 20 |
| Petroleum refineries: other sources not distinctly listed | 40 | 45 |
| Polymers and Resins Group I | 110 | 130 |
| Polymers and Resins Group II | 4.3 | 5.0 |
| Polymers and Resins Group IV | 5.3 | 6.7 |
| Printing and Publishing | 200 | 210 |
| Shipbuilding and ship repair | 0.4 | 0.5 |
| Wood furniture surface coating | 37 | 38 |
| **Total Title III Costs** | $ 480 | $ 520 |
| **Total Point Source VOC Control Costs (Title I and Title III)** | **$ 900** | **$ 960** |

Notes:

[1] Control costs reflect growth projections and CAAA control assumptions relative to a 1990 baseline.

[2] The costs for the joint MACT/RACT rule for marine vessel loading are allocated between Title I and Title III based on the 58 percent/42 percent distribution in the addendum to the final rule (EPA, 1995b).

**Table B-3**
**Point Source $NO_x$ Summary**

| | Annual Costs (million 1990 dollars) | |
|---|---|---|
| **Source Category** | **Post-CAAA 2000** | **Post-CAAA 2010** |
| **RACT (outside of OTAG Region)** | | |
| Adipic and Nitric Acid Manufacturing | $ <0.1 | $ <0.1 |
| Cement Manufacturing | 1.7 | 1.7 |
| Gas Turbines | 0.7 | 0.7 |
| Glass Manufacturing | 3.1 | 3.2 |
| ICI Boilers | 14 | 15 |
| Internal Combustion Engines | 0.8 | 0.8 |
| Iron & Steel Mills | <0.1 | <0.1 |
| Waste Combustors | 0.1 | 0.1 |
| Process Heaters | 0.8 | 0.8 |
| **Subtotal (RACT outside of OTAG Region)** | **$ 21** | **$ 22** |
| **RACT/OTAG Level 2 (OTAG Region)** | | |
| Adipic and Nitric Acid Manufacturing | $ 31 | $ 35 |
| Cement Manufacturing | 97 | 110 |
| Gas Turbines | 18 | 28 |
| Glass Manufacturing | 38 | 41 |
| ICI Boilers | 1,200 | 1,700 |
| Internal Combustion Engines | 190 | 190 |
| Iron & Steel Mills | 2.5 | 2.4 |
| Waste Combustors | 10 | 12 |
| Process Heaters | 21 | 23 |
| **Subtotal (RACT+OTAG Level 2+0.15 Cap)[1]** | **$ 1,600** | **$ 2,100** |
| **Total Point Source $NO_x$ Control Costs** | **$ 1,700** | **$ 2,100** |

Notes:
[1] The 0.15 lbs/MMBtu cap on fuel combustors of 250 MMBtu per hour and above is only applied under the 2010 Post-CAAA scenario.
* Totals may not add due to rounding.

### *Utility Sources*

The electric power industry is comprised of entities that generate and sell electricity under two types of conditions: (i) under firm contracts to electric utilities; (ii) directly to consumers as electric utilities. These entities include businesses, governmental agencies, and cooperative organizations. In this analysis, we include only independent power producers and cogeneration units in the contiguous United States that report to the North American Electricity Reliability Council (NERC).[6] We exclude a large number of electric utilities that simply distribute power since those facilities are unlikely to directly face CAAA regulations.

### Scenarios

Our assumptions of electricity demand are based on NERC's 1994 generation forecast with a slight downward adjustment to reflect expected changes in demand due to the Administration's Climate Change Action Plan. In general, we expect that the industry will respond to CAAA regulations by adjusting the mix of fuel types for future generation capacity (i.e., increasing electricity generation by combined cycles and decreasing use of combustion turbines), rather than significantly altering production levels. Consequently, modeled differences in total generation capacity for Pre- and Post-CAAA scenarios are also relatively small and demand for electricity under both scenarios is essentially the same.[7]

The predominant emitters of air pollutants by the electric power industry are generation units that use fossil fuels. This includes coal-fired steam, oil/gas-fired steam, oil/gas combustion turbine, and natural gas combined cycle units. Under the Pre-CAAA regulatory scenario, we fix standards at prevailing 1990 levels. We assume that existing controls of carbon monoxide and particulate matter remain constant in both Pre- and Post-CAAA scenarios. The Post-CAAA regulatory scenario reflect standards that target these generation units and their emissions of $SO_x$ and $NO_x$.[8]

In the Pre-CAAA scenario developed for utility $SO_x$ emissions, we assume existing units satisfied State Implementation Plan (SIP) requirements which specify unit-specific permits for individual boilers or plants. Typically, these permits restrict sulfur-content levels of coal or fuel oil that are burned. In addition, new coal-fired units must continue to meet the New Source Performance Standards (NSPS) set in 1978. In the Post-CAAA scenario, units subject to compliance with Title IV Acid Rain Allowance Trading program are existing units that burn fossil fuels and are over 25 megawatts (MW) and all new units that burn fossil fuels (regardless of size). Lastly, compliance with the trading program is phased in by 2000.

Under the Pre-CAAA scenario, we do not model $NO_x$ controls on existing sources. New sources must meet either existing New Source Performance Standards (NSPS) or Best Available Control Technology (BACT) standards, whichever is lower. In the Post-CAAA scenario, existing sources of $NO_x$ emissions are regulated: (i) under Title I, where existing units comply with RACT requirements in ozone transport regions (OTR) and non-attainment areas (NAA), and (ii) under Title IV, where coal-fired units must meet with phased requirements by 2000.[9] New sources must meet the most stringent standard among the following, NSPS requirements of Title I,

[6]We do not include trust territories, Alaska, and Hawaii in this analysis. Trust territories are not directly covered by the CAA. With respect to Alaska and Hawaii, these States generate such small amounts of power that excluding them does should not have a significant effect on the results of this analysis.

[7]Demand is 3.0 trillion kilowatt hours (kWh) in 2000 and 3.6 trillion kWh in 2010.

[8]Under both regulatory scenarios, we do not account for the costs of regulating air toxics. The Amendments mandate the Agency to evaluate the human health impact of utilities' air toxics emissions. In the case where harmful effects are determined, the Agency is required to promulgate regulation of their emissions. The Agency, however, has not reached any conclusions on air toxic emissions from power plants.

[9]The OTR consists of New England, New York, Pennsylvania, New Jersey, Delaware, Maryland, District of Columbia, and sections of northern Virginia.

BACT requirements of Title I, or Title IV requirements. We summarize the Post-CAAA scenarios for the control of these two pollutants are summarized in Table B-4.

**Table B-4**
**Differences in the Control of Utility $NO_x$ and $SO_x$ for the Pre-CAAA and the Post-CAAA Regulatory Scenarios**

| Pollutant | Pre-CAAA | Post-CAAA |
|---|---|---|
| **$SO_x$** | Existing units: Comply with State Implementation Plan (SIP) requirements prevailing in 1990 to ensure compliance with the National Ambient Air Quality Standard. | Existing units: Comply with the Acid Rain Allowance Program under Title IV of the CAAA 1990 with phased-in requirements. Phase I covers the largest 110 coal-fired power plants in 1995. All other units above 25 megawatts are covered in Phase II beginning in 2000. |
| | New units: Comply with New Source Performance Standards (NSPS) set in 1978 and BACT fixed at 1990 levels applied through the New Source Review (NSR) process. | New units: Comply with the NSPS set in 1978, BACT/LAER (Lowest Achievable Emission Requirements), and the Acid Rain Allowance Trading Program under Title IV of the CAAA 1990. |
| **$NO_x$** | Existing Units: No federal standards, except NSPS for new units built after passage of the law. | Existing units: Meet Reasonably Available Control Technology (RACT) in 1995 in the OTR and all non-attainment areas per Title I. States can file waivers from RACT requirements. Coal-fired units comply with Title IV $NO_x$ requirements that are phased in over time, or RACT, whichever is more stringent. Group 1/Phase I coal-fired units comply in 1996. Group 1/Phase II and Group 2 coal-fired units comply in 2000. Collective action by the 37 eastern States in the Ozone Transport Assessment Group (OTAG) will lead to additional requirements (known as "Level 2 controls") under Title I for reducing $NO_x$ emissions during the summer months (May - September). |
| | New units: Units using fossil fuels comply with the NSPS for each generation technology and fuel. Application of BACT in the NSR process at levels existing in 1990. | New units: Comply with Title I NSPS and BACT/LAER and Title IV standards for coal-fired units, whichever is more stringent. Units subject to OTAG Level 2 controls for reducing $NO_x$ during summer months. |

## Compliance Actions

In order to comply with the Title IV $SO_x$ Allowance Trading program under the Post-CAAA scenario, the electric power industry must install continuous emissions monitoring systems. In addition to the monitoring system, they may be required to adopt at least one of the following four types of action:

- Improve the performance of existing scrubber units and scrubbers that facilities will build on new units under the NSPS of the Pre-CAAA Scenario
- Add scrubbers on existing units
- Switch to lower sulfur coals
- Switch over from coal-fired to gas-fired units

We assume in the Section 812 cost analysis that the electric power industry faces four $NO_x$ regulatory programs. These programs require the industry to:

- Place RACT controls on existing generation units in States without EPA waivers
- Build new generation units to meet BACT requirements
- Comply with Title IV $NO_x$ rules for new and existing coal-fired units
- Comply with $NO_x$ Cap-and-Trade program for reducing emissions during the summer months in the eastern United States

## Cost Approach

We configured the IPM to forecast the operation of the electric power industry from 2000 to 2010. The baseline case, used in EPA's Clean Air Power Initiative (CAPI), includes the set of CAAA controls that the Agency promulgated or States established through their permit decisions by the middle of 1996. The baseline case also includes RACT and BACT decisions under the New Source Review program, Phase I and Phase II of the Title IV $SO_x$ Allowance Trading Program, and Phase I $NO_x$ control requirements applied to all tangentially-fired and wall-fired boilers that use coal.

In simplest terms, we sets up the Pre-CAAA scenario for the electric power industry by removing the CAAA controls from the CAPI base case and running the IPM model to forecast emission levels and costs of producing electric power. We fix standards under the Pre-CAAA 2000 and Pre-CAAA 2010 scenarios at 1990 levels. To establish the Post-CAAA scenario, we add further $NO_x$ controls to the CAPI base case, which focuses on the emissions and costs of producing electric power under the CAAA Title IV $SO_x$ Allowance Trading program. The Post-CAAA scenario reflects a $NO_x$ cap-and-trade program that EPA presented at OTAG meetings and was considered, at the time the utility analysis was initiated (1995-1996), to be a plausible outcome of the OTAG process. The $NO_x$ control program incorporated in the Post-CAAA scenario may not reflect the $NO_x$ controls that are actually implemented in a regional ozone transport rule.

## Cost Results

Cost results are presented in Table B-5 below. Based on the Section 812 cost analysis for the electric power industry, we estimate that the annual national costs of the CAAA will be roughly $3.1 billion in 2000 and $4.6 billion in 2010.

**Table B-5**
**Electric Power Industry Costs from Post-CAAA Controls for $SO_x$ and $NO_x$**

| | Annual Control Costs (millions 1990 dollars) | |
|---|---|---|
| **Pollutant** | **Post-CAAA 2000** | **Post-CAAA 2010** |
| $SO_x$ | $1,900 | $1,600 |
| $NO_x$ | $1,200 | $2,900 |
| **Total** | **$3,100** | **$4,600** |

## Non-Road Engines/Vehicles

Nonroad sources are mobile (non-highway) emission sources. They include the following: lawn and garden equipment, construction equipment, agricultural equipment, industrial equipment, aircraft and airport service vehicles, logging equipment, recreational vehicles, locomotives, and marine vessels. We use ERCAM to estimate future emissions from non-road engines/vehicles. This model incorporates Federal regulatory programs for controlling $NO_x$, $PM_{10}$, VOC, and CO emissions from nonroad engines and equipment under the Post-CAAA scenario.

### Cost Approach

To develop cost estimates for nonroad control measures, we apply cost-effectiveness values from several sources (e.g., draft or final rules and the Section 812 emission projections analysis (Pechan, 1997a)). The analysis includes costs for control inputs applied to the following nonroad source categories: small SI (gasoline) engines, CI (diesel) engines, locomotives, and marine vessels.

We calculate TACs in each implementation year to calculate the net present value (NPV) of both costs and benefits over the estimated period of fleet turnover.[10] Because we base the benefits analysis on projected emission reductions in 2000 and 2010, rather than the discounted stream of benefits, the inputs to this cost analysis represent the annualized cost per ton of reduction, not the NPV cost-effectiveness. The exception is the input used for the Federal locomotives rule; because TAC in each implementation year are not available, we use the average annualized cost per ton across the entire implementation period in both 2000 and 2010.

### Cost Results

Table B-6 summarizes the cost estimates for each nonroad engine/vehicle control measure modeled in this analysis for 2000 and 2010. Total nonroad engine/vehicle costs, under Post-CAAA scenarios, are $104 million and nearly $400 million in 2000 and 2010, respectively. Estimated SI engine costs are $56 million under the 2000 Post-CAAA scenario and $104 million under the 2010 Post-CAAA scenario. Reducing VOC emissions from lawn and garden equipment contributes to the majority of SI engine costs. CI engine control costs are $22 million in the 2000 Post-CAAA scenario, and $32 million in 2010. $NO_x$ emission reductions from construction equipment account for a significant proportion of total CI engine control costs. Locomotive and commercial marine vessel benefits are not realized until after 2000; costs under the 2000 Post-CAAA scenario are therefore zero.[11]

[10] We were unable to apply the per engine costs of modifying equipment/vehicles to meet EPA standards because engine populations were not available for all areas.

[11] $NO_X$ standards for locomotive and commercial marine vessels do not take effect until 2000. For the purpose of this analysis, costs are small enough in 2000 that they are omitted.

**Table B-6**
**Cost Estimates for Nonroad Engine/Vehicle CAAA Programs**

| Engine/Vehicle Category | Annual Cost (million 1990 dollars) Post-CAAA 2000 | Post-CAAA 2010 |
|---|---|---|
| **SI Engines:** | | |
| Construction Equipment | $ 1.7 | $ 3.1 |
| Industrial Equipment | 0.5 | 1.2 |
| Lawn and Garden Equipment | 41 | 74 |
| Farm Equipment | 0.2 | 0.4 |
| Commercial Equipment | 12 | 23 |
| Logging Equipment | 0.9 | 2.1 |
| **CI Engines:** | | |
| Construction Equipment | $ 12 | $ 17 |
| Industrial Equipment | 3.6 | 5.2 |
| Farm Equipment | 2.9 | 4.4 |
| Logging Equipment | 0.1 | 0.2 |
| Airport Service Equipment | 2.8 | 4.6 |
| Other | 0.2 | 0.2 |
| **Locomotives** | $ 0[1] | $ 35 |
| **Marine Vessels:** | | |
| Recreational | $ 27 | $ 230 |
| Commercial | 0[1] | 1 |
| **Total Nonroad Engine/Vehicle Control Costs** | **$ 104** | **$ 400** |

Note:
[1] Costs in 2000 are zero because program emissions reductions are not realized until after 2000. See text and Pechan (1998 and 1997a) for further explanation.

## *Motor Vehicles*

Motor vehicle emissions account for almost thirty percent of 1990 anthropogenic VOC emissions and thirty-two percent of $NO_x$ emissions. To determine the costs of controlling VOC and $NO_x$, we first project motor vehicle emissions with ERCAM-VOC and ERCAM-$NO_x$ (Pechan, 1998). Then we use the emissions projections to estimate future year motor vehicle program costs for each of the modeled control assumptions.[12]

[12] See the emission projection report for a discussion of the emission projection methodology and the control assumptions (Pechan, 1998).

## Cost Approach

We convert all motor vehicle-related control costs into one of three forms: cost per new vehicle, cost per registered vehicle, or cost per mile traveled. We calculate separate costs for each vehicle type (i.e., LDGV, light-duty gasoline truck (LDGT) 1, LDGT2). Motor vehicle calculations required projections of vehicle miles traveled (VMT), vehicle registrations, or vehicle sales estimates. We applied the following equations:

*Cost per new vehicle = projected vehicle sales * production cost ($/new vehicle)*

*Cost per registered vehicle = projected vehicle registrations * cost per vehicle ($/vehicle)*

*Cost per mile traveled = projected vehicle miles traveled (VMT) * cost per mile ($/mile)*

## Sources of Data

The 1990 NPI Inventory provides the 1990 VMT data, which we project in the same manner as it our emissions (Pechan, 1998).[13] National registrations from the MOBILE4 FCM are the source of vehicle registration data for 1990 (EPA, 1991d). The source of motorcycle registrations is *Highway Statistics* (FHWA, 1991). National sales data is based on projected sales compiled by Data Resources Incorporated (DRI). This information was also used by EPA in the onboard vapor recovery RIA (DRI, 1993; EPA, 1993d).[14]

## Cost Categories

The CAAA motor vehicle provisions generate costs in the following categories: emissions standards, fuel requirements, emissions inspections, and low emission vehicle programs. The following section describes the methodology we use calculate costs for each category of provisions.[15]

**Emission Standards:**

- **Tier 1 Certification Standards and Evaporative Controls.** We calculate costs for tailpipe standards and evaporative controls with per-vehicle production costs applied to projected sales.

- **Heavy-Duty Vehicle 2g/bhp-hr Equivalent $NO_x$ Standard.** We calculate the cost of complying with the 2004 model year emission standards by estimating the baseline package of emission control technology for meeting 1998 model year standards (EPA, 1997f). We use the 1994 model year sales of different size classes of diesel trucks to establish sales fractions, assumed to represent future sales as well. We multiply these sales fractions by the year 2009 per vehicle cost increases for light, medium, and HDVs to compute a sales-weighted per vehicle cost increase.

- **Onboard Vapor Recovery.** To estimate the costs of onboard vapor recovery, we use expected increases in vehicle price (also referred to as retail price equivalent) and average lifetime operating cost (net present value) (EPA, 1993f).

- **Cold Temperature CO Standard.** The cost of the cold temperature CO standard to the consumer includes the cost to the manufacturer, the manufacturer's and dealer's overhead and profits, and the increase or decrease in maintenance and fuel costs. We do not include fuel economy improvements in the analysis. We base cost estimates on retail price increases of $19 per LDV, $32 per LDT1, and $48.50 per LDT2 (Pechan, 1998).

- **Onboard Diagnostic (OBD) Systems.** With OBD now appearing on all 1996 model year cars and light-trucks, Federal OBD costs are approximately $65 to $100 per vehicle.[16]

**Fuels:**

- **Gasoline Volatility Limits.** In order to calculate the costs of lowering the Reid vapor pressure (RVP) from 10.5 to 9.0 in Class C areas, we apply the cost estimate of

[13] EPA uses MOBILE4 FCM national projections, scaled to metropolitan statistical areas (MSAs) according to population projections, to project VMT and vehicle registrations.

[14] EPA assumes that motorcycle sales from Highway Statistics (FHWA, 1991) increase at the same rate as light-duty gasoline vehicle (LDGV) sales.

[15] For more details on these standards see Appendix A.

[16] We apply the per vehicle cost estimate of $65. However, there is evidence that OBD costs are more likely in the range of $65 to $100 per vehicle (EPA, 1993d).

0.225 cents per gallon in the five month ozone season (Wysor, 1988).

- **Federal Reformulated Gasoline.** We base reformulated gasoline costs on an incremental refiner's cost increase and a monetized fuel economy disbenefit. An estimate of 3.9 cents per gallon for Phase I and 5.1 cents per gallon for Phase II was used (EPA, 1993g). Phase II reformulated gasoline modifications occur only in the summer. As a result, we consider Phase II costs to only five months out of the year. The Phase I benefits will occur year-round and are primarily due to the oxygenate (affecting the aromatic content) and the reduction of fuel benzene content.

- **California Reformulated Gasoline.** We use the estimates from the California Air Resources Board (CARB) to determine the increase in per gallon fuel costs to consumers (CARB 1990; CARB, 1991).

- **Oxygenated Fuels.** We base oxygenated fuel costs on an incremental cost of 3.8 cents per gallon (EPA, 1993g).

- **California Reformulated Diesel.** We base reformulated diesel costs on an incremental per gallon increase of six cents (Green, 1994).

- **Diesel Fuel Sulfur Limits.** We use an average value of 2.1 cents per gallon as an estimate of the incremental cost of reducing the sulfur content of conventional diesel fuel (EPA, 1990). The cost estimate do not include a fuel economy penalty for low sulfur diesel fuel because we estimate that energy content is essentially the same as that of conventional fuel (less than 1% lower).

**Emissions Inspection Programs:**

- **Basic I/M.** We use the RIA on enhanced I/M for deriving this program's basic costs. Total per vehicle costs include the inspection fee, average repair cost, and the fuel economy benefit. The average per vehicle cost is approximately $5.70. We apply this estimate to LDGVs, LDGT1s, and LDGT2s in areas where basic I/M is required. Basic I/M costs are evenly apportioned among VOC, $NO_x$, and CO. No additional costs are attributed to areas that face I/M program requirements, but already have a program in place (EPA, 1992c).

- **Low Enhanced I/M.** Costs for low enhanced I/M and OTR low enhanced I/M are not well defined. Therefore, we equate low enhanced I/M costs equivalent to those of basic I/M. The average cost per vehicle for this program is approximately $5.70. This per vehicle cost applies to all registered LDGV, LDGT1, and LDGT2 (EPA, 1992c).

- **Enhanced I/M.** Estimates of enhanced I/M costs are subject to change as States make decisions about their program designs. I/M program costs may be higher or lower according to each State's selected program designs such as centralized testing and caps on the costs of required repairs.[17] The estimated per-vehicle cost is $15.70. We base this figure on a test fee ($18), an average repair cost ($14.20 per vehicle), and an average fuel economy benefit ($16.50 per vehicle) (EPA, 1992c).[18] We estimate annual costs by applying the per vehicle costs to an area's projected vehicle registrations.

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[17]To date, only four States have implemented Enhanced I/M programs. Preliminary cost estimates indicate that opportunity costs to vehicle owners in the form of travel and wait time do not play as significant role as originally anticipated. (Harrington and McConnell, 1999.)

[18]Test fee and the relationship between test sites and States vary. In some cases the test fee represents a payment to the state or local government. In other cases, the fee covers the direct costs of the testing program. It is not clear, however, how the test fee could be apportioned between the two possibilities. To the extent the fee represents a transfer payment, we may be overestimating the direct cost and social cost of the program.

**Low Emission Vehicles:**

- **California Low Emission Vehicle Program.** We base costs for the California LEV on the incremental production cost of vehicles meeting each of the LEV standards (Pechan, 1998). The overall incremental production cost for a vehicle type reflects the projected fraction of sales of each type of LEV for each projection year.

- **National Low Emission Vehicle Program.** We calculate costs for the National LEV program by multiplying the incremental production cost of vehicles meeting each of the LEV standards by the estimated new vehicle sales volumes (Pechan, 1998).

**Additional Programs:**

- **Clean Fuel Fleet Program (CFFP).** CAAA mandated implementation of CFFP beginning in 1998 for ozone NAAs designated serious and above. We estimate that the model year 1998 fleet demand for clean-fuel vehicles under the CFFP will be approximately 47,000 LDVs and 12,000 HDVs (Oge, 1997). However, we do not include these costs in the analysis.[19]

- **Transportation Conformity.** Under the transportation conformity rule, the Metropolitan Planning Organizations (MPOs) must perform regional transportation and emissions modeling and document the regional air quality impacts of transportation plans and programs. We expect these requirements will generate the primary costs of this rule.

Table B-7 summarizes the motor vehicle unit costs used in this analysis. Costs of individual motor vehicle provisions are listed in 1990 dollars by vehicle type. Phase II RVP and Phase II Federal reformulated gasoline limits generate costs only in the ozone season, while oxygenated fuel provisions result in CO season (winter time) costs. All other fuel programs listed in Table B-7 generate year round costs.

[19] EPA cannot require manufacturers to produce CFVs and areas covered by the CAAA can opt out of the program.

**Table B-7**
**Motor Vehicle Unit Costs by Provision**

| Provision | Year Dollars | Cost Unit | Cost Estimate by Vehicle Type:[1] LDGV | LDGT1 | LDGT2 | MC | HDGV | HDDV | LDDV | LDDT |
|---|---|---|---|---|---|---|---|---|---|---|
| **Emission Standards:** | | | | | | | | | | |
| Tier 1 Tailpipe Standards: VOC | 1990 | Sales | 36.8 | 33.7 | 11.7 | | | | | |
| Tier 1 Tailpipe Standards: $NO_X$ | 1990 | Sales | 115.0 | 80.6 | 45.3 | | 16.0 | 78.0 | | |
| Cold Temperature CO Standard | 1989 | Sales | 15-23 | 15-48 | 42-55 | | | | | |
| Evaporative Controls (New Evaporative Emissions Test Procedure) | 1993 | Sales | 1.0 | 8.0 | 8.0 | | (13.0) | | | |
| On-Board Vapor Recovery System | 1992 | Sales | 4.54 | 4.48 | 4.48 | | | | | |
| On-Board Diagnostics | 1993 | Sales | 65.0 | 65.0 | 65.0 | | | | | |
| Heavy Duty Engine Standard (2 gram equivalent) | 1995 | Sales | | | | | | 140.0 | | |
| Low Emission Vehicles: | | | | | | | | | | |
| TLEV | 1996 | | 53.0 | 53.0 | | | | | | |
| LEV | | | 95.0 | 95.0 | | | | | | |
| ULEV | | | 145.0 | 145.0 | | | | | | |
| ZEV | | | 5,000.0 | 5,000.0 | | | | | | |
| **Fuels:** | | | | | | | | | | |
| Phase II RVP Limits | 1990 | Cents/gallon | 0.2 | 0.2 | 0.2 | 0.2 | 0.2 | | | |
| Federal Reformulated Gasoline: Phase I | 1993 | Cents/gallon | 3.9 | 3.9 | 3.9 | 3.9 | 3.9 | | | |
| Phase II | 1993 | Cents/gallon | 1.2 | 1.2 | 1.2 | 1.2 | 1.2 | | | |
| Oxygenated Fuels | 1993 | Cents/gallon | 3.8 | 3.8 | 3.8 | 3.8 | 3.8 | | | |
| Low-Sulfur Diesel Fuel Requirements (0.05% sulfur) | 1990 | Cents/gallon | | | | | | 2.1 | 2.1 | 2.1 |
| California Phase II Reformulated Gasoline | 1991 | Cents/gallon | 12.3 | 12.3 | 12.3 | 12.3 | 12.3 | | | |
| California Reformulated Diesel | | | | | | | | 6.0 | 6.0 | 6.0 |
| **Inspection/Maintenance Programs:** | | | | | | | | | | |
| Basic | 1992 | Registrations | 5.7 | 5.7 | 5.7 | | | | | |
| Low Enhanced | 1992 | Registrations | 5.7 | 5.7 | 5.7 | | | | | |
| High Enhanced | 1992 | Registrations | 15.7 | 15.7 | 15.7 | | | | | |

Notes:
[1] LDGV = light duty gasoline vehicle; LDGT = light duty gasoline truck; MC = motorcycle; HDGV = heavy duty gasoline vehicle; HDDV = heavy duty diesel vehicle; LDDV = light duty diesel vehicle; LDDT = light duty diesel truck

## Cost Results

Table B-8 summarizes the motor vehicle costs for 2000 and 2010. The total cost post-CAA is $9 billion in 2000 and $12 billion in 2010.

**Table B-8**
**Cost Estimates of Motor Vehicle Program**

| Program | Annual Cost (million 1990 dollars) Post-CAAA 2000 | Post-CAAA 2010 |
|---|---|---|
| **Title I** | | |
| California LEV | $ 320 | $ 1,100 |
| National LEV | 180 | 1,060 |
| Basic I/M | 57 | 69 |
| Low/OTR Enhanced I/M | 82 | 99 |
| High Enhanced I/M | 1,100 | 1,400 |
| **Title II** | | |
| Onboard Vapor Recovery* | $ 63 | $ 69 |
| Stage II Vapor Recovery* | 71 | 86 |
| Phase II RVP | 280 | 340 |
| Tailpipe/Extended Useful Life - VOC | 504 | 550 |
| Tailpipe/Extended Useful Life - $NO_x$ | 1,500 | 1,700 |
| Evaporative/Running Losses | 42 | 46 |
| Onboard Diagnostics | 880 | 960 |
| Cold Temperature CO Standard | 380 | 410 |
| Federal Reformulated Gasoline | 720 | 860 |
| California Reformulated Gasoline | 2,000 | 2,400 |
| Oxygenated Fuels | 160 | 204 |
| 2 gram $NO_x$ Heavy Duty Standard | 0 | 69 |
| Low Sulfur Diesel Fuel | 570 | 740 |
| California Reformulated Diesel** | 170 | 230 |
| **Total Motor Vehicle Control Costs** | **$ 9,070** | **$ 12,300** |

Notes:
* The benefits of onboard vapor recovery and stage II vapor recovery are accounted for under area sources. The cost for onboard vapor recovery systems is estimated assuming phase-in for light duty gasoline vehicles and light duty trucks. Heavy duty trucks are not affected.
** The analysis does not account for the benefits (emission reductions).

### *Area Sources*

Area sources comprise small stationary sources not listed in the point source database (e.g., dry cleaners, graphic arts, industrial fuel combustion, gasoline marketing) and solvent use (e.g., consumer solvents, architectural coatings). Area sources of $NO_x$ emissions include industrial fuel combustion units in the industrial, commercial/institutional, and residential sectors. The following are VOC sources: pharmaceutical manufacturing, wood furniture surface coating, aerospace manufacturing (surface coating), ship building and ship repair (surface coating), halogenated solvent cleaning, dry cleaning - perchloroethylene (PCE), and petroleum refinery fugitives. Area sources of $PM_{10}$ are paved roads, unpaved roads, construction, cattle feedlots, agricultural tilling, and agricultural burning.

## Cost Approach

To assess the costs of reducing emissions from area sources, we use annualized costs per ton reduced.[20] We estimate total annual costs under each of the Post-CAAA scenarios by applying annualized costs per ton from a variety of regulatory documents to corresponding emission reductions. The annual cost formula is:

*Annual Cost = Annualized Cost Per Ton * Emission Reduction.*[21]

ERCAM-$NO_x$ and ERCAM-VOC incorporate separate cost equations for each area source category.

## Cost Results

Table B-9 summarizes area source control measure costs for $NO_x$ and PM under the 2000 and 2010 Post-CAAA scenarios. The costs associated with applying $NO_x$ point source fuel combustion controls to smaller sources are approximately $16 million in 2000 and $18 million in 2010. Control measures applied to reduce PM emissions from area sources are estimated to cost $1.9 billion under the 2000 Post-CAAA scenario and $2.2 billion under the 2010 Post-CAAA scenarios. Controlling fugitive dust emissions from construction activity generates the majority of PM control costs.

[20] Source-specific data are not available for area and nonroad sources.

[21] In the present analysis, we annualize total capital costs with a five percent discount rate. In some cases, we re-calculate the annualized cost per ton reported in the source material if that estimate was based on a discount rate other than five percent.

**Table B-9**
**Cost Summary of Area Source $NO_X$ and PM Controls**

| Source Category | Annual Costs (million 1990 dollars) | |
|---|---|---|
| | Post-CAAA 2000 | Post-CAAA 2010 |
| **Area Source $NO_x$ Controls** | | |
| Industrial Fuel Combustion - Coal | $ 6.6 | $ 7.5 |
| Industrial Fuel Combustion - Oil | 0.8 | 0.8 |
| Industrial Fuel Combustion - Natural Gas | 8.5 | 9.9 |
| **Total Area Source $NO_x$** | **$ 16** | **$ 18** |
| **Area Source PM Controls** | | |
| Agricultural Burning | $ 37 | $ 39 |
| Agricultural Tilling | 4.1 | 3.6 |
| Beef Cattle Feedlots | 1.4 | 1.7 |
| Construction | 1,500 | 1,800 |
| Paved Roads | 350 | 440 |
| Unpaved Roads | 1.1 | 0.8 |
| **Total Area Source PM** | **$ 1,900** | **$ 2,200** |

## *Reasonable Further Progress Requirements*

Title I of the CAAA includes provisions that require ozone nonattainment areas to make steady progress toward compliance with NAAQS. NAAs classified as moderate, serious, severe, or extreme must demonstrate that they are working to lower ambient ozone concentrations at a reasonable rate of progress (ROP) and, by 1996, reduce annual VOC emission by fifteen percent from 1990 levels. In addition to satisfying ROP requirements, areas classified as having an ozone nonattainment problem that is serious or worse must continue to cut emissions and make reasonable further progress (RFP) toward attainment. To meet RFP standards, after 1996, NAAs have to reduce precursor emissions by three percent per year until they each reach their respective compliance deadlines. While ROP requirements mandate VOC cuts to comply with RFP standards it is often possible to substitute $NO_x$ for VOC. (Refer to Appendix A for more discussion of ROP/RFP requirements.)

Title I progress requirements establish minimum emissions reduction standards for ozone NAAs. In many cases, the areas subject to ROP/RFP regulations satisfy these requirements simply by complying with other existing emissions standards. VOC and $NO_x$ reductions made to meet other regulations are credited towards ROP/RFP requirements. For the purposes of the prospective analysis, we assume that where possible, credit is given for all available $NO_x$ cuts and that any remaining emission reduction needed to satisfy Title I progress requirements come from VOC. In the majority of cases, credited VOC cuts account for this remaining reduction. For NAAs that are not able to fulfill the remainder of their ROP/RFP obligations with credited VOC emissions reductions, there is a shortfall. This shortfall represents the quantity of VOC that ozone NAAs must reduce through control efforts beyond those mandated by other clean air provisions.

Tables B-10 and B-11 show, for the years 2000 and 2010 respectively, which NAAs are assumed to

satisfy, and not satisfy, their Title I progress requirement. Failure to meet the requirement is indicated by a shortfall. The shortfall is measured by the amount ozone season daily (OSD) level exceeds the maximum allowable daily VOC emission. The OSD level of VOC emission represents the predicted daily emission in the absence of RFP/ROP requirements, and VOC target presents the maximum allowable daily VOC emissions.

**Table B-10**
**2000 Rate of Progress Analysis**

| Ozone Nonattainment Area | VOC OSD[1] | VOC Target[2] | Shortfall |
|---|---|---|---|
| Atlantic City | 37.97 | 45.71 | 0.00 |
| Baltimore | 318.33 | 376.47 | 0.00 |
| Baton Rouge | 203.77 | 415.65 | 0.00 |
| Beaumont-Port Arthur | 340.66 | 450.66 | 0.00 |
| Chicago-Gary-Lake County | 1,240.89 | 1,202.25 | 38.64 |
| Cincinnati-Hamilton | 305.34 | 341.18 | 0.00 |
| Cleveland-Akron-Lorain | 521.14 | 573.07 | 0.00 |
| Dallas-Fort Worth | 694.53 | 673.97 | 20.56 |
| El Paso | 85.38 | 69.02 | 16.36 |
| Grand Rapids | 182.72 | 175.66 | 7.06 |
| Houston-Galveston-Brazoria | 1,426.65 | 2,268.31 | 0.00 |
| Lewiston-Auburn ME | 34.08 | 35.98 | 0.00 |
| Los Angeles-South Coast | 972.91 | 939.08 | 33.83 |
| Louisville | 219.66 | 215.97 | 3.69 |
| Milwaukee-Racine | 327.09 | 293.24 | 33.85 |
| Muskegon | 46.67 | 44.32 | 2.35 |
| Nashville | 231.71 | 205.60 | 26.11 |
| New York-N New Jersey-Long Is | 1,994.96 | 2,407.97 | 0.00 |
| Philadelphia-Wilmington-Trenton | 1,090.49 | 1,376.55 | 0.00 |
| Phoenix | 377.43 | 347.91 | 29.52 |
| Pittsburgh-Beaver Valley | 407.05 | 399.80 | 7.25 |
| Portland ME | 70.05 | 73.33 | 0.00 |
| Portsmouth-Dover-Rochester | 53.54 | 58.70 | 0.00 |
| Providence | 173.78 | 180.51 | 0.00 |
| Reading PA | 60.53 | 61.14 | 0.00 |
| Richmond-Petersburg | 179.97 | 201.70 | 0.00 |
| Sacramento Metro | 158.01 | 155.08 | 2.93 |
| St. Louis | 465.64 | 549.14 | 0.00 |
| Monterey Bay | 64.16 | 79.63 | 0.00 |
| Salt Lake City | 182.75 | 150.80 | 31.95 |
| San Diego | 192.90 | 189.71 | 3.19 |
| Santa Barbara-Santa Maria-Lomp | 82.75 | 83.10 | 0.00 |
| Sheyboygan | 24.49 | 22.44 | 2.05 |
| Washington DC | 402.76 | 477.03 | 0.00 |
| Knox & Lincoln Cos ME | 9.95 | 10.37 | 0.00 |
| Kewaunee Co Wi | 4.86 | 4.56 | 0.30 |
| Manitowoc Co WI | 19.72 | 17.20 | 2.52 |
| San Joaquin Valley | 470.50 | 532.41 | 0.00 |
| Ventura Co CA | 65.69 | 70.52 | 0.00 |
| Southeast Desert Modified | 227.71 | 219.32 | 8.39 |
| Boston-Lawrence-Worcester-E.MA | 822.65 | 918.01 | 0.00 |
| Springfield/Pittsfield-W. MA | 155.51 | 152.42 | 3.09 |
| Greater Connecticut | 316.27 | 370.11 | 0.00 |

Notes:

1 The VOC OSD (ozone season daily) values are the estimated daily emissions in the absence of ROP/RFP requirements. These estimates do, however, incorporate the effect of the VOC reductions that are credited towards Title I progress requirements.

2 The VOC target represents the maximum allowable daily VOC emission for NAAs to comply with ROP/RFP requirements. The VOC target is calculated based upon the assumption that all available NOx cuts are credited towards ROP/RFP requirements and that all necessary remaining reductions come from VOC.

**Table B-11**
**2010 Rate of Progress Analysis**

| Ozone Nonattainment Area | VOC OSD[1] | VOC Target[2] | Shortfall |
|---|---|---|---|
| Atlanta | 492.40 | 541.99 | 0.00 |
| Atlantic City | 33.21 | 45.71 | 0.00 |
| Baltimore | 293.56 | 376.47 | 0.00 |
| Baton Rouge | 206.65 | 415.65 | 0.00 |
| Beaumont-Port Arthur | 377.63 | 450.66 | 0.00 |
| Chicago-Gary-Lake County | 1,236.73 | 840.15 | 396.58 |
| Cincinnati-Hamilton | 283.74 | 341.18 | 0.00 |
| Cleveland-Akron-Lorain | 485.90 | 573.07 | 0.00 |
| Dallas-Fort Worth | 687.15 | 673.97 | 13.18 |
| El Paso | 84.33 | 69.02 | 15.31 |
| Grand Rapids | 183.11 | 175.66 | 7.45 |
| Houston-Galveston-Brazoria | 1,530.07 | 1,606.75 | 0.00 |
| Lewiston-Auburn ME | 32.03 | 35.98 | 0.00 |
| Los Angeles-South Coast | 847.66 | 670.95 | 176.71 |
| Louisville | 216.86 | 215.97 | 0.89 |
| Milwaukee-Racine | 321.89 | 204.72 | 117.17 |
| Muskegon | 46.86 | 44.32 | 2.54 |
| Nashville | 230.00 | 205.60 | 24.40 |
| New York-N New Jersey-Long Is | 1,842.53 | 2,407.97 | 0.00 |
| Philadelphia-Wilmington-Trenton | 1,070.05 | 1,194.41 | 0.00 |
| Phoenix | 347.52 | 347.91 | 0.00 |
| Pittsburgh-Beaver Valley | 358.67 | 399.80 | 0.00 |
| Portland ME | 66.88 | 73.33 | 0.00 |
| Portsmouth-Dover-Rochester | 52.49 | 58.70 | 0.00 |
| Providence | 166.61 | 193.15 | 0.00 |
| Reading PA | 55.33 | 61.14 | 0.00 |
| Richmond-Petersburg | 179.35 | 201.70 | 0.00 |
| Sacramento Metro | 135.99 | 120.24 | 15.75 |
| St. Louis | 439.47 | 549.14 | 0.00 |
| Monterey Bay | 61.76 | 79.63 | 0.00 |
| Salt Lake City | 189.83 | 150.80 | 39.03 |
| San Diego | 174.04 | 202.24 | 0.00 |
| Santa Barbara-Santa Maria-Lomp | 81.53 | 83.10 | 0.00 |
| Sheyboygan | 24.69 | 22.44 | 2.25 |
| Washington DC | 355.35 | 477.03 | 0.00 |
| Knox & Lincoln Cos ME | 8.98 | 10.37 | 0.00 |
| Kewaunee Co Wi | 4.77 | 4.56 | 0.21 |
| Manitowoc Co WI | 19.37 | 17.20 | 2.17 |
| San Joaquin Valley | 448.37 | 566.96 | 0.00 |
| Ventura Co CA | 62.33 | 63.11 | 0.00 |
| Southeast Desert Modified | 213.87 | 172.34 | 41.53 |
| Boston-Lawrence-Worcester-E.MA | 775.66 | 918.01 | 0.00 |
| Springfield/Pittsfield-W. MA | 147.45 | 166.51 | 0.00 |
| Greater Connecticut | 292.53 | 370.11 | 0.00 |

Notes:

1 The VOC OSD (ozone season daily) values are the estimated daily emissions in the absence of ROP/RFP requirements. These estimates do, however, incorporate the effect of the VOC reductions that are credited towards Title I progress requirements.

2 The VOC target represents the maximum allowable daily VOC emission for NAAs to comply with ROP/RFP requirements. The VOC target is calculated based upon the assumption that all available NOx cuts are credited towards ROP/RFP requirements and that all necessary remaining reductions come from VOC.

## Cost Results

We base the RFP cost estimate on the assumption that ozone nonattainment areas (NAAs) will take credit for $NO_x$ reductions for meeting progress requirements. Additional area-specific analysis would be necessary to determine the extent to which areas find $NO_x$ reductions beneficial in meeting attainment and progress requirement targets. Trading of $NO_x$ for VOC to meet RFP requirements may result in distributions of VOC and $NO_x$ emission reductions that differ from those used in this analysis. In part as a response to these uncertainties, we adopt a conservative strategy for estimating the costs of RFP reductions, using the relatively high cost per ton reduced value of $10,000 for all required reductions. We calculate these annual figures by multiplying the aggregate daily shortfall by 365, and then multiplying this number by the estimated cost of each ton of reduction, $10,000. Based on this calculation, the annual estimated cost of Title I progress requirements is $1,150 million in Post-CAAA 2000 and $2,460 million in Post-CAAA 2010.

Since the time we conducted our initial cost analysis, control measures for several nonattainment areas (NAA) have been identified that suggest controls may be much less. For example, the dollar per ton estimate associated with control measures selected in Chicago is $3,500. We incorporate this information in our sensitivity analysis. In the sensitivity test analysis, we calculate overall costs by applying the cost per ton of reduction associated with each identified control. Where the required reduction cannot be achieved through implementation of all of the identified controls, we assume unidentified controls will be used to eliminate the remaining shortfall. We apply the $10,000 per ton reduced estimate for these unidentified controls (see Appendix B for more details). Results of the sensitivity analysis suggest that our conservative approach of applying $10,000 per ton reduced to all VOC shortfalls may overstate cost by as much as several billion dollars in 2010.

### *Costs by Title*

Examining CAAA costs by title, in addition to reviewing them by source, is useful for understanding the cost components. Table B-12 summarizes the cost estimates generated in this analysis by year and Title. As shown in the table, the cost estimate under the Post-CAAA 2000 scenario is $19 billion, increasing to nearly $27 billion under the Post-CAAA 2010 scenario. All costs are in 1990 dollars.

**Table B-12**
**Summary of Cost Estimates by CAAA Title**

| Title | Sector/Pollutant | Annual Cost (million 1990 dollars) Post-CAAA 2000 | Post-CAAA 2010 |
|---|---|---|---|
| **Title I - Provisions for Attainment and Maintenance of NAAQS** | | | |
| | Non-utility Point/VOC | $ 420 | $ 440 |
| | Non-utility Point/$NO_x$ | 1,700 | 2,200 |
| | Utility/$SO_2$ and $NO_x$ | 790 | 2,500 |
| | Area/VOC | 920 | 1,040 |
| | Area/$NO_x$ | 16 | 18 |
| | Area/PM | 1,900 | 2,200 |
| | Motor Vehicle | 1,800 | 3,700 |
| | Progress Requirements | 1,200 | 2,500 |
| **Title II - Provisions Relating to Mobile Sources** | | | |
| | Motor Vehicle | $ 7,300 | $ 8,700 |
| | Nonroad | 104 | 400 |
| **Title III - Hazardous Air Pollutants** | | | |
| | Point/VOC | $ 480 | $ 520 |
| | Area/VOC | 130 | 150 |
| | Non-VOC MACT[1] | 170 | 170 |
| **Title IV - Acid Deposition Control** | | | |
| | Utility/$SO_2$ and $NO_x$ | $ 2,300 | $ 2,040 |
| **Title V - Permits** | | $ 300[2] | $ 300[2] |
| **TOTAL** | | **$ 19,400** | **$ 26,800** |

Notes:
1 Costs reflect estimate of annualized costs from final rules. We do not use ERCAM-VOC to model source categories, because the National Emission Standards for Hazardous Air Pollutants (NESHAPs) are associated with non-VOC HAP emission reductions. Consequently, they are not included in the Post-CAAA 2000 and 2010 inventories.
2 Includes costs only for State-implemented permitting programs, excluding the costs of Federally-implemented programs, since all Title V permit programs will be State-run in 2005.

## Joint Rules

Assigning costs to a CAAA Title is difficult in the case of "joint rules" issued under more than one Title. For example, the marine vessel rule incorporates controls to reduce VOC emissions through reasonably available control technology (RACT) standards and hazardous air pollutant (HAP) emissions through maximum achievable control technology (MACT) standards. In general, we assign the costs for joint rules to the CAAA title based on the implementation dates and the year by which emission reductions are expected to occur. In some cases, we assign joint rules costs to the more stringent rule (in terms of sources covered or reduction required). Examples of source categories with overlapping Title I and Title III control measures include the following: aerospace, surface coating, petroleum refineries, shipbuilding, synthetic organic chemical manufacturing industry (SOCMI) categories, printing, and wood furniture. In cost accounting, we generally allocate the costs for these source categories to Title III, rather than Title I.

## Title I

Title I, Provisions for Attainment and Maintenance of National Ambient Air Quality Standards (NAAQS), includes national VOC rules and any controls that NAAs will likely apply to meet Federal standards for ozone and PM. For Title I,

determining which rules are CAAA-related and which are associated with other legislation is sometimes difficult. For example, EPA actually promulgated the hazardous waste transport, storage, and disposal facilities (TSDF) rule under the authority of the Resource Conservation and Recovery Act (RCRA). We attribute, however, the costs and associated VOC emission reductions of the Phase I and Phase II RCRA rule to Title I, because the rule is consistent with CAAA programs that promote attaining and maintaining the NAAQS.

The costs associated with Title I consist of point and area source costs for VOC, $NO_x$, and PM control measures. Total Title I costs are $8.6 billion in 2000 and $14.5 billion in 2010.[22] The following two tables summarize the cost analysis results for provisions promulgated under Title I of the CAAA. Table B-13 presents costs specifically associated with Title I national point and area VOC rules. Table B-14 costs by national Title I provisions regulating sectors ranging from motor vehicles to utilities. To preserve consistency with the assumptions made in the emission projections analysis, we simulate attainment of the ozone and PM NAAQS as they were prior to the 1997 revisions.

[22] Note that provisions included in other CAAA Titles, as well as the decisions that individual States make about how best to meet progress requirements and attainment targets, affect Title I costs.

**Table B-13**
**Title I National Rules, Point and Area Source VOC Control Costs**

| | Annual Costs by Sector (million 1990 dollars) | | | | | |
|---|---|---|---|---|---|---|
| | Post-CAAA 2000 | | | Post-CAAA 2010 | | |
| **Title I National Rules** | **Point** | **Area** | **Total** | **Point** | **Area** | **Total** |
| Consumer Products | $ 0.0 | $ 81 | $ 81 | $ 0.0 | $ 88 | $ 88 |
| AIM Coatings | 0.0 | 24 | 24 | 0.0 | 27 | 27 |
| Automobile Refinish Coatings | 0.0 | 6 | 6 | 0.0 | 7 | 7 |
| Hazardous Waste TSDFs | <0.1 | 300 | 300 | <0.1 | 350 | 350 |
| Municipal Landfills | 0.0 | 160 | 160 | 0.0 | 170 | 170 |
| Marine Vessel Loading | 24 | 0 | 24 | 28 | 0 | 28 |
| **TOTAL** | **$ 24** | **$ 570** | **$ 590** | **$ 28** | **$ 650** | **$ 680** |

Notes:
[1] Costs reflect estimates of annualized costs from final rules. We do not use ERCAM-VOC to model source categories, because the NESHAPs are associated with non-VOC HAP emission reductions.
[2] The Off-site Waste Treatment NESHAP was not modeled in this analysis. We assume that the Title I modeling of the RCRA Phase I and Phase 2 rules for hazardous waste TSDFs will capture any future MACT reductions and costs.
[3] EPA estimated that the Medical Waste Incineration guideline would cost between $59 million per year to $120 million per year, depending on the extent to which affected facilities switch to less expensive methods of treatment and disposal. The cost above represents the midpoint of this range.

**Table B-14**
**Summary of Costs for Title I**

| | Annual Cost (million 1990 dollars) | |
|---|---|---|
| **Provision** | **Post-CAAA 2000** | **Post-CAAA 2010** |
| **Area Specific:** | | |
| California LEV | $ 320 | $ 1,100 |
| National LEV | 180 | 1,060 |
| Basic I/M | 57 | 69 |
| Low/OTR Enhanced I/M | 82 | 99 |
| High Enhanced I/M | 1,100 | 1,400 |
| **RACT:** | | |
| VOC RACT | 620 | 660 |
| Non-utility $NO_x$ RACT | 37 | 40 |
| Utility $NO_x$ RACT/Best Available Control Technology (BACT) | 140 | 530 |
| New CTG | 130 | 150 |
| **OTR:** | | |
| Utility Cap-and-Trade Program | 640 | 1,200 |
| $NO_x$ Stationary (Non-utility) | 1,600 | 2,100 |
| **PM NAA Controls** | 1,900 | 2,200 |
| **Progress Requirements** | $ 1,200 | $ 2,500 |
| **TOTAL** | **$ 8,050** | **$ 13,900** |

## Title II

Title II provisions include Federal motor vehicle and nonroad engine/vehicle rules, in addition to regulations requiring fuel reformulations. Table B-15 summarizes the results of our cost analysis for provisions promulgated under Title II of the CAAA.

**Table B-15**
**Summary of Title II Motor Vehicle and Nonroad Engine/Vehicle Program Costs**

| | Annual Cost (million 1990 dollars) | |
|---|---|---|
| **Provision** | **Post-CAAA 2000** | **Post-CAAA 2010** |
| **Motor Vehicles/Fuels:** | | |
| **Motor Vehicle Emission Standards:** | | |
| Tailpipe/Extended Useful Life - VOC | $ 504 | $ 550 |
| Tailpipe/Extended Useful Life - $NO_x$ | 1,500 | 1,700 |
| 2 gram $NO_x$ Heavy Duty Standard[1] | 0 | 69 |
| Onboard Vapor Recovery | 63 | 69 |
| Cold Temperature CO Standard | 370 | 410 |
| Onboard Diagnostics | 880 | 960 |
| Evaporative/Running Losses | 42 | 46 |
| **Fuels:** | | |
| Phase II RVP | 280 | 330 |
| Federal Reformulated Gasoline | 720 | 860 |
| California Reformulated Gasoline | 2,000 | 2,400 |
| Oxygenated Fuels | 170 | 204 |
| California Reformulated Diesel | 170 | 230 |
| Low Sulfur Diesel Fuel | 570 | 740 |
| Stage II Vapor Recovery | 71 | 86 |
| **Motor Vehicle Total** | **$ 7,300** | **$ 8,700** |
| **Nonroad Engines/Vehicles:** | | |
| Phase I CI engine standards | $ 22 | $ 32 |
| Phase I and II SI engine standards | 56 | 104 |
| Federal locomotive standards[1] | 0 | 35 |
| Federal commercial marine vessel standards[1] | 0 | 1 |
| Federal recreational marine vessel standards | 27 | 230 |
| **Nonroad Engine Vehicle Total** | **$ 104** | **$ 400** |
| **Total Title II Costs** | **$ 7,400** | **$ 9,100** |

Notes:
Columns may not sum to totals due to rounding.
[1] Costs under the 2000 Post-CAAA scenario are zero because emission reductions are not realized until after 2000.

**Table B-16**
**Title III, MACT Standards, Point and Area Source VOC Control Costs**

| | Annual Costs by Sector (in million 1990 dollars) | |
|---|---|---|
| **Source Category** | **Post-CAAA 2000** | **Post-CAAA 2010** |
| Benzene NESHAP | $ 0.2 | $ 0.2 |
| 2-Year MACT: | | |
| Dry Cleaning-Perchloroethylene | 28 | 31 |
| SOCMI HON | 26 | 29 |
| 4-Year MACT: | | |
| Aerospace Industry (surface coating) | 4 | 5.3 |
| Chromium Electroplating[1] | 17 | 17 |
| Coke Ovens | 21 | 21 |
| Commercial Sterilizers[1] | 7 | 7 |
| Gasoline Distribution-Stage I | 12 | 13 |
| Halogenated Solvent Degreasing | (37) | (42) |
| Industrial Process Cooling Towers[1] | 14 | 14 |
| Magnetic Tape[1] | 0.8 | 0.8 |
| Marine Vessels | 17 | 20 |
| Medical Waste Incineration[1,3] | 89 | 89 |
| Municipal Waste Combustors[1] | 43 | 43 |
| Off-Site Waste Treatment[2] | - | - |
| Petroleum Refineries-Other Sources Not Distinctly Listed | 160 | 180 |
| Printing/Publishing | 200 | 207 |
| Polymers & Resins Group I | 110 | 128 |
| Polymers & Resins Group II | 4.3 | 5.0 |
| Polymers & Resins Group IV | 5.3 | 6.7 |
| Secondary Lead Smelters[1] | 2.0 | 2.0 |
| Shipbuilding and Ship Repair | 8.5 | 11 |
| Wood Furniture (surface coating) | 49 | 50 |
| **TOTAL** | **$ 780** | **$ 840** |

Notes:

1 Costs reflect estimates of annualized costs from final rules. Source categories are not modeled in ERCAM-VOC because the NESHAPs are associated with non-VOC HAP emission reductions.

2 The Off-site Waste Treatment NESHAP was not modeled in this analysis. We assume that the Title I modeling of the RCRA Phase I and Phase 2 rules for hazardous waste TSDFs will capture any future MACT reductions and costs.

3 EPA estimated that the Medical Waste Incineration guideline would cost between $59 million per year to $120 million per year, depending on the extent to which affected facilities switch to less expensive methods of treatment and disposal. The cost above represents the midpoint of this range.

## Title III

Title III of the CAAA requires the promulgation of MACT regulations to control HAP emissions from specific source categories. Total Title III costs represent the TACs for individual two- and four-year MACT standards. Not all Title III regulations are modeled by ERCAM-VOC because Title III regulations target HAP emissions which are not included in the Section 812 base year inventory. To provide a complete cost accounting, we use the annual cost estimates from the final rules for MACT standards that are expected to reduce non-VOC HAP emissions. The cost for these MACT categories is $173 million. The cost estimates for Title III are summarized in Table B-16. Costs do not differ significantly under the Post-CAAA 2000 and the Post-CAAA 2010 scenarios because we do not derive the costs from ERCAM-VOC using future year emission estimates.[23]

## Title IV

Title IV of the CAAA is the Acid Deposition Control Program. Title IV controls include $SO_2$ and $NO_x$ controls at electric utilities and are summarized in Table B-17, below. The model for $SO_2$ controls incorporates EPA's program for $SO_2$ allowance trading. The annual national costs of Title IV of the CAAA are $2.3 billion in 2000 and $2.0 billion in 2010. The decline in annual costs from 2000 to 2010 results primarily from the increase in use of Western coal, the cost of which we project will decrease over time. Cost reductions also occur following the increased use of gas-fired combined-cycle units to generate electricity without $SO_x$ emissions. As employed technology becomes more efficient, we expect that generation costs will decrease.

**Table B-17**
**Annual Costs of Title IV**

| | Annual Costs (million 1990 dollars) | |
|---|---|---|
| Title | Post-CAAA 2000 | Post-CAAA 2010 |
| Title IV | $ 2,300 | $ 2,000 |

Note: These estimates reflect the base case used in the Clean Air Power Initiative. See EPA, "Analyzing Electric Power Generation Under the CAA" (1996) for detail on scenario development. For more information on its application to the cost analysis, see EPA, 1997a.

## Title V

Title V of the CAAA establishes requirements for a new operating permits program. Using costs from final regulations rather than models, we estimate that Title V will cost $300 million. Consequently, we base this on the estimated cost of State-developed programs, excluding Federally-implemented State programs. The States are expected to implement all Title V permit programs by 2005. We estimate each source's permit fees and administrative costs in the first five-year implementation period, including the explicit cost to the permitted sources (industry), State and local permitting agencies, and EPA. The $300 million cost estimate may be an overestimate, since many States already have operating permit systems with fee provisions in place, and we do not incorporate existing state programs into the baseline in the RIA documents (EPA, 1992a and EPA, 1995).

# Social Costs

In an ideal setting, a cost-benefit analysis would not only identify but also quantify and monetize an exhaustive list of associated social costs due to a regulatory option. This would include assessing how regulatory action targeting a specific industry or set of facilities can alter the level of production and consumption in the directly affected market and related markets. For example, regulation of emissions from the electric utility industry that results in higher electricity rates would have both supply-side and demand-side responses. In secondary markets, the increased electricity rates affect production costs for various industries and initiate behavioral changes (e.g., using alternative fuels as a substitute to electric

[23] We do not estimate the costs associated with seven- and ten-year standards due to the lack of adequate data regarding the implementation of these standards.

power). With each affected market, there are also associated externalities that should be included in estimating social costs. Returning to the utilities example, the externalities associated with electric power generation versus nuclear power generation can be very different. The mix of externalities could change as consumers substitute nuclear power for electric power. It is frequently difficult to accurately characterize one or all of these dimensions of market responses and estimate the resulting social costs.

There are three generally practiced approaches to estimating regulatory costs: (i) direct compliance cost, (ii) partial equilibrium modeling, and (iii) general equilibrium modeling. The direct compliance cost approach is the most straightforward of the three. Direct compliance cost estimates are calculated differently than economic welfare impact estimates that result from partial or general equilibrium. This technique develops *ex ante* estimates of increased production costs, and may in some cases (such as in the case of the IPM model for utilities in our analysis) measure supply-side response to a regulatory action by modeling changes in supply price and quantity. In general, the technique does not account for how demand and consumption levels may change in response to higher production costs and prices. Instead, this approach measures how an industry's or firm's marginal cost curve shifts due to the additional production costs associated with pollution abatement controls.

The direct compliance approach, however, is a reasonable estimate of incremental expenditures. In certain instances, this method may be a conservative approximation of primary social costs because it overstates direct costs by not reflecting efficiency enhancing demand-side responses. There are two major difference between direct and social costs that influence the results. First, direct cost methods may overstate the actual compliance costs that are associated with demand and consumption level changes in response to higher production costs. By not accounting for market responses, total direct costs reflect the incremental costs per unit of output multiplied by the higher, pre-regulation quantity produced. Second, a direct cost approach assumes firms incur the full costs of pollution abatement activities. The marginal cost curves of firms, however, do not necessarily increase by the full amount of the pollution abatement technology. For example, firms can adopt cost-saving activities that help to offset the new costs.[24] (Morgenstern *et al.*, 1998).

Capturing consumer and producer behavioral responses to regulatory action requires either partial or general equilibrium modeling. These more complicated approaches estimate social costs by accounting for a wider range of consequences associated with altered resource allocation due to the use of pollution abatement equipment. A partial equilibrium analysis requires modeling both supply and demand functions in affected markets. Therefore, measures of social cost reflect behavioral responses by both producers and consumers in one or more markets. The variation between results from a direct cost approach and a partial equilibrium approach will generally depend on the extent price and quantity demanded change. Moreover, the estimates of a partial equilibrium model can overstate or understate total, economy-wide social costs depending on the type of existing market distortions and the extent to which there are spillover effects from the targeted sector to other economic sectors.

The partial equilibrium approach is particularly appropriate when social costs are predominantly incurred in a limited set of directly affected markets, and has minimal effects on other sectors. In cases where the regulatory action is known to have an impact on a broad range of sectors in the U.S. economy, the general equilibrium model can be a more appropriate approach to estimating social costs. Like the partial equilibrium model, the general equilibrium model estimates social costs by accounting for direct compliance costs and producer and consumer dynamics. The general equilibrium model can capture large and small first-order effects that occur in multiple sectors of the economy.

It is difficult to determine the relationship between general equilibrium estimates and direct cost estimates. Relative to the general equilibrium estimates, direct cost estimates are likely to overstate costs in the primary markets because they do not reflect consumer and producer responses. General equilibrium estimates, however, have a broader basis from which to estimate social costs and reflect net social welfare changes across the economy's economic sectors. There still remain significant barriers to

[24]Morgenstern et al. (1998) estimates the multiplier of abatement expenditures to total costs can be as low as 0.8.

assessing the potential magnitude and direction of actual total welfare changes, as experienced by the economy as a whole, to those estimated by a general equilibrium model.[25] Without insight into the accuracy of general equilibrium model estimates, it is difficult to characterize how direct cost estimates relate to general equilibrium cost estimates.

In the 812 retrospective analysis (EPA, 1997), we opted for the general equilibrium approach, recognizing that the Clean Air Act has a pervasive impact on the U.S. economy. Moreover, the retrospective nature of the analysis provided us with fairly well-developed historical data sets of goods and service flows throughout the economy. These data sets facilitated the construction of extensive expenditure data from which we modeled producer and consumer behavior and estimated net social costs. In the retrospective, our central estimate of total annualized costs, from 1970 to 1990, was $523 billion. In comparison, the aggregate welfare effects were estimated between $493 and $621 billion.[26]

Although a general equilibrium approach represents a theoretically preferable, and potentially more accurate, method for measuring social costs, as described in Chapter 3 we adopted a direct compliance cost approach for the prospective analysis. We selected the simpler direct cost modeling method for three reasons:

- First, we believe that the direct cost approach provides a good first approximation of the economic impact of the CAAA on the U.S. economy. For example, recent research suggests that *ex ante* analyses of regulatory costs are far more likely to overstate than understate costs.[27] In addition, the direct cost approach, because it does not reflect adjustments to prices and quantities that might be adopted to mitigate the effects of regulation, likely overstates the producer surplus loss to the entity that incurs the pollution control cost expenditure. Under these conditions, direct cost may actually overestimate the social costs of a particular economic sector. It is also possible that the direct cost estimates understate the effects of long-term changes in productivity and the ripple effects of regulation on other economic sectors.

- Second, we believe that the precision in estimating social costs that might be gained through a general equilibrium approach could be compromised by the difficulty and uncertainty associated with projecting future economic and technological change. The general equilibrium approach could provide many insights that the direct cost approach cannot, but as a tool for estimating social costs it is very data-intensive and introduces a significant level of additional uncertainty as a result.

- Third, undertaking a general equilibrium modeling exercise remains a very resource-intensive task. In light of our concerns about the potential gains in precision or accuracy of our social costs estimates for the purposes of comparing costs to benefits, we concluded that more detailed modeling would not be the most cost-effective use of the project resources.

## Limitations and Uncertainties

Several factors contribute uncertainty to the cost estimates.

- **Emissions Projections.** We base total cost estimates for individual CAAA provisions on projected emission reductions in 2000 and 2010. As a result, the quality of the cost

[25] Harrington et al. (1999): "The general equilibrium effects of environmental regulations are likely to be important, but are likewise impossible to examine empirically. Computable general equilibrium models have not been tested against real-world outcomes and may be untestable."

[26] Estimates are presented in 1990 dollars. The retrospective states, "In general the estimated second order macroeconomic effects were small relative to the size of the U.S. economy." The rate of long term GNP growth between the control and no-control scenarios amounted to roughly one-twentieth of one percent less growth. It is important to note that although the difference is small, the direct compliance cost method does represent an underestimation.

[27] See, for example, Harrington et al (1999) for a comparative analysis of *ex ante* and *ex post* regulatory cost estimates.

analysis results is dependent, in part, on the quality of the emission projection estimates.

- **Evolving Rules.** We estimate many of the costs based on assumptions regarding how stringent evolving or draft rules may be when finalized. Costs are likely to change as these rules are amended or finalized.

- **Facility response to regulation.** Facilities may respond to regulations in a manner different from our model assumptions and thereby affect cost estimates. The cost estimates for individual CAAA provisions will ultimately depend on the mix of compliance options facilities choose to meet each rule's requirements. In addition, we do not quantify the effect of economic incentive provisions, which provide greater flexibility to facilities affected by the rules.[28]

- **SIPs for meeting ozone NAAQS.** It is difficult for us to predict how States will design control plans for meeting ozone NAAQS attainment requirements.

- **Technology assumptions.** We develop costs based on data for today's technologies. To the extent that control technologies improve over time and lower cost control alternatives become available, we may overstate costs.

- **Discount rate.** Discount rates affect costs. In some instances of this cost analysis, we use data sources that do not explicitly list the applied discount rate assumptions.

In this section, we first discuss the impact of the above listed key limitations. We then identify cost inputs and conduct quantitative uncertainty analyses for those factors. Table B-18 summarizes the limitations and the likely effect on the cost analysis results.

[28] For example, the cost savings associated with a cap-and-trade program, such as South Coast Air Quality Management's Regional Clean Air Incentive Market (RECLAIM), is not reflected in the prospective cost assessment.

**Table B-18**
**Potential Effects of Uncertainty on Cost Estimates**

| Description | Potential Effect on Cost Estimates |
|---|---|
| Innovations in future emission control technology | Decrease |
| Emission projections: | |
| Growth factors/activity indicators | Unknown |
| RACT controls for individual States | Unknown |
| Inclusion of economic incentive provisions[1] | Decrease |
| Use of costs for rules that are currently in draft form (not yet finalized) | Unknown |
| Uncertainty of final State strategies for meeting Reasonable Further Progress (RFP) RFP requirements | Unknown |
| Inclusion of 7- and 10-year MACT standards | Increase |
| Revisions to Title V cost estimate to reflect current State permit program costs | Decrease |

Note:
[1] Examples include banking, trading, and emissions averaging provisions.

## Emission Projections

The selection of activity indicators for individual source categories can have significant impacts on projected emissions, and in turn, on the cost estimates in this analysis. In addition, we select RACT controls based on representative controls, yet the controls for individual States/facilities may differ from these representative controls.

## Draft Rules

EPA is currently revising several promulgated rules in response to public comments, legal actions, or other factors. The cost data used in this analysis reflect the latest available estimates, yet these costs are subject to change as the Agency modifies existing rules. For example, while we were developing CAAA costs, the Agency was also proposing a rule to limit summer season $NO_x$ emissions in a group of OTAG-participating States. Cost estimates for the regional OTAG $NO_x$ strategy will most likely be different than those for the Ozone Transport Rulemaking due to uncertainty about the final form of the rulemaking.

In an effort to maintain consistency between emission and cost data, we do not update its costs to reflect modification to drafted and existing rules. For example, because EPA revised the ozone and PM NAAQS *after* projecting emissions, we continued to use earlier cost estimates that are consistent with the prior NAAQS control assumptions. Another example is that of estimating cost for the proposed compression ignition (CI) engine Phase II rule which was not proposed by the time we completed our emissions projection for the nonroad sector. Although costs are now available for the Phase II rule, to maintain consistency with the benefits analysis we include only Phase I costs.[29]

In general, rule amendments such as exemptions for particular types of sources or opportunities for sources to postpone compliance dates are likely to ease the regulatory burden on regulated sources, and therefore, will result in lower total costs than those estimated in this analysis.

## Economic Incentive Provisions

EPA created economic incentive provisions in several rules to provide flexibility for affected facilities that comply with the rules. These provisions include banking, trading, and emissions-averaging provisions. Flexible compliance provisions lower the cost of compliance. For example, the emissions-averaging program grants flexibility to facilities affected by the marine vessels rule, the petroleum refinery NESHAP, and the gasoline distribution NESHAP; these facilities can choose which sources to control, as long as they achieve the required overall emissions reduction. In many of the cost analyses, we do not

[29] However, this implies CI nonroad engine rule costs are understated.

attempt to quantify the effect that economic incentive provisions will have on the overall costs of a particular rule. In these cases, to the extent that affected sources use economic incentive provisions to minimize compliance costs, costs may be overstated.

### Reasonable Further Progress (RFP) and Attainment Costs

Considerable uncertainty surrounds the development of States' control plans for meeting ozone NAAQS attainment requirements. We develop the RFP cost estimate by assuming that ozone nonattainment areas (NAAs) will take credit for $NO_x$ reductions for meeting progress requirements. Additional area-specific analysis would be necessary to determine the extent to which areas find $NO_x$ reductions beneficial in meeting attainment and progress requirement targets. Trading of $NO_x$ for VOC to meet RFP requirements may result in distributions of VOC and $NO_x$ emission reductions that differ from those used in this analysis.

### Future Year Control Cost Assumptions

The regulatory documents which provide cost inputs to ERCAM and the IPM contain the most recent data available, given existing technological development. Between 2000 and 2010, additional control technologies will allow sources to comply with CAAA requirements at lower costs. For example, we anticipates technological improvements for complying with the multiple tiers of proposed emission standards during the phase-in of nonroad engine controls; these improvements will likely lead to reduced costs. In addition, the costs for certain control equipment may decrease over time as demand increases. The trend in cost of selective catalytic reduction (SCR) costs illustrate this. Costs have decreased over the past three years as more facilities begin to apply the technology. We also believes that even in the absence of new emission standards, manufacturers will eventually upgrade engines to improve performance or to control emissions more cost-effectively; firms will institute technologies such as turbocharging, aftercooling, and variable-valve timing, all of which improve engine performance.

### Discount Rate Assumptions

We apply a rate of five percent to both the discount rate and cost of capital. In some cases, we base costs on analyses that apply alternative discount rate assumptions (usually seven percent). Whenever possible we recalculate total annualized costs (TAC) for these rules in an effort to maintain consistency. We use TACs, in turn, to calculate cost per ton estimates that are applied to cost-equations. For some source categories, there was insufficient data available to identify the discount rate assumptions used in the TAC estimate and the relevant cost per ton estimates. For example, the national municipal landfills rule applies only to facilities that emit above 50 megagrams of non-methane organic compounds (NMOC). However, the cost analysis for the proposed rule used a different emissions cutoff (150 megagrams of NMOC). Because we did not revise the cost analysis to reflect the new cutoff, it is impossible to replicate the calculations used to estimate the TAC in the final rule. Additional research would be necessary to calculate costs for the national municipal landfills rule under alternative discount rate assumptions.

### Source-Specific Cost Equations

We estimate the costs of Title III control measures for point and area source emitters using an average annual cost per ton value. For future analyses, assuming sufficient data are available, it may be possible to develop source-specific control equations using a similar approach to that used for point source $NO_x$ emitters. The point source inventory generally includes larger, inventoried point sources, and the area source inventory includes emissions for smaller emission points. For this reason, we try to determine whether sufficient cost data by plant size are available to model costs specific to smaller plants, rather than using an overall cost effectiveness value across all plant sizes. If costs are available for sources by size, we apply the cost estimates for larger sources to point sources and apply cost estimates for smaller model plants to area sources. We do not, however, use this approach in all cases due to insufficient data.

## Sensitivity Analyses to Quantify Key Uncertainties

We develop cost estimates based on a variety of studies and assumptions regarding future behavioral responses to provisions of the CAAA. These assumptions (i.e., changes in consumption patterns, input costs, and technological innovation) introduce some uncertainty to the cost projections. In order to characterize the potential importance of these uncertainties with respect to several provisions, we conduct sensitivity tests on selected Post-CAAA 2010 cost estimates. They are:

- Progress Requirements
- $PM_{10}$ Nonattainment Area Controls
- Non-utility Stationary Source $NO_X$ Costs
- California Reformulated Gasoline
- Low Emission Vehicle Costs
- $NO_X$ Tailpipe/Useful Life Standards

These provisions represent the most significant contributors to total costs. Collectively, they constitute nearly half of the total 2010 Post-CAAA estimated costs. In addition, we examine the impact of alternative discount rates on the cost assessments. We summarize the results of the sensitivity analyses in Table B-19.

A significant portion of the cost of attaining and maintaining the one-hour average ozone NAAQS is attributable to rate of progress (ROP) and rate of further progress (RFP) compliance expenses. The costs associated with reducing VOC (and $NO_x$) emissions in order to satisfy these progress requirements are particularly difficult to estimate because cost-effective control measures have not been identified that will readily enable some ozone NAAs to make the required precursor emissions cuts. The estimated costs of unidentified VOC controls is one source of uncertainty that affects the overall cost of ROP/RFP requirements.

In the prospective, we assume that the cost of Title I progress requirements is equal to the cost of eliminating the VOC shortfall. We expect NAAs will reduce VOC emissions using identified control measures. The cost-effectiveness of each of these measures is known, and we assume that the control technique yielding the greatest reduction per dollar is the first to be implemented, followed by the second most cost-effective option, and so on until further VOC cuts are no longer necessary to satisfy ROP/RFP requirements.

We estimate that it will be possible to sufficiently lower VOC emissions through implementation of identified VOC controls for every NAA with a shortfall, except Chicago and Milwaukee. These two exceptions, however, have NOx waivers and cannot credit $NO_x$ cuts towards RFP requirements. As a result, they will have to significantly lower VOC emissions; the necessary reduction is so sizable in both areas that neither will be able to make the required cuts, even if it adopts all of the identified control measures. Thus, in order to satisfy ROP/RFP requirements, Chicago and Milwaukee will have to implement unidentified VOC emissions control techniques. The estimated costs associated with these measures are a source of uncertainty potentially influencing the overall cost of Title I progress requirements. We conduct a sensitivity analysis to help characterize the influence of this uncertainty on the 2010 progress requirements cost estimate.

We base the three scenarios of the sensitivity analysis upon different assumptions regarding the cost-effectiveness of VOC shortfall controls. For the lower estimate, after applying identified controls (the approximate cost per ton of reduction is known for these measures), the remaining shortfall is eliminated through the implementation of unidentified controls. In the lower estimate scenario, we estimate the marginal cost of these unidentified measures is equal to the weighted average of the cost per ton estimate from the recently revise ozone NAAQS RIA, which is $10,000, and the average dollar per ton cost for identified measures.[30] The central estimate is identical to the lower estimate with one exception, unidentified controls are assumed to cost $10,000 per ton of

[30] For example, in Chicago sixty percent of the required reduction of VOC emissions will come from identified measures at an average cost of $3,500 per ton. The remaining forty percent will thus come through unidentified controls. This means that, according to the lower estimate, the approximate cost per ton of reduction through the implementation of unidentified controls is $6,000 [$3,500(.60) + $10,000(.40)].

reduction in the central scenario. A flat $10,000 per every ton of shortfall VOC emissions reduced, from both identified and unidentified measures, is assumed for the upper estimate. Our sensitivity analysis shows that ROP/RFP costs range from $0.61 billion to $2.5 billion, with a central estimate of $1.1 billion. We provide a more detailed breakdown of these costs in Table B-20. It is important to note, that this sensitivity analysis is the only case in which the primary estimate of our cost analysis differs from the central case in a sensitivity test.

**Table B-19**
**Factors Affecting Cost of Major CAAA Provisions**

| Provision | Factors Affecting Cost | Conduct Sensitivity Analysis? | Strategy for Sensitivity Analysis | Potential Effect of Uncertainty on Post-CAAA 2010 Cost Estimates [1] |
|---|---|---|---|---|
| Progress Requirements | Cost for unidentified measures is most uncertain. | Yes | Continue to examine costs of identified measures in other specific areas.<br><br>Lower Bound: Assume average per ton cost of identified measures (e.g., $3,500 in Chicago) for all reductions, including unidentified measures.<br>Central Estimate: Use cost figure for identified measures for that fraction of reductions (e.g., $3,500 for 60 percent in Chicago) and assume $10,000 per ton cost for unidentified measures. This central estimate reflects more recent cost per ton information than was applied to our primary cost estimate.<br>Upper Bound: Assume $10,000 per ton cost for all reductions, including identified measures. Our cost analysis adopts a conservative approach and applies this cost per ton value to our primary cost analysis. | Central Estimate: $1.1<br><br>Range: ($0.06 - $2.5) |
| | Impact of revised ozone standard. | No | Emissions projections in the 812 Prospective do not include revisions to the ozone NAAQS. | no estimate |
| California Reformulated Gasoline | Incremental fuel costs show wide range and are most uncertain. | Yes | Lower Bound: Assume 7.3 cents per gallon cost from CARB.<br>Central Estimate: Current analysis assumes 12.3 cents per gallon cost from CARB.<br>Upper Bound: Assume 17.3 cents per gallon cost from CARB. | Central Estimate: $2.5<br><br>Range: ($1.4 - $3.5) |
| | Gasoline sale quantities are important, but less uncertain. | Yes | Gasoline sales are a function of vehicle miles traveled (VMT).<br>Apply alternative VMT projection for California: *California Motor Vehicle Stock, Travel, and Fuel Forecast*, California Department of Transportation, November 1997.<br>Alternative VMT would impact emission scenario. | |

| Provision | Factors Affecting Cost | Conduct Sensitivity Analysis? | Strategy for Sensitivity Analysis | Potential Effect of Uncertainty on Post-CAAA 2010 Cost Estimates [1] |
|---|---|---|---|---|
| PM NAA Controls | Base year emissions and growth. | No | Emissions projections and growth estimates are underlying assumptions of the cost analysis.[2] | no estimate |
| | Area specific plans may differ from the "model plan" applied. | Yes | Strategy for sensitivity analysis includes:<br>1) Apply area-specific control measures where available.<br>2) Use "model plan" when area plans are unknown. | Central Estimate:<br><br>$2.2 |
| | Cost per ton estimates and effectiveness of individual measures. | Yes | Apply upper and lower bound cost estimates for model plan controls based on the SCAQMP, the MRI study of agricultural operations, and the PM NAAQS study:<br>Agricultural Tilling: Low $154/ton (1997 SCAQMP)<br>High $5,900/ton (midpoint of range from MRI study)<br>Construction: Low $1,900/ton (50% below value used)<br>High $5,700/ton (50% above value used)<br>Paved Roads: Low $50/ton (1997 SCAQMP)<br>High $1,350/ton (50% above value used)<br>Unpaved Roads: Low $560/ton (1997 SCAQMP)<br>High $2,700/ton for rural roads (PM NAAQS) | Range:<br><br>($0.9 - $3.3) |

| Provision | Factors Affecting Cost | Conduct Sensitivity Analysis? | Strategy for Sensitivity Analysis | Potential Effect of Uncertainty on Post-CAAA 2010 Cost Estimates [1] |
|---|---|---|---|---|
| LEV Costs | Will 49-State LEV occur? | No | Recently agreed to by the 23 automobile manufacturers that sell cars in the US and are regulated by EPA. Four States in the Northeast (MA, ME, NY, VT) have opted not to join the NLEV program. | no estimate |
| | Per vehicle costs. | Yes | Current analysis uses CARB's per vehicle cost estimates. These estimates are the lowest and most fully documented, and differ from other industry estimates by a factor of ten.<br><br>Lower Bound: 50% below study per vehicle cost estimates.<br>Central Estimate: Use current study (CARB adjusted for national sales volume) per vehicle cost estimates.<br>Upper Bound: Use unadjusted CARB per vehicle cost estimates. | Central Estimate: $2.2<br><br>Range: ($1.08 - $2.5)<br><br>(national and CA LEV combined) |
| | Projected vehicle sales. | Yes | Vehicle sales data were obtained from EPA's onboard vapor recovery RIA.<br><br>Apply alternative sales projection: DOE's *Annual Energy Outlook 1998* - NEMS Transportation Demand Model. | |

| Provision | Factors Affecting Cost | Conduct Sensitivity Analysis? | Strategy for Sensitivity Analysis | Potential Effect of Uncertainty on Post-CAAA 2010 Cost Estimates [1] |
|---|---|---|---|---|
| Non-Utility Stationary Source NOx Costs | Unit-level cost equations and cost per ton. | Yes | ICI boilers account for 79 percent of the total point source NOx control cost estimate in 2010. Apply ±50 percent range. Other available data are 3-4 years old and would not reflect the fact that the control technology is being manufactured and applied by more sources. | Central Estimate: $2.2<br><br>Range: ($1.1 - $3.2) |
| | Inventory data elements (e.g., capacity, operating rate) used in cost calculations. | No | Inventory data elements are well-defined for each point source category. | no estimate |
| | Cap applied to 37 States, proposed NOx budgets affect only 22 States. | No | Current estimates overstate costs for fuel combustors in the 15 States not affected by the NOx cap. NOx SIP call RIA provides estimates for 22-state program. | no estimate |
| | Banking not accounted for. | No | None | no estimate |
| NOx Tailpipe/Useful Life Standards | Per vehicle costs date to 1991 FR Notice. | Yes | Lower Bound: No alternative estimates. Scale down medium estimate by 50 percent.<br>Central Estimate: Use current $115 estimate from EPA.<br>Upper Bound: No alternative estimates. Scale up medium estimate by 50 percent. | Central Esimate: $1.7<br><br>Range: ($0.83 - $2.5) |
| | Vehicle Sales | Yes | Same as LEV projected vehicle sales. See above. | |
| High Enhanced I/M | Per vehicle costs are most uncertain. | No | Alternative per vehicle cost estimates are similar to the costs currently used in the model. | no estimate |

| Provision | Factors Affecting Cost | Conduct Sensitivity Analysis? | Strategy for Sensitivity Analysis | Potential Effect of Uncertainty on Post-CAAA 2010 Cost Estimates [1] |
|---|---|---|---|---|
| | Vehicle registrations are important, but less uncertain. | No | 1990 vehicle registrations are well-documented, and the method used to project future registrations based on population projections is sound. | no estimate |
| Discount Rate | Vary the discount rate. | Yes | Current cost estimates use a five percent discount rate. Vary the cost estimates using two alternative discount rates, three percent and seven percent. | Central Estimate: $3.0<br><br>Range: ($2.8 - $3.2) |
| Economic Growth Case Study | Macroeconomic growth projections may affect cost drivers. | No | The current methodology for calculating PM emissions relies on activity level projections more than macroeconomic growth rates. For example, the model uses the USDA agricultural baseline projections of farm acres planted to calculate PM emissions from agricultural production, the largest source of PM. The PM sources influenced by macroeconomic growth rates contribute only about five percent of total PM emissions. | no estimate |

Notes: [1]Estimates are in billion 1990 dollars.

An additional source of uncertainty associated with estimating the cost of ROP/RFP requirements stems from the fact that the impact of the revised ozone NAAQS is not incorporated into the Post-CAAA scenario. We do not, however, conduct a sensitivity test designed to characterize the influence of the stricter NAAQS. In this instance, developing a cost range would not be very meaningful since benefits, as well as costs, would be affected by the changed NAAQS.

**Table B-20**
**Rate-of-Progress Cost Sensitivity Summary**

| Ozone Nonattainment Area | Annual Post-CAAA 2010 Costs (million 1990 dollars) | | |
|---|---|---|---|
| | Low Estimate | Central Estimate | High Estimate |
| Chicago-Gary-Lake County | $ 430 | $ 810 | $ 1,400 |
| Dallas-Fort Worth | 0.4 | 0.4 | 61 |
| El Paso | 8.5 | 8.5 | 76 |
| Grand Rapids | 0.2 | 0.2 | 27 |
| Los Angeles-South Coast | 0.8 | 0.8 | 44 |
| Louisville | 0.1 | 0.1 | 30 |
| Milwaukee-Racine | 120 | 210 | 430 |
| Muskegon | 0.6 | 0.6 | 9.3 |
| Nashville | 15 | 15 | 130 |
| Salt Lake City | 28 | 28 | 180 |
| Sheyboygan | 0.5 | 0.5 | 8.2 |
| Kewaunee Co. WI | 0.2 | 0.2 | 1.7 |
| Monitowoc Co. WI | 1.1 | 1.1 | 11.3 |
| **Total** | **$ 607** | **$ 1,080** | **$ 2,500** |

$PM_{10}$ nonattainment area controls account for roughly seven percent of total annual costs in 2010. Two sources of uncertainty with respect to our estimate are: (i) how well the model plan mirrors actual application of controls in nonattainment areas, and (ii) how representative the cost per ton estimates used in the model are of actual control measure costs. We develop a sensitivity analysis that incorporates both factors. We analyze how well the model replicate selected SIP controls by applying the prospective cost equations to areas that have already implemented controls.[31] The second part of the sensitivity analysis assesses the impact of higher and lower cost per ton estimates on the original set of control measures. In cases where there were no alternative estimates, we adjust values up and down by fifty percent.[32]

[31] We use a survey of SIPs designed in 1991 and implemented in 1995.

[32] The sensitivity analysis does not reflect point source controls.

Our analysis highlights two primarily differences between model and actual SIP designs. First, the model plan probably overstates the application of fugitive dust controls in practice. Second, over half of the areas that have adopted emission measures emphasized point source controls, at lower cost than reductions that could be achieved through other measures. Testing the uncertainty of cost per ton values resulted in a range between $0.9 and $3.3 billion in $PM_{10}$ nonattainment costs. Total area source PM control costs had a low estimate of $1.5 billion and a high estimate of $3.3 billion. We summarize these results in Table B-21.

**Table B-21**
**Area Source PM Control Cost Sensitivity Analysis, Year 2000**
(in million 1990 dollars)

| Source Type | Central Estimate | Model Plan Sensitivity Analysis | Low Estimate | High Estimate |
|---|---|---|---|---|
| Agricultural Burning | $ 39 | $ 23 | $ 20 | $ 58 |
| Agricultural Tilling | 3.9 | 1.5 | 0 | 20 |
| Beef Cattle Feedlots | 1.7 | 0.3 | 0.9 | 2.6 |
| Construction Activity | 1,700 | 1,070 | 870 | 2,600 |
| Paved Roads | 440 | 380 | 13 | 650 |
| Unpaved Roads | 0.8 | 0.5 | 0.2 | 1.2 |
| **Total** | **$2,200** | **$1,500** | **$907** | **$3,300** |

Note:
[1] Examples include banking, trading, and emissions averaging provisions.
* Costs are in millions of 1990 dollars.

Another significant portion of total CAAA cost is incurred by non-utility stationary $NO_X$ sources. This category contributes approximately eight percent of total costs in 2010. Its costs reflect unit-level costs for combustion processes at industrial, commercial, and institutional facilities. We identify the accuracy of future control costs assessment (nationwide and for the subset of OTAG states subject to the $NO_X$ SIP Call) as a source of uncertainty that may affect cost projections. Our evaluation of this uncertainty involves applying alternative unit cost estimates. For the sensitivity analysis, we estimate upper and lower estimates by varying prospective costs up and down by fifty percent.[33] As a result, the central estimate for 2010 is $2.1 billion, and costs range from $1.1 billion to $3.2 billion.

[33] After reviewing alternative cost studies sponsored by STAPPA/ALAPCO and the OTAG stationary source committee EPA found the studies relied on the same sources used by the 812 Project Team. Consequently, the agency opted for the above mentioned approach to varying cost inputs.

By 2010, we estimate California's reformulated gasoline program will cost $2.5 billion annually. The program is a significant factor in the our cost analysis, because it represents eight percent of total annual CAAA costs and ten percent of national gasoline sales. We identify two primary sources of uncertainty, projected car sales and projected gasoline consumption levels. The sensitivity analysis varies costs by applying the high and low cost per gallon estimates developed by CARB. The test indicates that cost may range between $1.4 and $3.5 billion. Moreover, we integrate into the sensitivity test alternative projections of car sales as a proxy for consumption levels.[34] California's Department of Transportation calculated VMT projection approximately five percent lower than the projection we use in the prospective. This lower VMT estimate estimaes costs of $2.27 billion in 2010 (compared with the central estimate of $2.45 billion).

Our estimate of low emission vehicle (LEV) costs are also subject to similar sources of uncertainty. We rely on assumptions regarding the types of implemented emission control technology, its associated costs (estimated as costs per vehicle), projected vehicle sales, and the extent to which LEV will be adopted around the country. We conduct sensitivity analyses for costs of both the California LEV program and a 49-State LEV program. The analyses reflect uncertainty with respect to cost per vehicle and vehicle sales. To test the uncertainty related to car sales, we use an alternative set of projections from the Department of Energy. The low estimate reflects per vehicle costs that are fifty percent below baseline costs. We use CARB's high estimates, which were unadjusted for national sales volume, to develop a high estimate. Our sensitivity analysis result are a low estimate of $0.55 billion and a high estimate of $1.34 billion for California's LEV costs in 2010. For a 49-State LEV program, the estimates range between $0.53 billion to $1.34 billion. The central estimates are $1.1 billion for the California LEV program and $1.06 billion for the 49-State LEV program. We summarize results in Table B-22.

[34] EPA used VMT projections by California Department of Transportation.

**Table B-22**
**Results of Sensitivity Analysis of LEV Costs**

| | Post-CAAA 2010 Annual Cost (million dollars) | | | |
|---|---|---|---|---|
| Program | Central | Low | High | Alternative VMT |
| California LEV | $ 1,100 | $ 550 | $ 1,100 | $ 870 |
| 49-State LEV | 1,060 | 530 | 1,300 | 820 |
| **TOTAL** | **$ 2,200** | **$ 1,080** | **$ 2,500** | **$ 1,700** |

Note: Columns may not sum to totals due to rounding

Costs associated with NOx Tailpipe/Useful Life Standards are a sizable portion of both Title II and total CAAA costs. By 2010, we estimate these costs will contribute nineteen percent of the Title II motor vehicle costs. As a share of total CAAA costs, it is six percent. Key sources of uncertainty are the same as those associated with LEV, per vehicle costs and projected car sales. Similarly, the sensitivity analyses: (i) scaled per vehicle costs up and down by fifty percent; and (ii) used alternative sales projections generated by the Department of Energy. The variation of cost inputs produced a cost range of $0.83 billion to $2.48 billion for 2010. Application of sales projections by the Department of Energy resulted in costs slightly lower than the central estimate, $1.25 billion compared with $1.65 billion respectively.

Unlike the other sensitivity analyses, the discount rate affects cost estimates for multiple provisions. As noted, we calculate total annualized cost estimates using a 5 percent discount rate. However, variations in the discount rate can potentially have a significant effect on the overall cost estimate, because the discount rate is also used as an estimate of the real cost of capital to finance pollution control equipment. Our sensitivity analysis of annualized focuses on source categories where available information is available to distinguish capital from operating and maintenance expenses. Source sectors and pollutants include non-utility VOC and $NO_X$ and area source VOC, $NO_X$, and PM.

We present the discount rate sensitivity analysis results in Table B-23. We estimate total annualized costs using three discount rates, three percent, five percent, and seven percent. As a result, costs estimates vary from two percent to fifteen percent. The results of the analysis do not assess how the discount rate would impact a large fraction of the total estimated costs in the Post-CAAA scenarios. Excluded costs include motor vehicles and $PM_{10}$ area source categories in nonattainment areas. Most of the capital costs associated with motor vehicle provisions are in the form of research and development. $PM_{10}$ area source costs are generally calculated using a cost per ton estimate, which does not have sufficient available data for identifying the discount rate.

**Table B-23**
**Discount Rate Sensitivity Analysis for 2010 Cost Estimates**

| Sector | Pollutant | Capital Cost (million 1990 dollars) Discount Rate 3% | 5% | 7% | Percent Difference Between 3% and 7% Rate |
|---|---|---|---|---|---|
| Non-Utility | VOC | $ 480 | $ 501 | $ 530 | 11% |
| | $NO_x$ | 1,400 | 1,500 | 1,600 | 12% |
| Area | VOC | 508 | 540 | 570 | 11% |
| | $NO_x$ | 17 | 18 | 20 | 15% |
| | PM | 430 | 440 | 440 | 2% |
| **Total** | | **$ 2,800** | **$ 3,000** | **$ 3,100** | **11%** |

The sensitivity analyses assess the potential effect of uncertainty on components of the total CAAA costs. We project costs for progress requirements, PM nonattainment area controls, non-utility $NO_X$ sources, and utility emissions, based on modeling future emission controls. Accurately identifying the set of controls that will be adopted introduces a key source of uncertainty. The analyses indicate that there may be considerable variability in the cost estimates. However, it is important to note that for most of these scenarios, the high estimates are most likely representative of upper bounds. There are two sources of uncertainty associated with the motor vehicle provisions. The first source is projecting future car sales. The second is the accuracy of per vehicle costs. The high and low estimates relative to the central estimate do not present as wide a range.

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# Air Quality Modeling

Appendix C

## Introduction

Section 812 of the 1990 Clean Air Act Amendments (CAAA) requires the U.S. Environmental Protection Agency (EPA) to perform periodic, comprehensive, prospective analyses of the costs and benefits associated with programs implemented pursuant to the Clean Air Act (CAA). Such analysis requires the estimation of future-year emissions levels and associated air-quality-related values for scenarios reflecting compliance with the CAA, as well as for scenarios for which the effects of programs associated with the CAA are not accounted for in establishing the future-year estimates. This report summarizes the results of an air quality modeling and analysis study designed to estimate the effects of the CAAA on future air quality. The Section 812 prospective study includes analysis of following criteria pollutants: ozone, particulate matter (PM), sulfur dioxide ($SO_2$), nitrogen oxide (NO), nitrogen dioxide ($NO_2$), and carbon monoxide (CO). Future-year estimates of these atmospheric constituents were obtained through the application of air quality modeling tools and techniques, as described in this report.

An integral component of the modeling analysis was the estimation of future-year emission levels associated with the two CAAA scenarios and two future years. Scenarios that incorporate the emission reductions associated with CAAA are referred to as Post-CAAA while those that incorporate growth but reflect 1990 regulations are referred to as Pre-CAAA. The two future years considered in the analysis are 2000 and 2010. The emissions estimates (Pechan, 1998) provide the basis for the estimation of ozone, PM, and other criteria pollutant concentrations associated with each scenario and future year.

Future-year estimates of ozone concentrations were obtained through the combined application of the Urban Airshed Model (UAM) and the variable-grid UAM (UAM-V), yielding urban- and/or regional-scale estimates of ozone concentrations for each scenario and future year for the entire U.S. (48 contiguous states).

Concentrations of primary and secondary PM for the future-year scenarios (including $PM_{10}$, with a diameter of less than 10 micrometers, and $PM_{2.5}$, with a diameter of less than 2.5 micrometers) were estimated through the combined application of the Regional Acid Deposition Model/Regional Particulate Model (RADM/RPM) and the Regulatory Modeling System for Aerosols and Acid Deposition (REMSAD). RADM/RPM was used for the eastern U.S., while REMSAD was applied to the analysis of PM within the western U.S.

An emissions-based, linear "roll-back" technique was used to estimate future-year concentrations for the other pollutants considered as part of this analysis – $SO_2$, NO, $NO_2$, and CO.

Following application of the modeling techniques, site-specific estimates of future-year air quality were obtained by adjusting observational data (corresponding to a base year of 1990) in accordance with the changes in air quality predicted by the modeling systems. Statistical quantities or "profiles" describing the predicted concentration distributions for each monitoring site were then calculated. The resulting statistical concentration distributions provide the basis for the examination and quantification of the effects of changes in air quality on health, agriculture, and the economy (i.e., physical effects and economic valuation modeling) resulting from compliance with the CAAA.

The remainder of this report summarizes the methods and results of the section 812 prospective air quality modeling analysis. An overview of the modeling/analysis methodology is provided in section 2. The methods and results for ozone are presented in section 3. The methods and results for PM are

provided in section 4. The linear-rollback modeling for the other criteria pollutants is summarized in section 5. A discussion of the attributes and limitations of the modeling analysis methodologies is provided in section 6. Finally, recommendations for further research are given in section 7.

## Overview of the Section 812 Prospective Modeling Analysis

The air quality modeling component of the section 812 prospective analysis included the application of a variety of air quality modeling tools and techniques, as well as the combined use of observational data and modeling results to estimate future-year concentrations of several criteria pollutants. An overview of the modeling approach is provided in this section of the report.

The overall objective of the modeling exercise was to provide base- and future-year estimates of ozone, PM, $SO_2$, NO, $NO_2$, and CO for the subsequent analysis of the effects of the CAAA on health, agriculture, and the economy within the continental U.S. Although the CAAA applies to the entire nation, due to geographical considerations, the modeling domain includes the contiguous 48 states. The modeling was performed for a base year (1990) and for four future-year scenarios. The future-year scenarios include Post-CAAA and Pre-CAAA scenarios (the former incorporating emission changes associated with measures and programs pursuant to the CAAA) for the years 2000 and 2010. These years were selected to accommodate implementation schedules and time for effectiveness periods associated with many of the CAAA measures and programs.

### *Air Quality Models and Databases*

To the extent possible, the section 812 prospective modeling analysis utilized existing modeling databases (from State Implementation Plan or other regional-scale modeling efforts). To accommodate the geographical extent and resolution required for this study, these included the input databases corresponding to both urban- and regional-scale applications of several different modeling systems. The lack of an existing comprehensive, fully tested, integrated modeling system (and associated databases) for use in this study precluded the integrated analysis of the various pollutants. This, however, may be an area for improving future prospective analyses.

The UAM and UAM-V modeling systems were applied to the analysis of the effects of the CAAA on ozone air quality. Specifically, the UAM-V modeling system was applied for the regional-scale analysis of ozone concentrations within both the eastern and western portions of the U.S. (separately). The analysis of the eastern U.S. relied upon the use of modeling databases developed as part of the Ozone Transport Assessment Group (OTAG) regional-scale modeling analysis. This modeling system was also applied for the western U.S., but at a relatively coarse resolution. To enhance the analysis for selected urban areas in the western U.S., the regional-scale modeling results were supplemented with higher-resolution modeling results for Los Angeles, Phoenix, and the San Francisco Bay Area. The results for both Los Angeles and Phoenix were obtained using the UAM modeling system, while those for the San Francisco Bay Area were obtained using the UAM-V modeling system.

The RADM/RPM and REMSAD modeling systems were used to estimate PM concentrations for the eastern and western U.S., respectively. Again, many of the inputs for application of these models were developed as part of other studies and adapted for use in the section 812 prospective modeling analysis.

As noted earlier, an emissions-based, linear "roll-back" technique was used to estimate future-year concentrations for $SO_2$, NO, $NO_2$, and CO. This approach was used for all areas of the continental U.S.

All of the modeling applications relied on the use of detailed estimates of emissions for the base year and each of the future-year scenarios. These are described by (Pechan, 1998). Modeling emission

inventories were prepared using the Emissions Preprocessing System (EPS2.5) (SAI, 1992).

## Methodology for the Combined Use of Observations and Air Quality Modeling Results

The 812 prospective modeling analysis included several steps. First, concentration estimates for each pollutant of interest, corresponding to a base year of 1990, were prepared based on 1990 emissions and application of the appropriate modeling tool(s). For each scenario, the remaining steps consisted of (1) preparation of future-year, model-ready emission inventory estimates, (2) application of the appropriate modeling technique to estimate the change in air quality from the base year of 1990, (3) adjustment of the 1990 observed data to reflect the change as predicted by the modeling system, and (4) calculation of statistical quantities or "profiles" describing the predicted pollutant concentration distribution for each monitoring site.

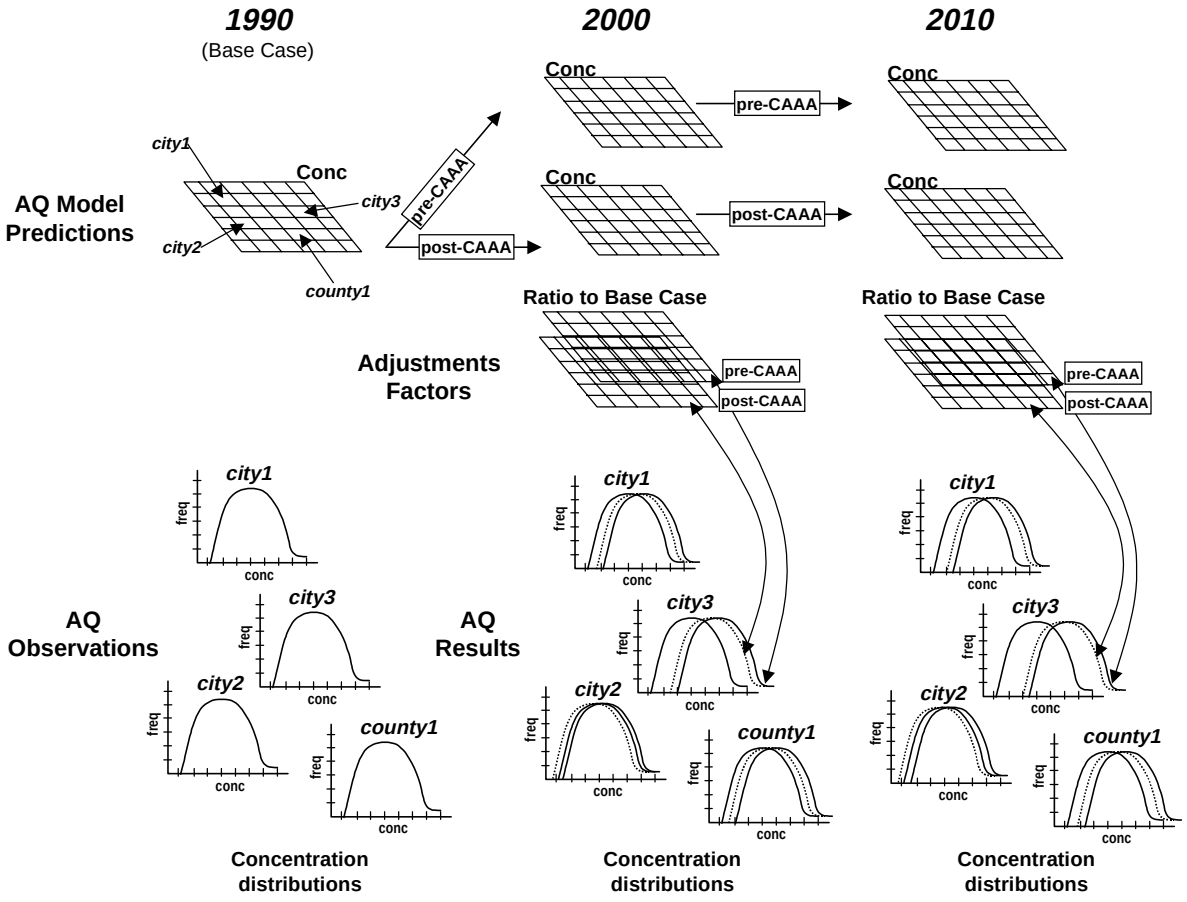
Conceptually, the methodology for estimating future-year ozone air quality using both observations and modeling results is rather simple. The modeling results are used to calculate adjustment factors for each monitoring site that is located within the modeling domain. This is done on a grid-cell by grid-cell basis (i.e., the adjustment factor for a monitoring site is based on the simulation results for the grid cell in which it is located). The adjustment factor represents the ratio of the future-year-scenario concentrations to the base-year concentrations and is calculated using appropriately matched values for several different concentration levels (i.e., the changes in concentration are dependent upon concentration level). The observed concentrations for each monitoring site are then modified using the site-specific (or grid-cell-specific) adjustment factors. The resulting values represent the estimated future-year concentrations.

This approach to estimating future air quality differs from that for a typical air quality model application (e.g., for ozone attainment demonstration purposes) in that the modeling results are used in a relative sense, rather than an absolute sense. This may enhance the reliability of the future-year concentration estimates, especially in the event that the uncertainty inherent in the absolute concentration values is greater than that associated with the response of the modeling system to changes in emissions.

Although the ratios are calculated using modeling results for a limited number of simulation days, it is assumed, using this methodology, that the ratios can be used to represent longer time periods. Consequently, all observations contained within the dataset (a few exceptions are discussed later in this document) are adjusted using the model-derived ratios. Thus, by applying the model-derived ratios to observed values representing longer periods, this approach also permits the estimation of seasonal and annual concentration distributions – a requirement for this study. Following the calculation of various n-hour rolling averages for each monitoring site, statistical quantities, or "profiles", describing the ozone distribution for each monitor are then calculated.

The future-year air quality profile estimation methodology, as applied to the analysis of results for the section 812 prospective analysis, is described in detail in the remaining sections of this document. A flowchart illustrating the methodology is provided in Figure C-1. The procedure makes use of the statistical functions and data handling capabilities of the Statistical Analysis Software (SAS) package.

**Figure C-1**
**Schematic diagram of the future-year concentration estimation methodology.**




[NOTE: Figure illustrates how model results and observations are used to produce air quality profiles (concentration distributions) for the benefits analysis. The figure shows model runs at the top; four sets of "ratios" of model results in space in the middle; and frequency distributions of pollutant monitor concentrations and the space-dependent scaling of these by the ratios of the model predictions on the bottom.]

## Estimating the Effects of the CAAA on Ozone Air Quality

Future-year ozone concentrations corresponding to the Post-CAAA and Pre-CAAA scenarios were estimated through application of the UAM and UAM-V modeling systems. This section of the report contains an overview of the modeling systems and, for each geographical domain, a description of the application procedures and results. The calculation of ozone air quality profiles using the combined modeling results from the regional- and urban-scale modeling applications is also described.

For ease of reading, all figures follow the text in this section.

## Overview of The UAM and UAM-V Photochemical Modeling Systems

### *UAM*

The UAM is a three-dimensional photochemical grid model that calculates concentrations of pollutants by simulating the physical and chemical processes that occur in the atmosphere. It is formulated based on the atmospheric diffusion or species continuity equation. This equation represents a mass balance that includes all of the relevant emissions, transport, diffusion, chemical reaction, and removal processes in mathematical terms. The UAM incorporates the Carbon Bond IV chemical mechanism, which groups pollutant species to limit the number of chemical reactions, while permitting reasonable accuracy in simulating ozone and its precursors.

The major factors that affect photochemical air quality include:

- spatial distribution of emissions of volatile organic compounds (VOC) and $NO_x$, both natural and anthropogenic,
- composition of the emitted VOC and $NO_x$,
- spatial and temporal variations in the wind fields,
- dynamics of the boundary layer, including stability and the level of mixing,
- chemical reactions involving VOC, $NO_x$, and other important species,
- diurnal variations of solar insolation and temperature,
- loss of ozone and ozone precursors by dry and wet deposition, and
- ambient background concentration of VOC, NOx, and other species in, immediately upwind, and above the region of study.

The UAM simulates all of these processes. The species continuity equation is solved using the following fractional steps: emissions are injected; horizontal advection/diffusion is calculated; vertical advection/diffusion and deposition are calculated; and chemical transformations are performed for reactive pollutants. The UAM performs these four calculations during each time step. The maximum time step is a function of the grid size and the maximum wind velocity and diffusion coefficient. The typical time step is 10-15 minutes for coarse (10-20 km) grids and a few minutes for fine (1-2 km) grids.

Because it accounts for spatial and temporal variations as well as differences in the reactivity of emissions, the UAM is ideal for evaluating the air-quality effects of emission control scenarios. This is achieved by first replicating a historical ozone episode to establish a base-case simulation. Model inputs are prepared from observed meteorological, emissions, and air quality data for the episode days using prognostic meteorological modeling and/or diagnostic and interpolative modeling techniques. The model is then applied with these inputs, and the results are evaluated to determine model performance. Once the model results have been evaluated and determined to perform within prescribed levels, the same base-case meteorological inputs are combined with *modified* or *projected* emission inventories to simulate possible alternative/future emission scenarios.

The current UAM modeling system was released by the EPA in 1990 and is fully documented in the UAM user's guide (SAI, 1990). Features of the modeling system include a mixing-height-based vertical coordinate system and flux- and process-analysis capabilities to facilitate the comprehensive assessment of model performance and the interpretation of simulation results.

### *UAM-V*

The UAM-V modeling system represents an extension of the UAM. Like UAM, the UAM-V incorporates the Carbon Bond IV chemical mechanism. Other features of the UAM-V modeling system include:

- *Variable vertical grid structure:* The structure of vertical layers can be arbitrarily defined. This allows for higher resolution near the surface and facilitates matching with output from prognostic meteorological models.

- *Three-dimensional meteorological inputs:* The meteorological inputs for UAM-V vary spatially and temporally. These are usually calculated using a prognostic meteorological model.

- *Variable grid resolution for chemical kinetic calculations:* A chemical aggregation scheme can be employed, allowing chemistry calculations to be performed on a variable grid while advection/diffusion and emissions injections are performed on a fixed grid.

- *Two-way nested grid:* Finer grids can be imbedded in coarser grids for more detailed representation of advection/diffusion, chemistry, and emissions. Several levels of nesting can be accommodated.

- *Updated chemical mechanism:* The original Carbon Bond IV chemical mechanism has been updated to include the $XO_2/RO_2$ reaction, along with new temperature effects for PAN reactions. Aqueous-phase chemistry is also an option.

- *Dry deposition algorithm:* The dry deposition algorithm is similar to that used by the Regional Acid Deposition Model (RADM).

- *True mass balance:* Concentrations are advected and diffused in the model using units of mass per unit volume rather than parts per million. This maintains true mass balance in the advection and diffusion calculations.

- *Plume-in-grid treatment:* Emissions from point sources can be treated by a subgrid-scale Lagrangian photochemical plume model. Pollutant mass is released from the subgrid-scale model to the grid model when the plume size is commensurate with a grid cell size.

- *Plume rise algorithm:* The plume rise algorithm is based on the plume rise treatment for a Gaussian dispersion model.

## Regional-Scale Modeling of the Eastern U.S.

For this study, the UAM-V modeling system was applied separately for the eastern and western portions of the U.S. For the eastern U.S., the application was based, in part, on the regional-scale modeling analysis conducted by the Ozone Transport Assessment Group (OTAG). With the exception of the emission inventories, all inputs were those used for the OTAG modeling analysis. The application procedures and modeling results are summarized in this section.

### UAM-V Application Procedures for the Eastern U.S.

#### Modeling Domain

The modeling domain for application to the eastern U.S. is identical to that used for the OTAG modeling analysis. The domain encompasses the 37 eastern most states and the District of Columbia and consists of two grids. The horizontal resolution for the outer grid is approximately 36 km; this grid consists of five vertical layers. The horizontal resolution for the smaller inner grid is approximately 12 km; this grid consists of seven vertical layers. The top of the modeling domain is 4000 meters above ground level.

#### Simulation Periods

Two of the OTAG multi-day simulation periods were selected for use in this study. These are 20-30 July 1993 and 7-18 July 1995. Both simulation periods are characterized by high ozone concentrations in the eastern U.S.; numerous exceedances of the 1-hour National Ambient Air Quality Standard (NAAQS) for ozone were recorded. During the 1993 simulation

period, the exceedances occurred mostly within the southeastern U.S. During the 1995 period, high ozone concentrations were observed in several regions including the Lake Michigan area, the Northeast Corridor, and the Southeast. These periods were chosen to be representative of regional-scale ozone transport events for the southeastern and eastern U.S. respectively. In both cases, the extent of the high ozone concentrations is attributable to persistent, regional-scale ozone conducive meteorological conditions. The simulation periods include two and three initialization (or start-up) days, respectively. These are included to reduce the effects of uncertainties in the initial conditions on the simulation results.

## Input Preparation

The UAM-V modeling system requires a variety of input files that contain information pertaining to the modeling domain and simulation period. These include gridded, day-specific emissions estimates and meteorological fields; initial and boundary conditions; and land-use information.

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

## Modeling Emission Inventories

The UAM-V requires detailed emission inventories containing temporally allocated emissions for each grid cell in the modeling domain and for all primary pollutant species represented by the chemical mechanism. An extended version of EPA's UAM Emissions Preprocessor System, Version 2.0, or EPS 2.0 (SAI, 1992) called EPS 2.5e was used to process the inventories. In addition to the capabilities of EPS 2.0, this system has been enhanced to facilitate regional-scale model applications of particulate matter and toxic species, as well as ozone precursors.

Each inventory includes weekday/weekend area source emissions, typical summer day utility emissions, weekday/weekend non-utility point source emissions, and day-specific biogenic emissions. The on-road motor-vehicle emissions were based on typical summer weekday/weekend estimates.

Anthropogenic input emissions inventory data were provided by Pechan (1998). These included area and point source emissions data from the National Particulates Inventory (by county and for specific point sources), county-level vehicle miles traveled (VMT) estimates, and mobile-source emission factors for VOC, $NO_x$, and CO. Area source emissions include emissions from a variety of sources such as commercial and residential fuel combustion, non-point-source industrial emissions, solvent utilization, construction equipment, off-highway vehicles, gasoline distribution, furniture refinishing, and lawn mowers. Day-specific, model-ready biogenic emission inventories were obtained from the OTAG database. Preparation of the emission inventory data is described in detail by Pechan (1998). A brief description of the emissions processing is provided in this section.

Preliminary processing of the data prior to the application of the EPS 2.5e system was necessary. This consisted of generating the on-road mobile emissions and reformatting all data into Atmospheric Information Retrieval System (AIRS) Mobile-Source Subsystem (AMS) and Facility Subsystem (AFS) work-file formats. On-road mobile emissions were generated using the inputs provided by EPA and the MOBILE5a model. The outputs from MOBILE5a include future-year emissions of the ozone precursor pollutants VOC, $NO_x$, and CO. MOBILE5a accesses a matrix of emissions factors that are based on temperature, speed, and other site-specific parameters. Estimates of VMT were multiplied by emission factors to generate on-road motor vehicle emission estimates. The VMT estimates were provided at county level and were broken down into 12 different urban and rural roadway classifications.

All anthropogenic emission inputs to UAM-V were preprocessed through the EPS 2.5e emissions processing system. Photochemical grid models such as the UAM-V require detailed emission inventories, containing hourly emissions for each grid cell in the modeling domain for each species being simulated. The core EPS system is a series of FORTRAN modules that incorporate spatial, temporal, and chemical resolution into an emissions inventory used for photochemical modeling. Point, area, and mobile source emission data were processed separately to facilitate both data tracking for quality control and the use of the data in evaluating the effects of alternative control strategies on simulated air pollutant concentrations. The mobile source component was further broken down into rural and urban motor vehicle emissions based on the roadway classifications. The model-ready components (including biogenic) were then merged to generate the final model inputs.

The UAM-V requires hourly estimates of emissions for each grid cell to accurately simulate hourly concentrations of ozone. Accordingly, annual average or peak ozone season daily emission rates must be adjusted to reflect the conditions of the ozone episode being modeled, including seasonal adjustments for activity levels (if base year emissions are reported as annual averages), adjustments for the day of the week, and hourly temperature and activity adjustments for each hour of the episode day. EPA has developed a default set of temporal allocation factors (TAF) for each source category and these have been incorporated into EPS 2.5e. TAF were applied to all model inputs. For the eastern U.S. domain, the available typical peak ozone season day $NO_x$ and VOC emissions were adjusted for day of week and hourly allocation.

The Carbon-Bond IV chemical mechanism employed by the UAM-V modeling system, groups or "lumps" pollutants to limit the number of reactions and species to a reasonable level while permitting reasonable accuracy in predicting air quality. Ozone precursor hydrocarbon emissions were aggregated into the carbon-bond species required by the UAM-V using speciation profiles developed by the EPA (1991) and the default assignments provided with EPS. The chemical speciation scheme for VOCs includes eight categories: olefins, paraffins, toluene, xylene, formaldehyde, higher aldehydes, ethenes, and isoprene. For this study, the default $NO_X$ speciation of 90 percent NO and 10 percent $NO_2$ by weight, included in EPS 2.5e, was assumed for all point and area sources.

For the UAM-V model to accurately simulate observed air quality concentrations for the selected grid, it must be supplied with emissions data that have the same degree of spatial resolution (i.e., by grid cell). The effort required to implement this resolution varies depending on the type of source. For point sources, geographical coordinates for each source, typically reported to within a fraction of a kilometer, are used for direct assignment of emissions to the appropriate grid cells. By contrast, spatial resolution of emissions reported as county totals, as is usually the case for area sources and motor vehicles, requires substantially more effort. The most commonly employed approach for apportioning county-level emissions to grid cells is to use a surrogate indicator for spatial distribution of emission levels or activity (e.g., population, type of land use, or location of major links such as interstate roadways or airport runways). A spatial allocation surrogate is a quantity whose areal distribution is either known or has been estimated and is assumed to be similar to the areal distribution of emissions from some source category whose spatial distribution is not well known. County-level emissions are spatially allocated to the grid cells of the modeling domain. Surrogate data input used to create the spatial allocation factors included U.S. Geological Survey (USGS) land-use data, 1990 census data, and digitized county boundaries.

Emissions totals by component for VOC, $NO_x$, and CO for the base- and future-year scenarios are provided in Table C-1. This table shows increases in VOC and $NO_x$ under the Pre-CAAA scenarios and substantial decreases under the Post-CAAA scenarios. The decreases in VOC are primarily attributable to reductions in area- source and motor-vehicle emissions. The decreases in $NO_x$ are due to decreases in motor-vehicle and utility and non-utility point-source emissions.

## Air Quality, Meteorological, and Land-Use Inputs

The air quality, meteorological, and land-use inputs for application of the UAM-V modeling system for this study were identical to those used for the OTAG modeling exercise. The initial and boundary concentrations for the OTAG simulations were represented by "clean" air values for all species. The individual species concentrations vary slightly with elevation (or height above the ground) and with time of day, and are approximately 0.1 parts per billion (ppb) of $NO_x$, 5 parts per billion carbon (ppbC) of reactive hydrocarbons (RHC), and 100 ppb of CO. The boundary concentrations for ozone range from about 31 to 34 ppb. Further detail on the OTAG initial and boundary concentrations has been presented in OTAG publications (e.g., Deuel et. al., 1996).

Other input data required by the UAM-V model for simulating the ozone episodes (including the meteorological and land-use inputs) were obtained directly from the OTAG datasets without modification. Model options were the same in the current application as in the OTAG application, except that the plume-in-grid (P-i-G) treatment (a detailed treatment of the chemistry and geometry of plumes from elevated point sources) was not employed. This exception was made in order to reduce the demands on computer resources.

**Table C-1**
**Emission Totals by Component for each Scenario for the OTAG Domain (tpd)**

| | | | VOC | | |
|---|---|---|---|---|---|
| | **Base 1990** | **2000 Pre-CAAA** | **2000 Post-CAAA** | **2010 Pre-CAAA** | **2010 Post-CAAA** |
| Area | 33,417 | 38,517 | 27,982 | 43,113 | 8,638 |
| Onroad Mobile | 17,518 | 15,102 | 10,074 | 17,400 | 8,552 |
| Point | 8,247 | 9,027 | 7,317 | 10,194 | 8,204 |
| Utility | 87 | 81 | 82 | 111 | 113 |
| **Total** | **59,270** | **62,727** | **45,454** | **70,818** | **45,507** |
| | | | **NOx** | | |
| | **Base 1990** | **2000 Pre-CAAA** | **2000 Post-CAAA** | **2010 Pre-CAAA** | **2010 Post-CAAA** |
| Area | 12,109 | 13,858 | 13,351 | 15,770 | 13,741 |
| Onroad Mobile | 17,915 | 17,463 | 14,923 | 20,222 | 12,616 |
| Point | 6,647 | 7,345 | 4,444 | 8,365 | 4,681 |
| Utility | 17,637 | 20,668 | 8,254 | 22,670 | 5,182 |
| **Total** | **54,307** | **59,335** | **40,972** | **67,026** | **36,220** |
| | | | **CO** | | |
| | **Base 1990** | **2000 Pre-CAAA** | **2000 Post-CAAA** | **2010 Pre-CAAA** | **2010 Post-CAAA** |
| Area | 46,606 | 53,087 | 51,544 | 58,952 | 57,100 |
| Onroad Mobile | 147,842 | 112,656 | 84,569 | 124,385 | 78,396 |
| Point | 13,766 | 15,463 | 15,463 | 17,192 | 17,192 |
| Utility | 710 | 784 | 811 | 1,225 | 1,327 |
| **Total** | **208,924** | **181,990** | **152,388** | **201,753** | **154,015** |

## UAM-V Simulation Results for the Eastern U.S.

### Model Performance

The assessment of model performance is an important component of a modeling analysis and is used to ensure that the modeling system, including the inputs, is able to replicate the observed concentration levels associated with the historical modeling episode period. The evaluation of model performance is typically achieved through the comparison of simulated concentrations with observed data. In this case, the observed data correspond to the actual episode period and the emissions reflect emission levels for that same period/year. For the OTAG modeling component, model performance considered the base-case applications for 1993 and 1995.

Model performance for the OTAG episodes is documented in the OTAG modeling report (OTAG, 1997). In general, the observed ozone concentration levels were represented in the simulations, with some over- and under-estimation of the maximum values. Scatter plots comparing the simulated and observed concentrations for key modeling days for each episode (28 July 1993 and 15 July 1995) show generally good agreement between the simulated and observed values, with some tendency for over- and underestimation on all days, distributed among the concentration levels (scatter along the axis). These are typical of the comparisons for the other simulation days.

Since the simulation results corresponding to all concentration levels will be used to adjust the observed data for Section 812 modeling analysis a comparison of the mean values was also performed. Plots comparing the mean values for each simulation day of the 1993 and 1995 simulation periods in both cases show that the mean simulated values are slightly higher than the mean observed values, but the day-to-day tendencies are similar.

For the 1993 simulation period, the mean unsigned relative error (or normalized bias) ranges from approximately -15 percent to 1 percent. The corresponding values for the 1995 simulation period are -12 to 9 percent. These are all within the EPA recommended range (for urban-scale modeling) of ±15 percent. For both simulation periods, the mean relative error (or gross error) is less than 25 percent for each simulation day. The EPA recommended range is less than 35 percent.

The good agreement between the simulated and observed ozone concentrations, suggests that the OTAG modeling system (including the meteorological, air quality, and geographical inputs) provides an appropriate basis for the Section 812 prospective modeling.

### UAM-V Modeling Results

The UAM-V simulation results for the Pre- and Post-CAAA scenarios were used in this study to calculate factors for adjustment of observed data and estimation of future-year concentration levels. These were calculated by comparing the simulated concentrations corresponding to each future-year/scenario simulation with those for the base-year simulation (1990). Examples of this comparison are illustrated using isopleth plots for maximum ozone concentration in Figures C-2 and C-3.[1] These isopleth plots correspond to 1995 simulation period and depict differences in maximum ozone concentration for 15 July between the 1990 baseline and the 2010 Pre- and 2010 Post-CAAA scenarios, respectively. The differences are calculated as scenario minus base, so that negative values indicate lower concentrations for the future-year scenario. These plots indicate that for 2010 the Pre-CAAA simulation results are characterized by increases in ozone, while the Post-CAAA results show decreases in ozone. Similar results were found for both future years and for both episodes modeled (SAI, 1999). The increases occur over the mid- and southern sections of the domain while the decreases are more widespread. Both the

[1]For many of the figures in this appendix the Pre-CAAA scenario and Post-CAAA Scenario are referred to as Pre-CAAA90 and post CAAA90, respectively.

increases and decreases are larger and more widespread for 2010.

It is also useful to directly compare the Pre- and Post-CAAA simulation results for each future year. This gives a direct indication of the effects of the CAAA on the simulated ozone concentrations. For example, Figure C-4 illustrates the differences in maximum simulated ozone concentration between the Pre- and Post-CAAA simulations for 2010 for the 15 July 1995 simulated ozone episode. The differences are calculated as Post-CAAA minus Pre-CAAA, so that negative values indicate lower concentrations for the Post-CAAA scenario. In general the results of these comparisons indicate that except for isolated increases (single grid cells), the simulated daily maximum ozone concentrations for the Post-CAAA scenario are lower than the corresponding Pre-CAAA values for both future years. The spatial extent of the decreases is greater for 2010.

## *Regional-Scale Modeling of the Western U.S.*

Application of the UAM-V modeling system to the western U.S. utilized inputs from the regional-scale application of REMSAD (as described in the next section of this report). The objective of this application was to provide regional-scale ozone concentration estimates for those areas that are neither included in the OTAG domain nor in the urban-scale analyses. The application procedures and modeling results are summarized in this section.

### UAM-V Application Procedures for the Western U.S.

#### Modeling Domain

The modeling domain used to obtain results for the western U.S. is identical to that used for application of the REMSAD modeling system (as described in the following section of this report) for the PM-related analysis of the CAAA. The modeling domain encompasses the contiguous 48 states, extending from 126 degrees west longitude to 66 degrees west longitude, and from 24 degrees north latitude to 52 degrees north latitude. A grid cell size of 2/3 longitude by 1/2 latitude (approximately 56 by 56 km) results in a 90 by 55 grid (4,950 cells) for each vertical layer. Eight vertical layers were used. Note that although the domain includes the entire contiguous 48 states, results using this domain configuration were only used to estimate ozone concentrations for the western states.

#### Simulation Period

For the western U.S., the simulation period included 1-10 July 1990. As noted earlier, this simulation period was selected to accommodate use of the REMSAD inputs and, therefore, represents the summertime simulation period for PM modeling of the western U.S. This period is characterized by high ozone concentrations (in excess of the 1-hour ozone NAAQS) in the Los Angeles area on all days, and in the San Joaquin Valley on 9 and 10 July. Relatively high concentrations were also observed in the San Francisco Bay Area, the Sacramento Valley, the San Diego area, and Denver. Throughout the remainder of the domain, concentrations typically did not exceed 100 ppb. The simulation period includes three initialization (or start-up) day that were included to limit the influence of the initial conditions on the simulation results.

#### Input Preparation

Preparation of the model-ready emission inventories for this application utilized the same data and followed the same procedures outlined in the previous section of this report. Emissions totals for the base- and future-year scenarios are provided in Table C-2 for VOC, $NO_x$, VOC, and CO.

The meteorological, air quality, and land-use related inputs were identical to those used for the application of the REMSAD modeling system to the western U.S. The reader is referred to Section 4 of this report for a description of these inputs and input preparation procedures.

**Table C-2**
**Emission Totals by Component for each Scenario for the Entire U.S. (tpd).**

| | Base 1990 | 2000 Pre-CAAA | 2000 Post-CAAA | 2010 Pre-CAAA | 2010 Post-CAAA |
|---|---|---|---|---|---|
| **VOC** | | | | | |
| Area | 33,972 | 39,154 | 27,620 | 43,708 | 28,575 |
| Onroad Mobile | 18,659 | 16,454 | 10,683 | 18,776 | 8,804 |
| Point | 9,503 | 10,298 | 8,457 | 11,606 | 9,454 |
| Utility | 96 | 85 | 85 | 134 | 137 |
| **Total** | **62,229** | **65,991** | **46,845** | **74,224** | **46,970** |
| **NOx** | | | | | |
| | **Base 1990** | **2000 Pre-CAAA** | **2000 Post-CAAA** | **2010 Pre-CAAA** | **2010 Post-CAAA** |
| Area | 13,766 | 15,659 | 15,252 | 17,697 | 15,794 |
| Onroad Mobile | 20,399 | 20,660 | 17,421 | 24,142 | 14,696 |
| Point | 7,964 | 8,694 | 5,645 | 9,803 | 5,985 |
| Utility | 20,188 | 22,787 | 11,170 | 24,808 | 10,319 |
| **Total** | **62,316** | **67,800** | **49,487** | **76,450** | **46,793** |
| **CO** | | | | | |
| | **Base 1990** | **2000 Pre-CAAA** | **2000 Post-CAAA** | **2010 Pre-CAAA** | **2010 Post-CAAA** |
| Area | 70,069 | 80,679 | 79,155 | 90,198 | 88,240 |
| Onroad Mobile | 171,181 | 142,346 | 103,332 | 153,706 | 92,058 |
| Point | 16,478 | 18,257 | 18,257 | 20,210 | 20,210 |
| Utility | 861 | 796 | 804 | 1,243 | 1,269 |
| **Total** | **258,589** | **242,078** | **201,547** | **265,357** | **201,777** |

## UAM-V Simulation Results for the Western U.S.

### Model Performance

Model performance for ozone was assessed for the entire western region and for five subregions. Model performance was evaluated through graphical comparison of the simulated and observed regional and subregional maximum ozone concentration patterns and values. Quantitative measures of model performance were calculated on a subregional basis, although typical model performance criteria are not applicable for the grid resolution and domain scale used for this analysis. Overall, the results indicate that ozone concentrations in the western U.S. are somewhat underestimated, relative to the observed values. On a subregional basis, the results vary from day to day and can be characterized as follows:

- Southern California Coast: Gross concentration gradients are directionally represented in the simulation results, with lower values along the coast and higher ozone inland. However, the resolution is not sufficient to

resolve even the higher values within the Los Angeles Basin. The maximum simulated value on any day is 83 ppb, while the maximum observed value exceeds 100 ppb (at a number of the sites within the region) on any given day of the simulation period. Results corresponding to Los Angeles were not used in subsequent portions of the analysis.

- Northern California/Southern Oregon/Central and Western Nevada: Simulated ozone concentrations tend to be lower than observed in the Sacramento Valley and San Joaquin Valley as well as (toward the end of the simulation period) the eastern portion of the San Francisco Bay Area. Daily maximum simulated ozone concentrations in the San Joaquin Valley range from approximately 60 to 86 ppb. Observed values greater than or equal to 100 ppb were recorded during 7-10 July. Representation of the observed concentration pattern improves throughout the simulation period. Concentrations at monitors in Oregon and Nevada are generally well represented. Results corresponding to northern California were not used in the subsequent analysis.

- Pacific Northwest/Eastern Washington: Low observed ozone concentrations are slightly to moderately overestimated through 7 July and underestimated (in some cases just slightly) for 8-10 July. For days with ozone concentrations greater than 40 ppb, the normalized bias ranges from approximately -12 to 20 percent. The normalized gross error is less than about 38 percent.

- Four Corners States: Maximum ozone concentration in Phoenix, Las Vegas, and Salt Lake City are reasonably well represented in the simulation results. Concentrations for the Denver are consistently underestimated. Those for Albuquerque and El Paso are well represented for certain of the days and underestimated for others. There are also a few isolated sites for which maximum ozone is reasonably well simulated (the observed concentrations are low). The normalized bias ranges from approximately -5 to 14 percent. The normalized gross error is less than 40 percent. Results for Phoenix were not used in the subsequent analysis.

- Montana/Idaho/Wyoming/ Western Portion of Dakotas: Day-to-day differences in concentrations are not well represented, however, the simulated values are generally consistent with the limited observations. The normalized bias ranges from zero to approximately 30 percent. The normalized error is greater than 35 percent for four of the simulation days.

- Note on the Eastern U.S.: Maximum simulated ozone concentrations range from approximately 160 to 250 ppb during the simulation period. Simulated peaks occur over Baton Rouge, Houston, St. Louis, and Atlanta with some high values along the NE corridor. These values have not yet been compared with observations, but simulated ozone concentrations are much higher in the east than in the west.

In general, the coarse resolution limits the ability of the modeling system to resolve peak concentrations and, in some cases, concentration gradients (such as those that occur along the coast of California). Based on these results, it was decided that the western ozone modeling results could be used to characterize the regional-scale concentration changes but would be supplemented with higher resolution modeling for Los Angeles, the San Francisco Bay Area (and portions of northern California), and Phoenix.

Simulated and observed concentrations for two of the modeling days (4 and 8 July 1990) were compared by SAI (1999). The differences between the simulated and observed values are typically larger than those for the OTAG simulations (possibly due to the coarser grid resolution) and represent both under- and overestimation of the maximum observed

concentrations. Underestimation of the higher concentrations is prevalent for nearly all of the simulation days.

In addition, a comparison of mean simulated and observed values by SAI showed that, while the highest values are underestimated, the mean simulated values are slightly greater than the observed means (SAI, 1999).

The model performance evaluation for the western ozone modeling application suggests that the modeling results can be used for the regional-scale analysis. Although the peak concentrations tend to be underestimated, there is not a uniform bias in the representation of the daily maxima. In addition, the mean values are fairly well characterized.

### UAM-V Modeling Results

The UAM-V simulation results corresponding to the Pre- and Post-CAAA scenarios for 2010 are compared to the base-year values in Figures C-5 and C-6, respectively. The isopleth plots depict the differences in maximum ozone concentration for 8 July between the base (1990) simulation and the 2010 Pre- and Post-CAAA simulations, respectively. The differences are calculated as scenario minus base, so that negative values indicate lower concentrations for the future-year scenario. Similar results were found for both future years modeled (SAI, 1999) and indicate increases in daily maximum ozone for large portions of the western U.S. with smaller areas of decrease (e.g., over California) for the Pre-CAAA scenario. For the Post-CAAA scenarios, the plots indicate widespread decreases with small areas of increase.

A comparison of the Pre- and Post-CAAA simulation results for 2010 is provided in Figure C-7. The differences are calculated as Post-CAAA minus Pre-CAAA, so that negative values indicate lower concentrations for the Post-CAAA scenario. This comparison indicates lower ozone concentrations for the Post-CAAA scenario compared to the Pre-CAAA scenario over most of the western U.S., with some increases in the San Francisco Bay Area, Los Angeles, and Seattle. The simulation results suggest that $NO_x$ reductions within these areas are disbeneficial with respect to ozone air quality. This is likely attributable to the reduced ozone titration that occurs in the simulation when $NO_x$ emissions are reduced. This phenomenon is most frequently apparent in area where $NO_x$ emissions are large relative to VOC emissions (VOC-limited areas).

## *Urban-Scale Modeling of the San Francisco Bay Area*

High-resolution, urban-scale modeling of the San Francisco Bay Area (northern California) was intended to provide an improved basis (compared to the regional-scale application of UAM-V for the western U.S.) for the estimation of future-year ozone profiles for the Bay Area and portions of northern California. With the exception of the emission inventories, all inputs for this application were obtained from the Bay Area Air Quality Management District (BAAQMD), and used by permission. The application procedures and modeling results are summarized in this section.

### UAM-V Application Procedures for the San Francisco Bay Area

### Modeling Domain

The modeling domain for this application of the UAM-V modeling system includes the San Francisco Bay Area, the Monterrey Bay Area, Sacramento, and a portion of the San Joaquin Valley. The location and geographical extent of the domain is illustrated in Figure C-8. The domain consists of 102 by 102 horizontal grid cells with a grid spacing of 4 km. It also includes 16 vertical layers.

### Simulation Period

The simulation period for the application to northern California is 3-6 August 1990. This episode period occurred during the San Joaquin Valley Air Quality Study and was characterized by moderate to

high ozone concentrations in the San Francisco Bay Area on 5 and 6 August, and in the Sacramento area and the San Joaquin Valley on 4, 5, and 6 August. The observed peak in the Bay Area was 120 ppb, while that for the other two more inland areas reached 150 ppb. The simulation period includes one initialization (or start-up) day that was included to limit the influence of the initial conditions on the simulation results.

## Input Preparation

Preparation of the model-ready emission inventories for this application utilized the same data and followed the same procedures outlined in the previous section of this report. Emissions totals for the base- and future-year scenarios are provided in Table C-3 for VOC, $NO_x$, and CO. This table indicates a downward trend in emissions (between 1990 and 2000) followed by an upward trend (between 2000 and 2010) for the Pre-CAAA scenario. The increases are attributable to area- source and motor-vehicle emissions (i.e., increases in population and vehicle miles traveled). Emissions for both future years are lower than the base-year for the Post-CAAA scenario. The decreases are primarily due to a reduction in motor-vehicle emissions.

The meteorological, air quality, and land-use related inputs were prepared by the BAAQMD for use in their SIP modeling analysis. Documentation of the input preparation procedures and resulting inputs is available on-line (BAAQMD, 1998). Initial and boundary conditions for the future-year applications were estimated based on the corresponding emission reductions for VOC and $NO_x$; for ozone the square root of the product of the VOC and $NO_x$ reduction factors was used.

**Table C-3**
**Emission Totals by Component for each Scenario for the San Francisco Bay Area (tpd)**

| | Base 1990 | 2000 Pre-CAAA | 2000 Post-CAAA | 2010 Pre-CAAA | 2010 Post-CAAA |
|---|---|---|---|---|---|
| **VOC** | | | | | |
| Area | 783 | 795 | 644 | 886 | 698 |
| Onroad Mobile | 900 | 572 | 223 | 680 | 97 |
| Point | 111 | 110 | 110 | 110 | 93 |
| Utility | 1 | 0 | 0 | 1 | 1 |
| **Total** | **1,795** | **1,477** | **977** | **1,677** | **889** |
| **NOx** | | | | | |
| | Base 1990 | 2000 Pre-CAAA | 2000 Post-CAAA | 2010 Pre-CAAA | 2010 Post-CAAA |
| Area | 381 | 407 | 392 | 458 | 396 |
| Onroad Mobile | 850 | 827 | 545 | 1,014 | 337 |
| Point | 202 | 197 | 140 | 197 | 140 |
| Utility | 46 | 4 | 4 | 2 | 2 |
| **Total** | **1,479** | **1,435** | **1,081** | **1,671** | **874** |
| **CO** | | | | | |
| | Base 1990 | 2000 Pre-CAAA | 2000 Post-CAAA | 2010 Pre-CAAA | 2010 Post-CAAA |
| Area | 1,846 | 2,113 | 2,098 | 2,411 | 2,394 |
| Onroad Mobile | 7,414 | 5,652 | 2,526 | 6,630 | 1,444 |
| Point | 123 | 119 | 119 | 119 | 119 |
| Utility | 46 | 9 | 10 | 26 | 26 |
| **Total** | **9,429** | **7,893** | **4,753** | **9,186** | **3,983** |

## UAM-V Simulation Results for the San Francisco Bay Area

### Model Performance

Model performance was evaluated by the BAAQMD as part of their SIP modeling analysis and the inputs (with the exception of the modeling emission inventories) were used directly for the 812 prospective modeling analysis. Sscatter plots comparing the maximum simulated and observed ozone concentrations for both simulation periods are available in (SAI, 1999). These comparisons indicate good agreement between the simulated and observed values with a tendency for underestimation of the high observed ozone concentrations. Mean values are underestimated by about 10 to 15 percent on all days, which is within the current EPA range for acceptable urban-scale model performance (SAI, 1999).

Since good model performance is achieved, results of the model performance evaluation for ozone suggest that the UAM-V modeling platform for northern California (including the meteorological, air quality, and geographical inputs) provides an appropriate basis for the Section 812 prospective modeling.

### UAM-V Modeling Results

Comparison of the UAM-V simulation results for the Pre- and Post-CAAA scenarios with the base-year values indicates both increases and decreases in the simulated concentrations for the Bay Area , both of which are greater in magnitude and more widespread for the Post-CAAA scenario and for 2010. Isopleth plots for the Bay Area are available in (SAI, 1999).

A comparison of the Pre- and Post-CAAA simulation results for 2010 is provided in Figure C-9. The differences are calculated as Post-CAAA minus Pre-CAAA, so that negative values indicate lower concentrations for the Post-CAAA scenario. This comparison indicates that the CAAA results in higher daily maximum ozone in the Bay Area but lower ozone throughout the remainder of the domain. Similar results were obtained for 2000 (SAI, 1999). These results are qualitatively consistent with the regional-scale modeling results presented in the previous section of this report. However, the extent of the increases is more limited and the decreases are greater in the refined modeling. Note the increases occur in areas where the base-year ozone concentrations are low to very low.

## *Urban-Scale Modeling of the Los Angeles Area*

High-resolution, urban-scale modeling of the Los Angeles area was intended to provide an improved basis (compared to the regional-scale application of UAM-V for the western U.S.) for the estimation of future-year ozone profiles for this area. With the exception of the emission inventories, all inputs for this application were obtained from the South Coast Air Quality Management District (SCAQMD), and used by permission. As noted earlier, modeling of this area was performed using the UAM modeling system. The model formulation is similar to that for the UAM-V modeling system, but lacks certain features that make UAM-V suitable for regional-scale applications. The application procedures and modeling results are summarized in this section.

## UAM Application Procedures for the Los Angeles Area

### Modeling Domain

Application of the UAM-IV for the Los Angeles area was based on modeling performed by SCAQMD, as reported in the 1994 Air Quality Management Plan (SCAQMD, 1994). The modeling domain for this application is a 65 by 40 array of 5 km resolution grid cells. The domain also contains 5 vertical layers. The domain encompasses the South Coast Air Basin (SoCAB) (from Los Angeles to beyond Riverside) and a portion of the Mojave Desert. The location and geographical extent of the domain is illustrated in Figure C-8.

### Simulation Period

Two simulation periods were included in the modeling analysis for Los Angeles: 23-25 June 1987 and 26-28 August 1987. Both of these episodes occurred during the 1987 Southern California Air Quality Study (SCAQS). In both cases, the simulation period includes one initialization, or start-up, day (in order to reduce the influence of the somewhat uncertain initial concentrations on model results).

### Input Preparation

Preparation of the model-ready emission inventories for this application utilized the same data and followed the same procedures outlined in a previous section of this report. Emissions totals for the base- and future-year scenarios are provided in Table C-4 for VOC, $NO_x$, and CO. The Post-CAAA scenarios are characterized by lower emissions than the base year and the Pre-CAAA scenarios. The differences are largely attributable to changes in the motor-vehicle emissions.

The meteorological, air quality, and land-use related inputs were prepared by the SCAQMD for use in their SIP modeling analysis. The reader is referred to SCAQMD (1994 and 1996) for detailed information on the input preparation procedures and resulting inputs. Initial and boundary conditions for

the future-year applications were estimated based on the corresponding emission reductions for VOC and $NO_x$; for ozone the square root of the product of the VOC and $NO_x$ reduction factors was used.

**Table C-4**
**Emission Totals by Component for each Scenario for Los Angeles (tpd)**

| | Base 1990 | 2000 Pre-CAAA | 2000 Post-CAAA | 2010 Pre-CAAA | 2010 Post-CAAA |
|---|---|---|---|---|---|
| **VOC** | | | | | |
| Area | 758 | 770 | 607 | 871 | 700 |
| Onroad Mobile | 1,179 | 999 | 410 | 1,168 | 213 |
| Point | | | | | |
| Low Level | 197 | 196 | 196 | 196 | 158 |
| Elevated | 1 | 3 | 3 | 2 | 2 |
| **Total** | **2,135** | **1,968** | **1,216** | **2,237** | **1,073** |
| **NOx** | | | | | |
| | Base 1990 | 2000 Pre-CAAA | 2000 Post-CAAA | 2010 Pre-CAAA | 2010 Post-CAAA |
| Area | 450 | 467 | 453 | 529 | 463 |
| Onroad Mobile | 993 | 1,280 | 879 | 1,573 | 626 |
| Point | | | | | |
| Low Level | 216 | 186 | 139 | 186 | 139 |
| Elevated | 19 | 19 | 18 | 12 | 8 |
| **Total** | **1,678** | **1,953** | **1,489** | **2,300** | **1,236** |
| **CO** | | | | | |
| | Base 1990 | 2000 Pre-CAAA | 2000 Post-CAAA | 2010 Pre-CAAA | 2010 Post-CAAA |
| Area | 1,142 | 1,302 | 1,286 | 1,515 | 1,495 |
| Onroad Mobile | 9,046 | 10,043 | 5,046 | 11,278 | 3,728 |
| Point | | | | | |
| Low Level | 208 | 197 | 197 | 197 | 197 |
| Elevated | 2 | 43 | 44 | 34 | 35 |
| **Total** | **10,398** | **11,586** | **6,573** | **13,024** | **5,456** |

## UAM Simulation Results for the Los Angeles Area

### Model Performance

Model performance was evaluated by SCAQMD as part of their SIP modeling analysis and the inputs (with the exception of the modeling emission inventories) were used directly for the 812 prospective modeling analysis. Comparisons of maximum simulated and observed concentrations for each of the simulation periods are available in (SAI, 1999). They indicate a tendency for underestimation of the high observed ozone concentrations. This underestimation

also shows up in the comparison of the mean values (SAI, 1999).

While the urban-scale results are much better than those obtained with the coarser-resolution grid, both the maximum and mean values are underestimated. For the primary episode days, the normalized bias exceeds the EPA recommended range, while the normalized gross error is within approximately 35 percent. While this does not preclude the use of these results for the 812 study, it should be noted that the simulated changes in ozone between the base- and future-year scenarios may be influenced by the lack of good model performance. Use of the simulation results in the relative sense (through the calculation of adjustment factors) should reduce the uncertainty, compared to use of the absolute values.

### UAM Modeling Results

Comparison of the UAM-V simulation results for the Pre- and Post-CAAA scenarios with the base-year values shows large reductions in daily maximum ozone for all four future-year scenarios. Some increases in ozone are simulated for 2010 for the Pre-CAAA scenario; overall, the extent and magnitude of the reductions is greater for the Post-CAAA scenario for both years. Isopleth plots for the Los Angeles area are available in (SAI, 1999).

A comparison of the Pre- and Post-CAAA simulation results for 2010 is provided in Figure C-10. The differences are calculated as Post-CAAA minus Pre-CAAA, so that negative values indicate lower concentrations for the Post-CAAA scenario. This comparison indicates lower maximum ozone concentrations under the Post-CAAA scenario for both years. Small increases occur over the urban area; these are smaller in extent and magnitude than for the regional modeling application. Similar results were found for 2000 (SAI, 1999).

## *Urban-Scale Modeling of the Maricopa County (Phoenix) Area*

High-resolution, urban-scale modeling of Maricopa County, Arizona (which includes the Phoenix urban area) was intended to provide an improved basis (compared to the regional-scale application of UAM-V for the western U.S.) for the estimation of future-year ozone profiles for this area. With the exception of the emission inventories, all inputs for this application were obtained from the Maricopa Association of Governments (MAG), and used by permission. As noted earlier, modeling of this area was performed using the UAM modeling system.

### UAM Application Procedures for the Phoenix Area

### Modeling Domain

The modeling domain for the application of the UAM modeling system to the Phoenix area encompasses the urbanized portion of Maricopa County, Arizona; this domain was based on that used for a previous application of UAM for the area (Douglas et al., 1994). The domain consists of a 44 by 33 array of 2 km grid cells and 5 vertical layers. The location and geographical extent of the domain is illustrated in Figure C-8.

### Simulation Period

Two ozone episodes were also simulated for the Phoenix area: 9-10 August 1992 and 13-14 June 1993. Exceedances of the 1-hour NAAQS for ozone were recorded during both episodes. Each period also includes one initialization day.

### Input Preparation

Preparation of the model-ready emission inventories for this application utilized the same data and followed the same procedures outlined in a previous section of this report. Emissions totals for the base- and future-year scenarios are provided in

Table C-5 for VOC, $NO_x$, and CO. The changes in emissions are characterized by both increases and decreases, reflecting an expected growth in population that is offset by fleet turnover and other emission reduction measures.

The meteorological, air quality, and land-use related inputs were prepared by Douglas et al. (1994). The reader is referred to this technical report for detailed information on the input preparation procedures and resulting inputs. Initial and boundary conditions for the future-year applications were estimated based on the corresponding emission reductions for VOC and $NO_x$; for ozone the square root of the product of the VOC and $NO_x$ reduction factors was used.

## UAM Simulation Results for the Phoenix Area

### Model Performance

Model performance was evaluated by Maricopa Association of Governments (MAG) as part of their SIP modeling analysis and the inputs (with the exception of the modeling emission inventories) were used directly for the 812 prospective modeling analysis. Comparison of hourly simulated and observed ozone concentrations indicates good agreement between the simulated and observed values. Mean values are well represented as well. Plots of these comparisons are available in (SAI, 1999). For the primary modeling days, the normalized bias and error statistics indicate very good model performance. The values are less than 5 percent (bias) and 20 percent (error) respectively (SAI, 1999).

The model performance results indicate that the UAM modeling system (including the meteorological, air quality, and land-use input) are appropriate for use in the Section 812 prospective analysis.

### UAM Modeling Results

Comparison of the UAM-V simulation results for the Pre- and Post-CAAA scenarios with the base-year values indicates both increases and decreases for the Pre-CAAA scenario simulations and large decreases for the Post-CAAA scenario simulations. Isopleth plots for the Phoenix area are available in (SAI, 1999).

A comparison of the Pre- and Post-CAAA simulation results for each future year indicates that the CAAA measures reduce daily maximum ozone concentrations within the Phoenix domain by approximately 10 to 20 ppb (more or less in some areas) for both future years. Isopleth plots for the Phoenix area are available in (SAI, 1999).

**Table C-5**
**Emission Totals by Component for each Scenario for Phoenix (tpd)**

| **VOC** | | | | | |
|---|---|---|---|---|---|
| | Base 1990 | 2000 Pre-CAAA | 2000 Post-CAAA | 2010 Pre-CAAA | 2010 Post-CAAA |
| Area | 241 | 310 | 201 | 369 | 250 |
| Onroad Mobile | 184 | 215 | 141 | 231 | 85 |
| Point | | | | | |
| Low Level | 2 | 2 | 1 | 2 | 2 |
| Elevated | 0 | 0 | 0 | 0 | 0 |
| **Total** | **426** | **527** | **344** | **602** | **337** |
| **NOx** | | | | | |
| | Base 1990 | 2000 Pre-CAAA | 2000 Post-CAAA | 2010 Pre-CAAA | 2010 Post-CAAA |
| Area | 213 | 270 | 259 | 326 | 290 |
| Onroad Mobile | 151 | 214 | 174 | 264 | 147 |
| Point | | | | | |
| Low Level | 1 | 1 | 1 | 2 | 2 |
| Elevated | 0 | 0 | 0 | 0 | 0 |
| **Total** | **371** | **485** | **434** | **592** | **438** |
| **CO** | | | | | |
| | Base 1990 | 2000 Pre-CAAA | 2000 Post-CAAA | 2010 Pre-CAAA | 2010 Post-CAAA |
| Area | 684 | 820 | 795 | 941 | 907 |
| Onroad Mobile | 1,186 | 2,001 | 1,538 | 1,883 | 1,002 |
| Point | | | | | |
| Low Level | 0 | 0 | 0 | 0 | 0 |
| Elevated | 1 | 1 | 1 | 1 | 1 |
| **Total** | **1,871** | **2,822** | **2,334** | **2,825** | **1,910** |

## Calculation of Ozone Air Quality Profiles

The overall objective of the photochemical modeling exercise was to provide estimates of future-year ozone air quality for 2000 and 2010 (for assessment of the effects of the CAAA). This was accomplished using an approach that combines observed data and air quality modeling results to estimate future-year concentrations. The methodology is designed to provide site-specific, seasonal and annual ozone concentration distributions. The statistical concentration distributions are estimated (based on the results of air quality modeling) for specific future-year scenarios and, in turn, provide the basis for examination and quantification of the effects of changes in air quality on health, agriculture, etc. (i.e., physical effects and economic valuation modeling). Through comparison with corresponding results for a baseline simulation (in this case without the CAAA measures and programs), the effects of the CAAA can be assessed. The future-year air quality profile estimation methodology, as applied to the analysis of the CAAA, is described in this section.

### *Overview of the Methodology*

Conceptually, the methodology for estimating future-year ozone air quality using both observations and UAM-V simulation results is rather simple. The UAM-V simulation results are used to calculate adjustment factors for selected ozone monitoring sites within the modeling domain. This is done on a grid-cell by grid-cell basis (i.e., the adjustment factor for a monitoring site is based on the simulation ozone concentrations for the grid cell in which it is located). The adjustment factor represents the ratio of the future-year-scenario to the base-year concentrations and is calculated (using the appropriately matched values) for several different concentration levels (i.e., the changes in concentration are dependent upon relative concentration level). The observed ozone concentrations for each monitoring site are then modified using the site-specific (or grid-cell-specific) adjustment factors. The resulting values represent the estimated future-year ozone concentrations for the modeled scenario.

As noted earlier in this report, the overall approach to estimate future air quality differs from that for a typical air quality model application (e.g., for attainment demonstration purposes) in that the modeling results are used in a relative sense, rather than an absolute sense. This may enhance the reliability of the future-year concentration estimates, especially in the event that the uncertainty inherent in the absolute concentration values is greater than that associated with the response of the modeling system to changes in emissions. This approach also permits the estimation of seasonal and annual concentration distributions, a requirement for this study.

Although the ratios are calculated using modeling results for a limited number of simulation days, it is assumed, using this methodology, that the ratios can be used to represent longer time periods. Consequently, all observations contained within the dataset are adjusted using the model-derived ratios. Following adjustment of the observed data, statistical quantities, or "profiles", describing the ozone distribution for each monitoring site are then calculated.

### *Description of the Observation Dataset*

One of the unique aspects of this approach to evaluating future ozone air quality is the use of observed ozone concentrations to supplement model results. As such, one of the earliest tasks was the creation of a dataset containing the observed hourly ozone concentrations for all monitoring sites located within the modeling domains for the months of May through September 1990.

Hourly ozone concentrations for 1990 were extracted from the Aerometric Information Retrieval System (AIRS) and input into a single AMP350-format datafile. From the information contained in this file, two SAS datasets were created: a concentration dataset and a monitor information dataset). The concentration dataset contains the

hourly concentrations for each monitor, with each record in the dataset representing a single monitor-day. The monitor information dataset contains monitor-specific information such as land-use and location.

In creating the concentration dataset, some data handling issues arose and were addressed in the following manner:

- In some instances, multiple ozone monitoring devices were operated at the same location. Even though these different devices have the same AIRS state-county-site identification code (ID), they are differentiated by a parameter occurrence code (POC). The AIRS state-county-site ID was concatenated with the POC to form a unique identifier for each monitor. A POC greater than 5 typically indicates that a device was being calibrated; information/data for these monitors was/were not included in either the monitor or the concentration dataset.

- In the AIRS database, ozone concentrations are reported using the default unit of the reporting agency. Thus, multiple units were present in the AMP350 file. For ease of analysis, all of the concentrations were converted to a single unit, ppm.

- Missing ozone concentrations in the AIRS AMP350 report are indicated by a blank in the decimal field. In the concentration dataset for this study, the SAS missing value code was used to indicate missing data.

- For each monitor a method detection limit (MDL) was provided. The MDL indicates a threshold below which reported ozone concentrations do not accurately reflect the sample distribution. For most monitors the MDL is 0.005 ppm. Because this value is low relative to typical ambient concentration levels, observed values below the MDL were not reset to the MDL and instead were left unchanged.

Only monitors with "complete" data were used in the analysis. For the ozone data, a monitor record was considered to be complete if data were available for 50 percent of the days in the peak ozone season (May-September). Each of these days in turn had to have at least 12 hourly observations. There were 842 ozone monitors with complete data.

## Calculation of Percentile-Based Adjustment Factors

For each future-year modeling scenario, grid-cell-specific adjustment factors were calculated using the hourly simulated ozone concentrations contained in the UAM-V or UAM *xymap*[2] output files. Individual monitoring sites were mapped onto the gridded model output (to determine the grid cell in which each monitor was located) and the concentrations for the corresponding grid cells were used to calculate a set of ten adjustment factors for each future-year modeling scenario. The adjustment factors were specified to be the ratio of the percentile concentrations for the future- and base-year simulations, where the percentile concentrations were calculated using data for each hour of each simulation day:

$$Adjustment\ Factor_i = \frac{x_i th\ Percentile\ Concentration_{future\ year}}{x_i th\ Percentile\ Concentration_{base\ year}}$$

$$\{x_i\} = \{5, 15, 25, 35, 45, 55, 65, 75, 85, 95\}$$

For calculation of the percentile concentrations, the empirical distribution function with averaging was employed. Because the concentrations for the lower percentiles can be rather small, a threshold value of 1 ppb was set to keep the adjustment factors reasonable. In other words, all concentrations below 1 ppb were reset to 1 ppb. This percentile-based approach was selected due to the limitations of using a single adjustment to represent the change in the modeled ozone concentrations in moving from the base- to the future-year scenarios. Finally, if either the base-year

[2] The UAM-V *xymap* file contains hourly, gridded, surface-layer ozone concentrations.

or future year percentile concentration was set equal to 1 ppb, the adjustment factor was set equal to 1.

A SAS dataset containing the monitor-level adjustment factors was created for each future-year modeling scenario considered in this study.

## Use of Adjustment Factors to Modify Observed Concentrations

Using the calculated adjustment factors for each future-year scenario and the observed monitor- and pseudo-site-level observations, a dataset containing modified observed hourly ozone concentrations for each of the two scenarios was created. Because each monitor has ten adjustment factors per scenario, it was first necessary to rank order the observed concentrations into 10 decile-based groups with ties being assigned to the higher group. Once each of the observed concentrations was identified with a particular decile group, the appropriate adjustment factor was selected and applied:

$$AdjustedConc_i = ObsConc_i * Adj.Factor_{k[ObsConci]}$$

In this equation, {ObsConci} is the set of observed hourly ozone concentrations (in ppm) for a given monitor or pseudo-site. The $k_{[ObsConc_i]}$ is the number of the decile group to which $ObsConc_i$ belongs. $Adj.Factor_{k[ObsConci]}$ is then the appropriate adjustment factor for the decile group to which $ObsConc_i$ belongs. The resulting set of adjusted hourly concentrations, $\{AdjustedConc_i\}$, represents the future-year estimates of the hourly ozone concentrations.

## Calculation of Ozone Profiles

Datasets containing the ozone air quality "profiles" were compiled for the base 1990, 2000 Pre-CAAA, 2000 Post-CAAA, 2010 Pre-CAAA, and 2010 Post-CAAA simulations. The profiles used data for the period May through September. The databases contained the number, the arithmetic mean, the median, the (seasonal) second highest, and the 2.5 to 97.5 percentiles (in increments of five) of the hourly concentrations. The profiles are reported at the monitor level and include 842 locations.

The histograms in Figures C-11a through 12b illustrate the distribution of ratios for the 95$^{th}$ percentile monitor-level ozone concentrations corresponding to the 2000 and 2010 simulations, respectively. In this figure, ratios greater than one indicate that the future-year/scenario concentration is greater than the base-year (1990) value, whereas ratios less than one indicate a lower value for the future-year.

The 2000 Pre-CAAA ratios (Figure C-11a) indicate that the 95$^{th}$ percentile ozone concentrations corresponding to this scenario are higher in some areas and lower in other areas than the base-year (1990) values. The ratios generally range from approximately 0.8 to 1.2, but also include some lower values. In contrast, the ratios corresponding to the 2000 Post-CAAA simulation (Figure C-11b) are generally less than one. In this case, the ratios range from approximately 0.75 to 1.1 with only a very small number of values greater than one. There are also some lower values.

Figure C-12a and 12b displays the distribution of ratios of the future-year-scenario to base-year 95$^{th}$ percentile concentrations for 2010. Compared to the histogram plots for 2000, the shift in distribution is such that the ratios are higher for the Pre-CAAA scenario and lower for the Post-CAAA scenario. That is, compared to 2000, concentrations for 2010 are higher relative to the base year under the Pre-CAAA scenario and lower relative to the base year under the Post-CAAA scenario.

For both future years, the ratios indicate that the Post-CAAA concentrations (95$^{th}$ percentile level) are lower than the corresponding Pre-CAAA values (with a few exceptions). This is illustrated in Figures C-13a and C- 13b. The smaller ratios for 2010 (Figure C-13b) reflect the larger differences between the Pre- and Post-CAAA scenarios for this year.

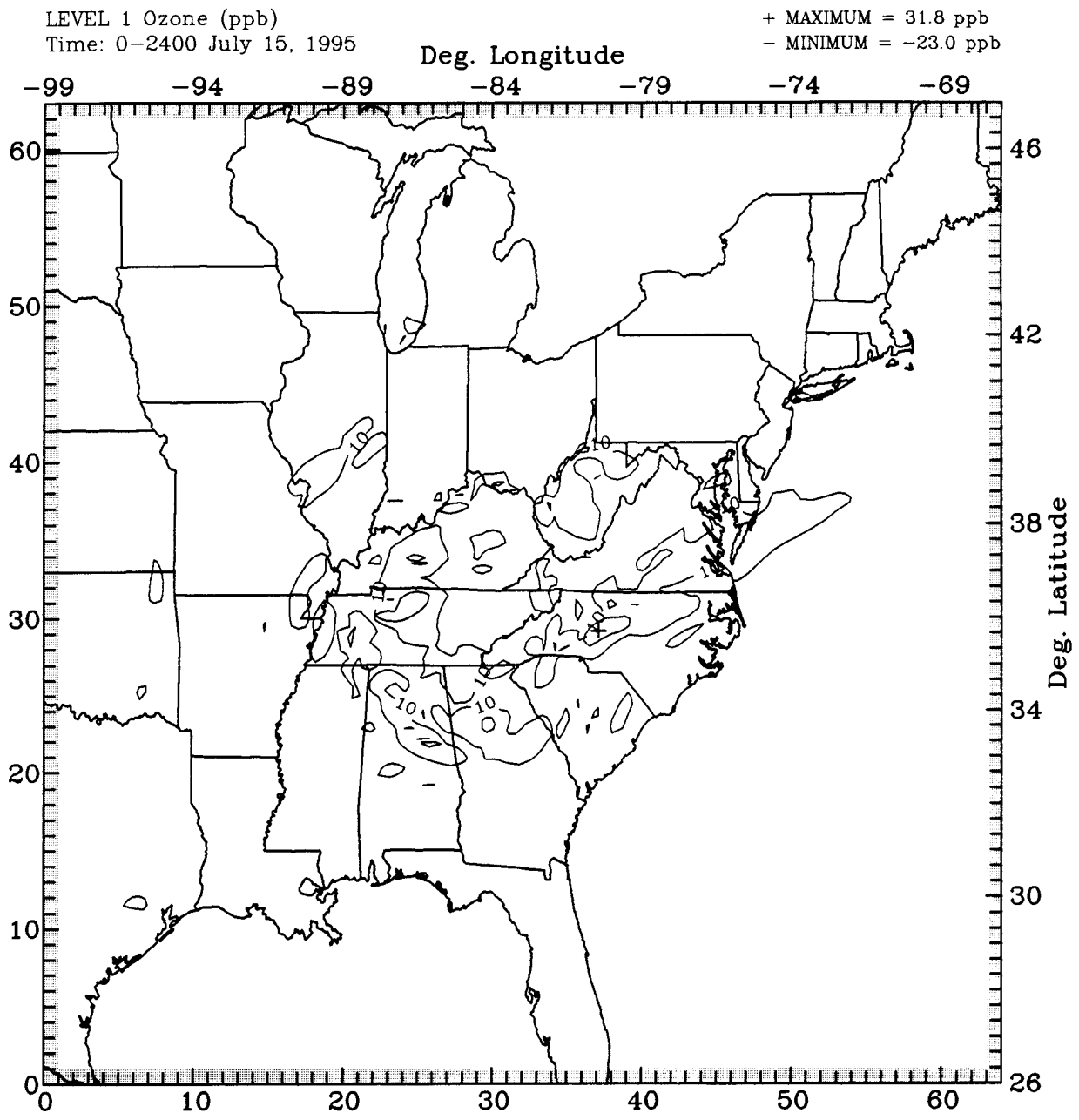



Figure C-2: Difference in daily maximum simulated ozone concentration (ppb) for the 15 July 1995 OTAG episode day: 2010 pre-CAAA90 minus base 1990.

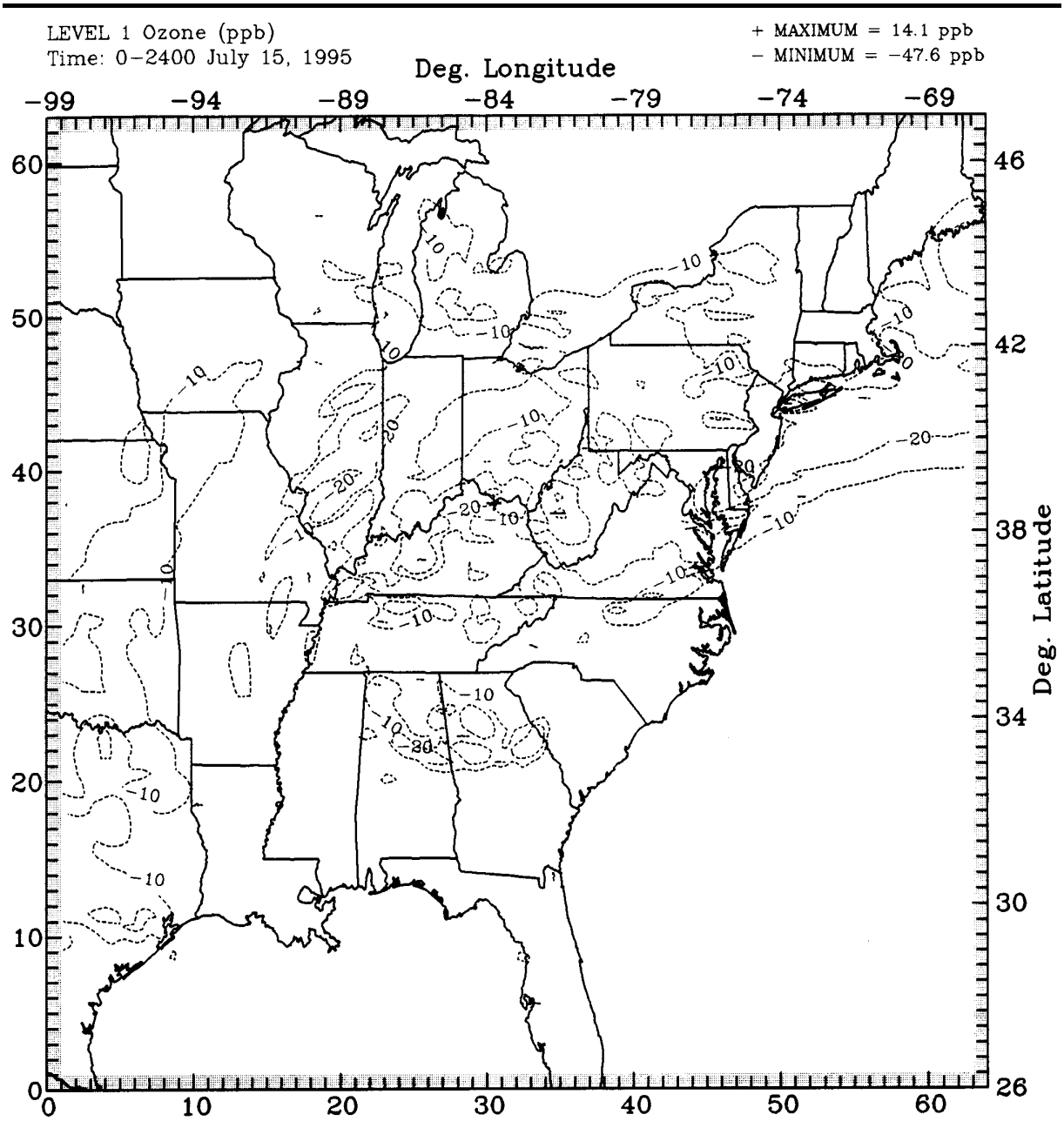



Figure C-3. Difference in daily maximum simulated ozone concentration (ppb) for the 15 July 1995 OTAG episode day: 2010 post-CAAA90 minus base 1990.

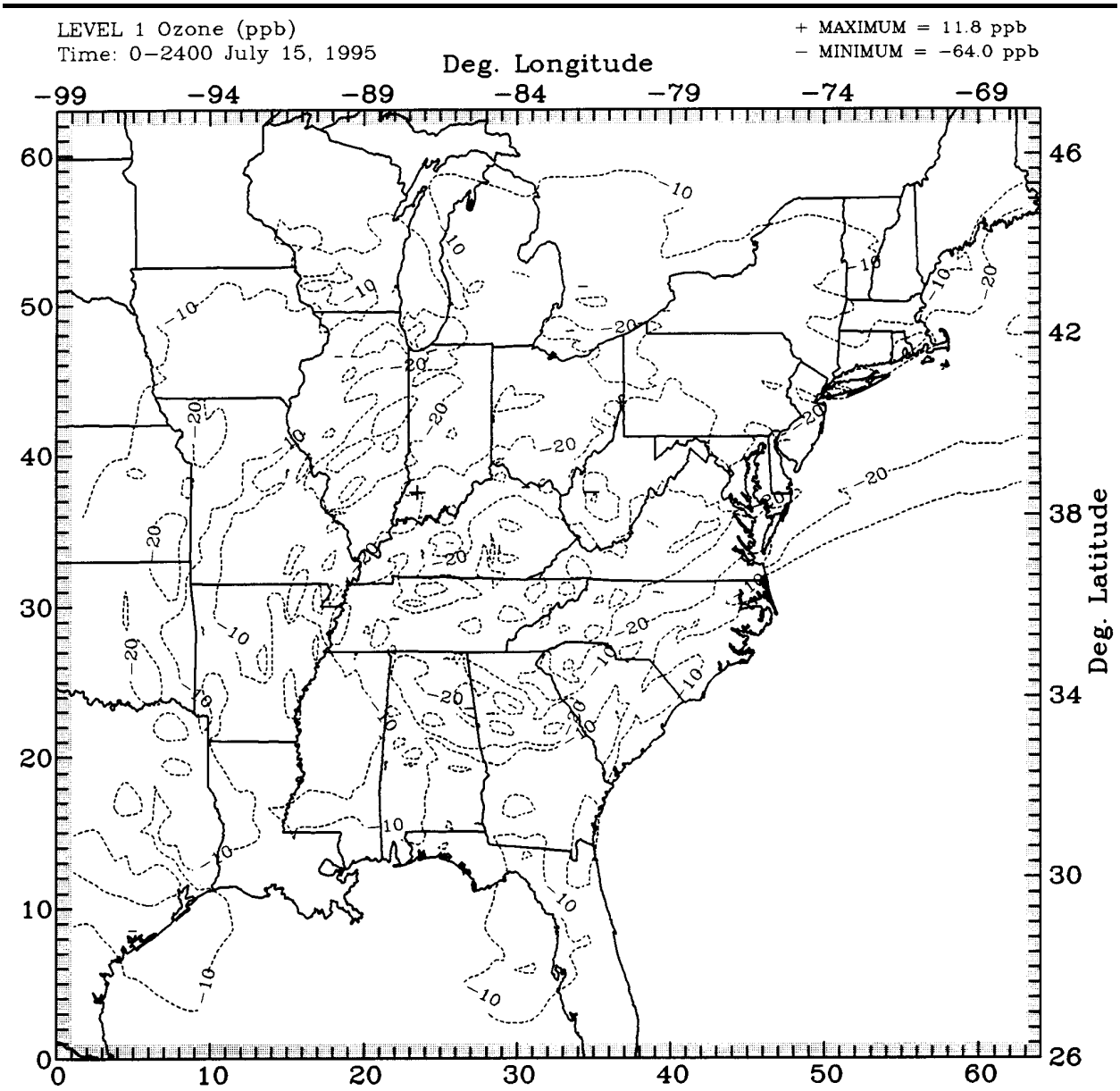



Figure C-4. Difference in daily maximum simulated ozone concentration (ppb) for the 15 July 1995 OTAG episode day: 2010 post-CAAA90 minus pre-CAAA90.

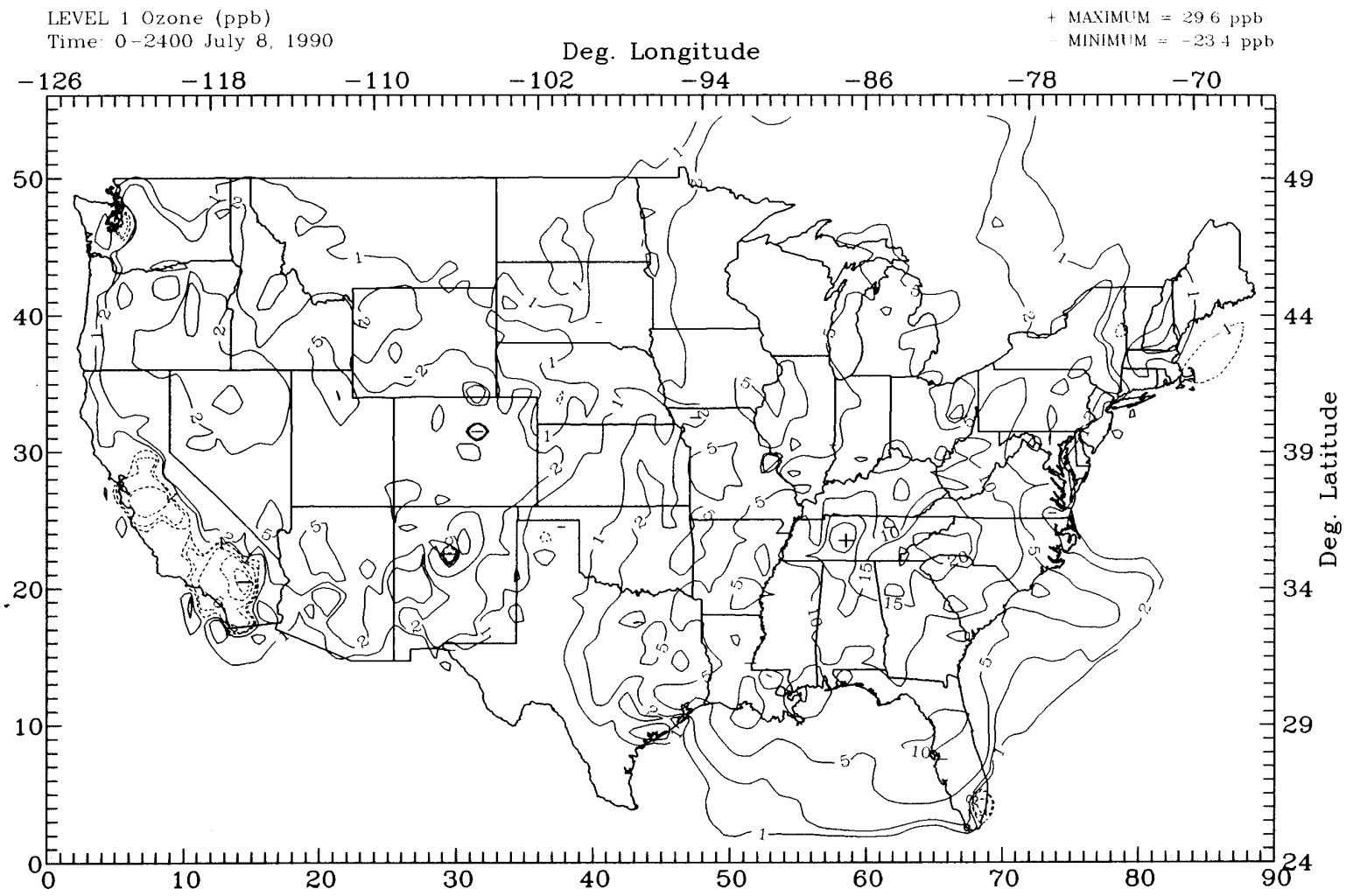



Figure C-5. Difference in daily maximum simulated ozone concentration (ppb) for the 8 July 1990 western U.S. simulation day: 2010 pre-CAAA90 minus base 1990.

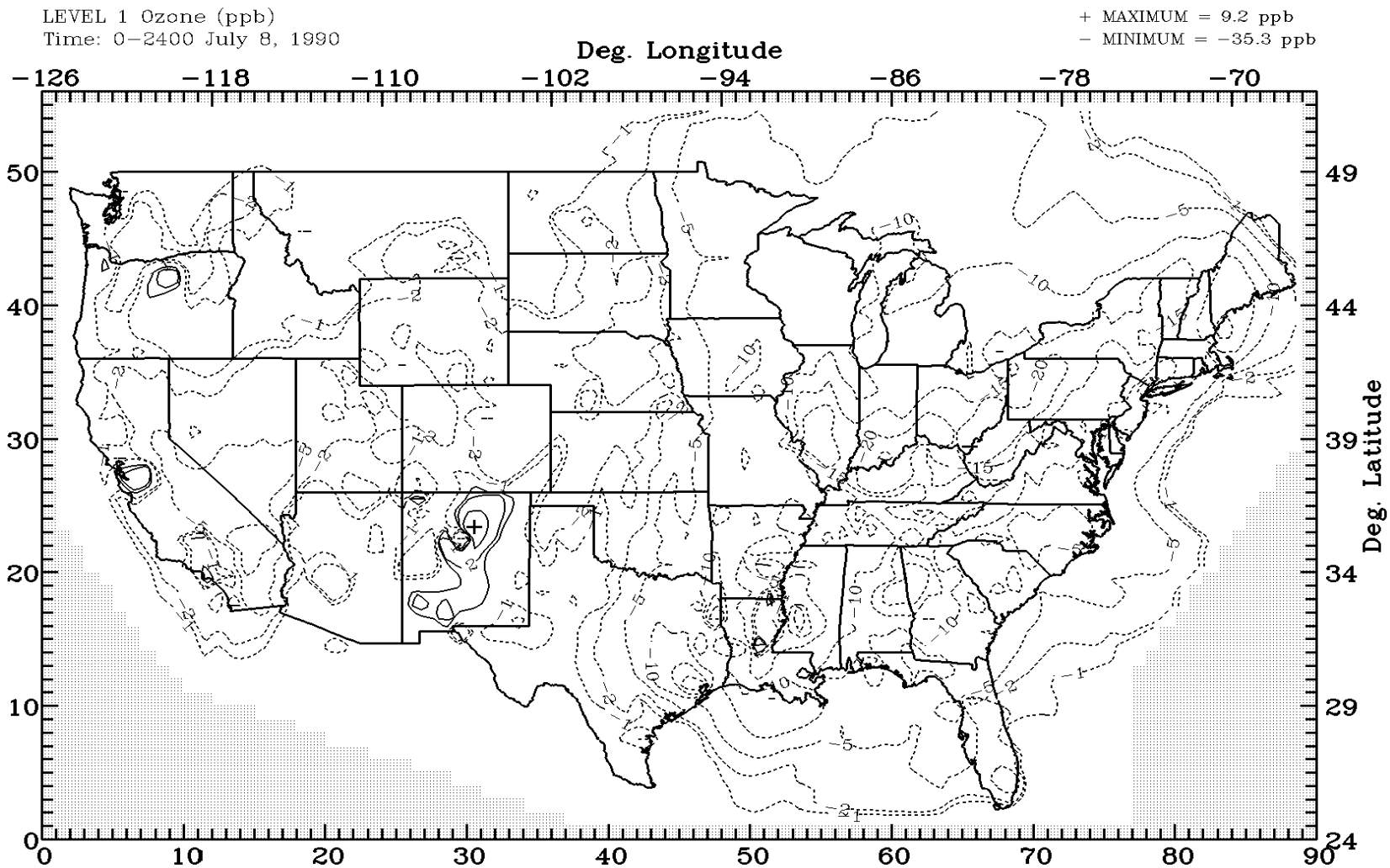



Figure C-6. Difference in daily maximum simulated ozone concentration (ppb) for the 8 July 1990 western U.S. simulation day: 2010 post-CAAA90 minus base 1990.

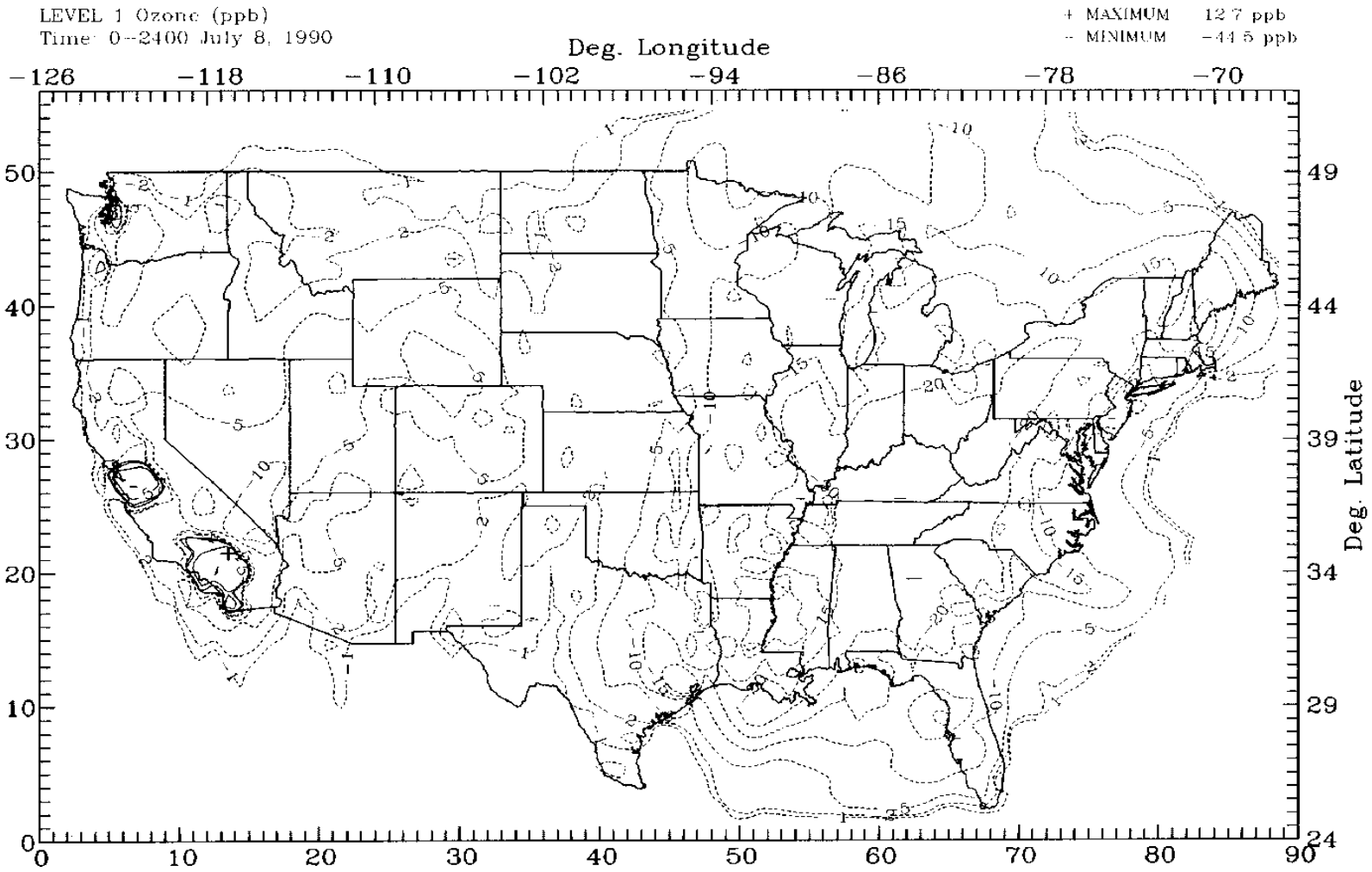



Figure C-7. Difference in daily maximum simulated ozone concentration (ppb) for the 8 July 1990 western U.S. simulation day: 2010 post-CAAA90 minus pre-CAAA90.

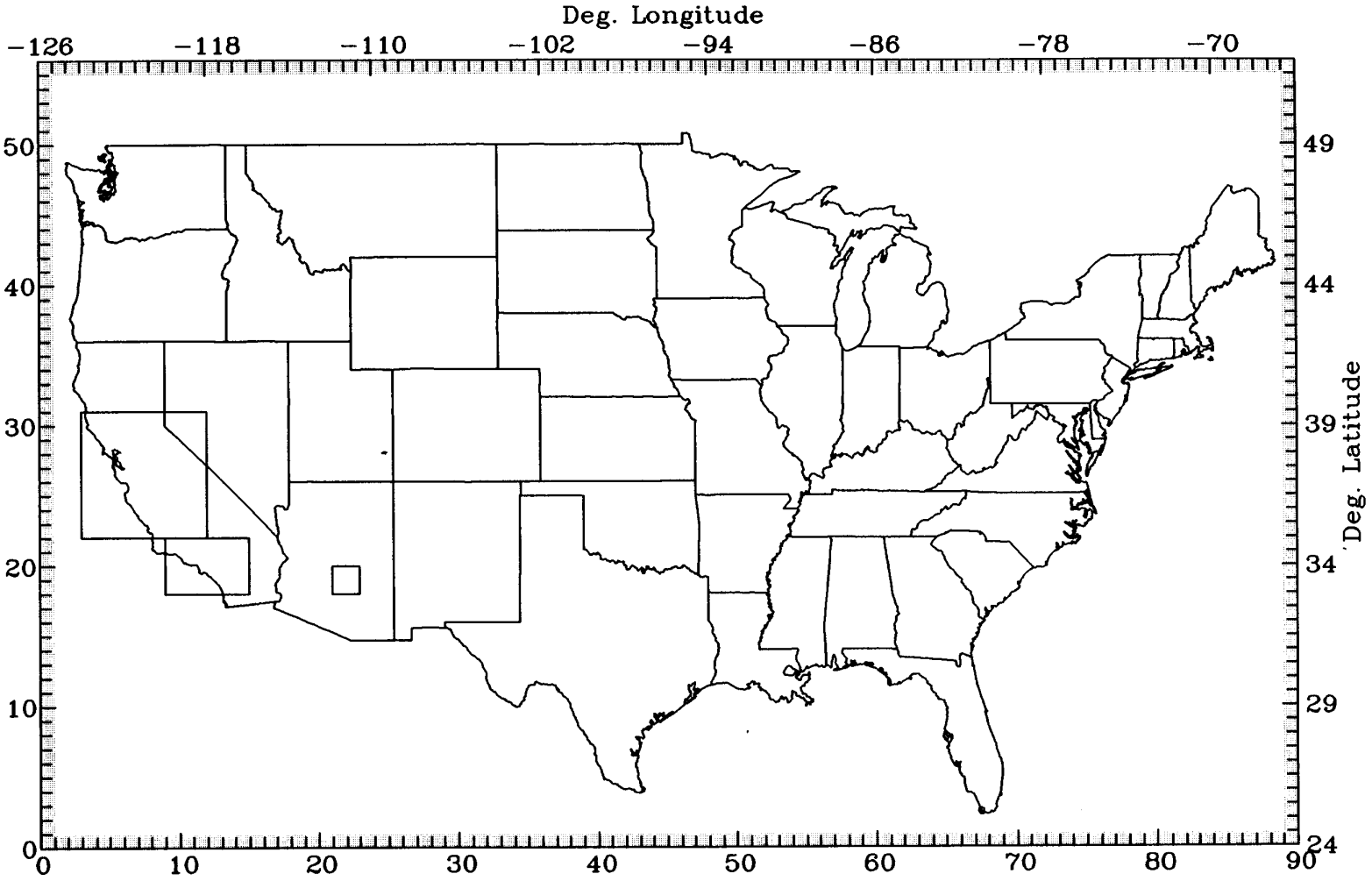



Figure C-8. UAM-V modeling domain for western U.S. analysis with the high-resolution modeling domains for the San Francisco Bay Area, Los Angeles, and Phoenix.

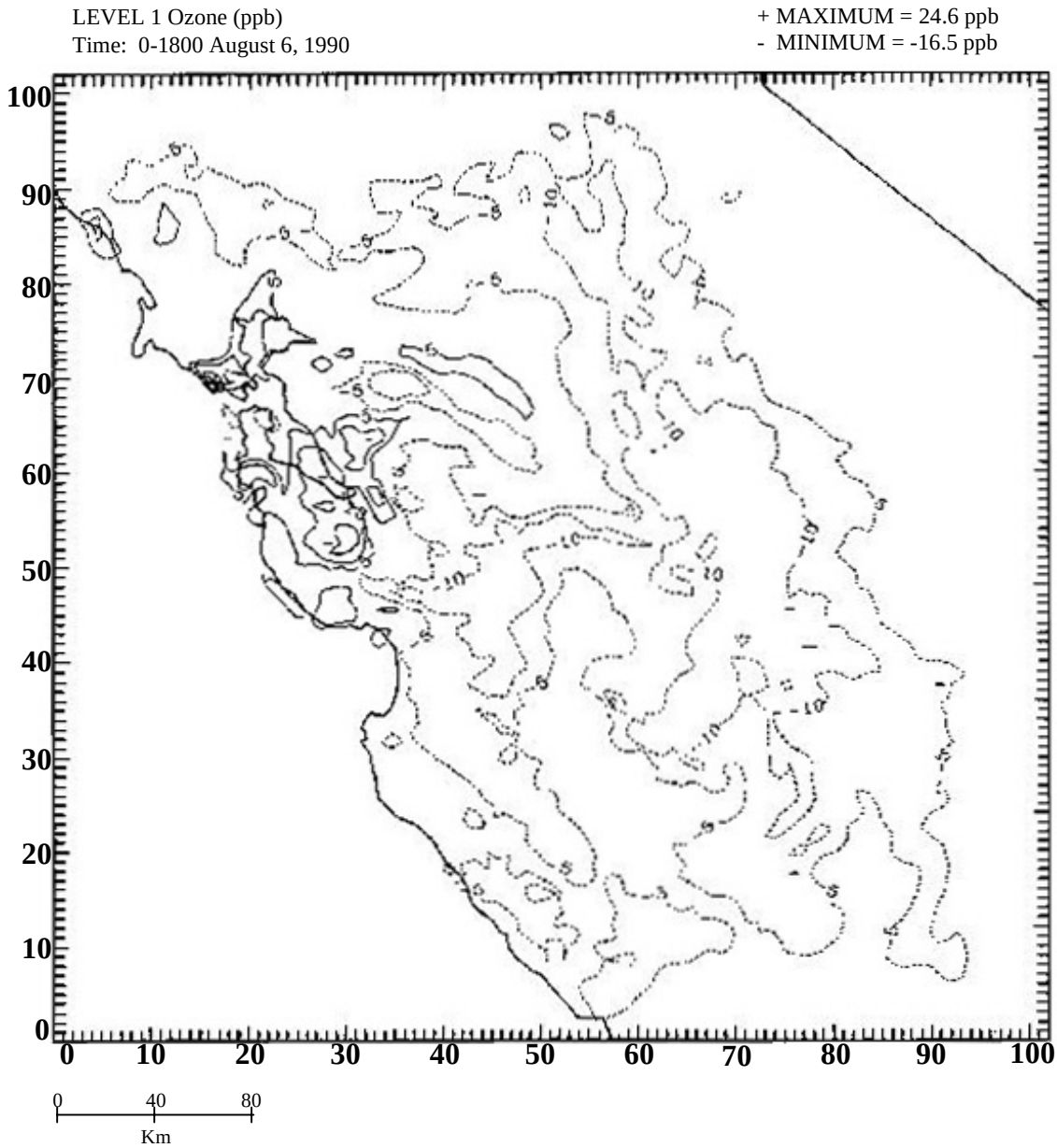



Figure C-9. Differences in daily maximum simulated ozone concentration (ppb) for the 6 August 1990 simulation day for northern California: 2010 post-CAAA90 minus pre-CAAA90.

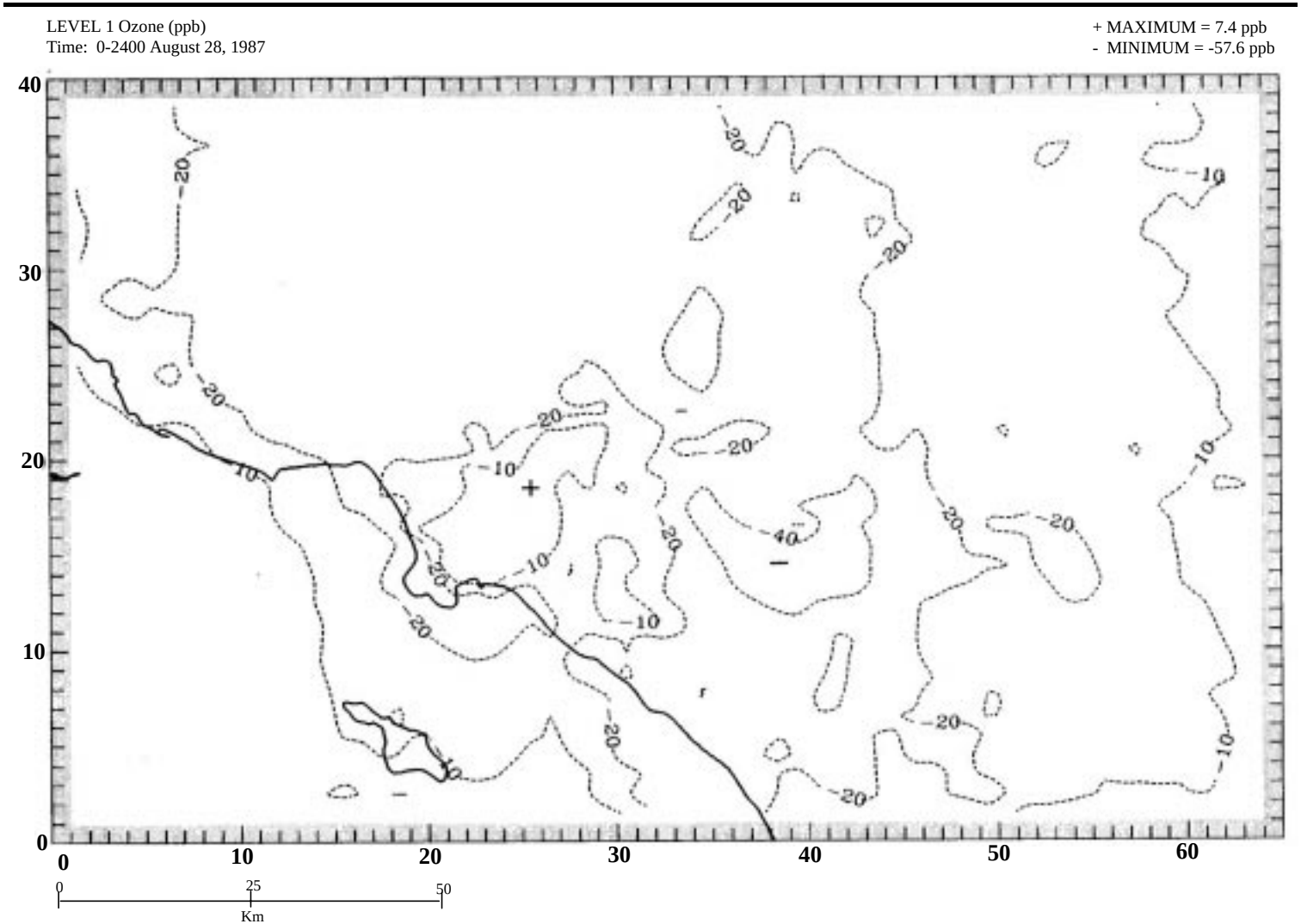



Figure C-10. Difference in daily maximum simulated ozone concentration (ppb) for the 28 July 1987 simulated day for Los Angeles: 2010 post-CAAA90 minus pre-CAAA90.

**Figure C-11a. Distribution of Monitor-Level Ratios for 95th Percentiles Ozone Concentration: 2000 Pre-CAAA / 1990 Base-Year**

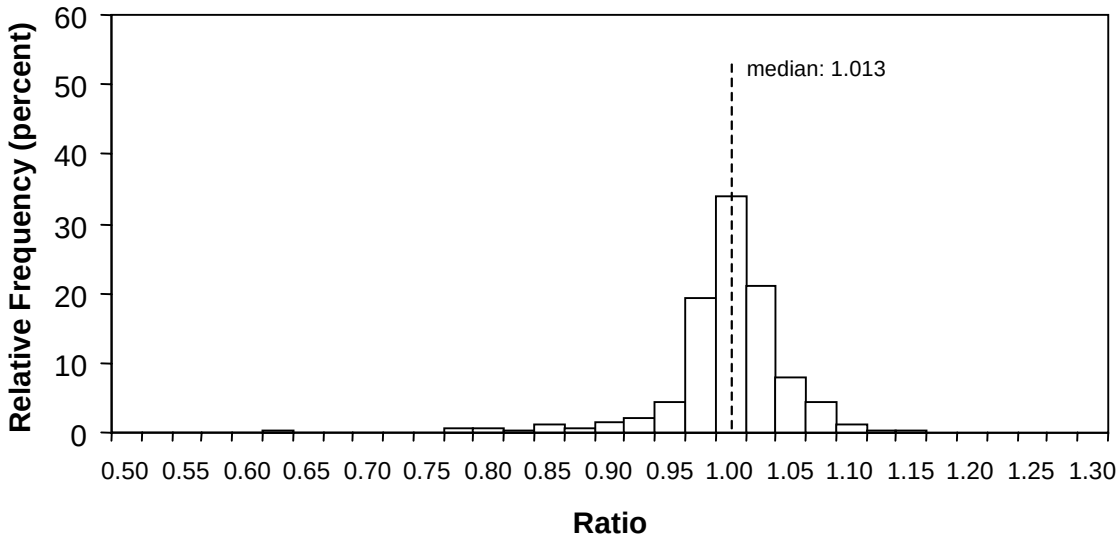



**Figure C-11b. Distribution of Monitor-Level Ratios for 95th Percentiles Ozone Concentration: 2000 Post-CAAA / 1990 Base-Year**

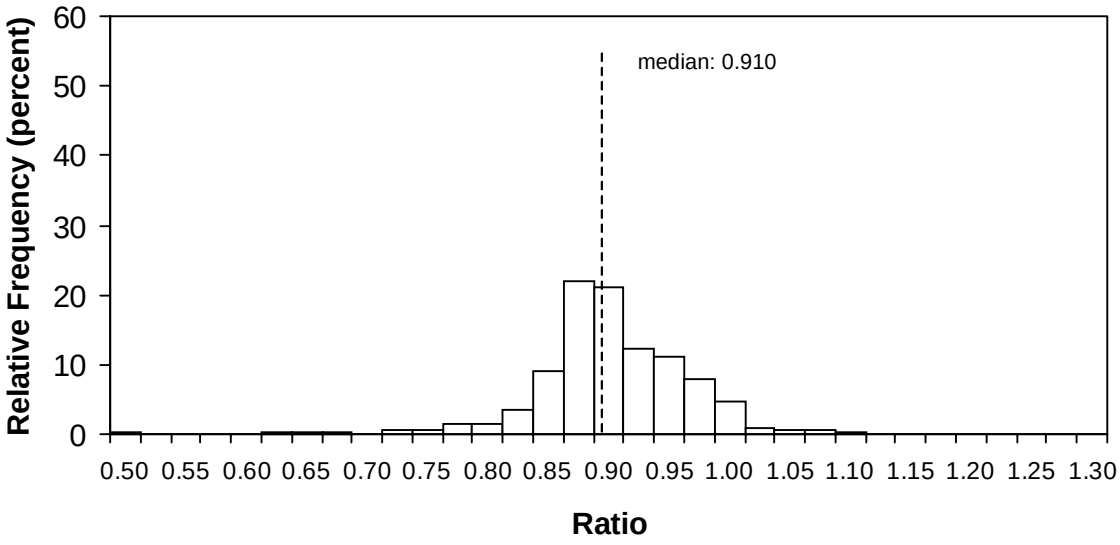


**Figure C-12a. Distribution of Monitor-Level Ratios for 95th Percentiles Ozone Concentration: 2010 Pre-CAAA / 1990 Base-Year**

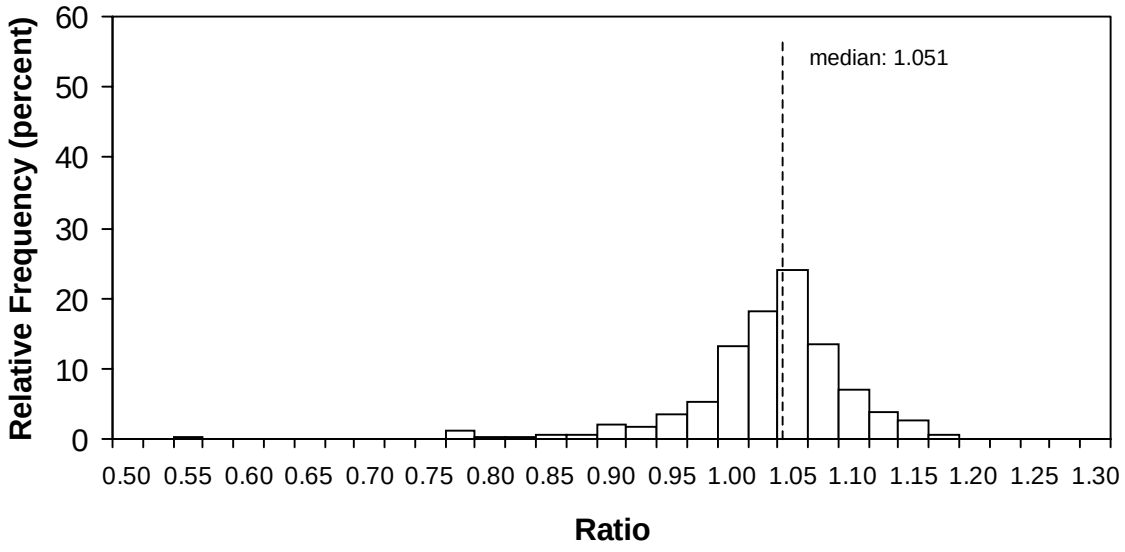



**Figure C-12b. Distribution of Monitor-Level Ratios for 95th Percentile Ozone Concentration: 2010 Post-CAAA / 1990 Base-Year**

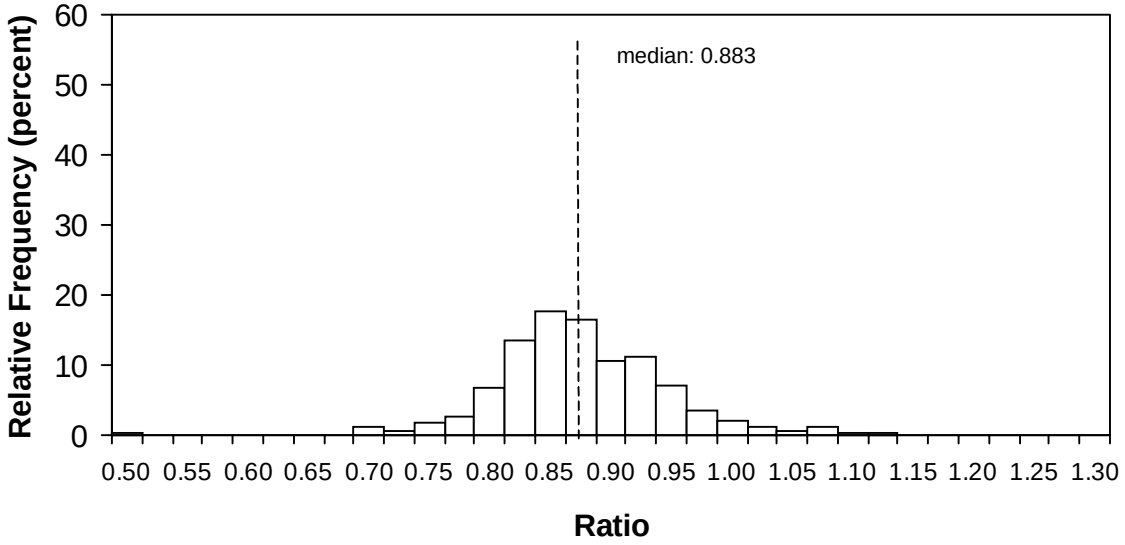


**Figure C-13a. Distribution of Monitor-Level Ratios for 95th Percentiles Ozone Concentration: 2000 Post-CAAA / 2000 Pre-CAAA**

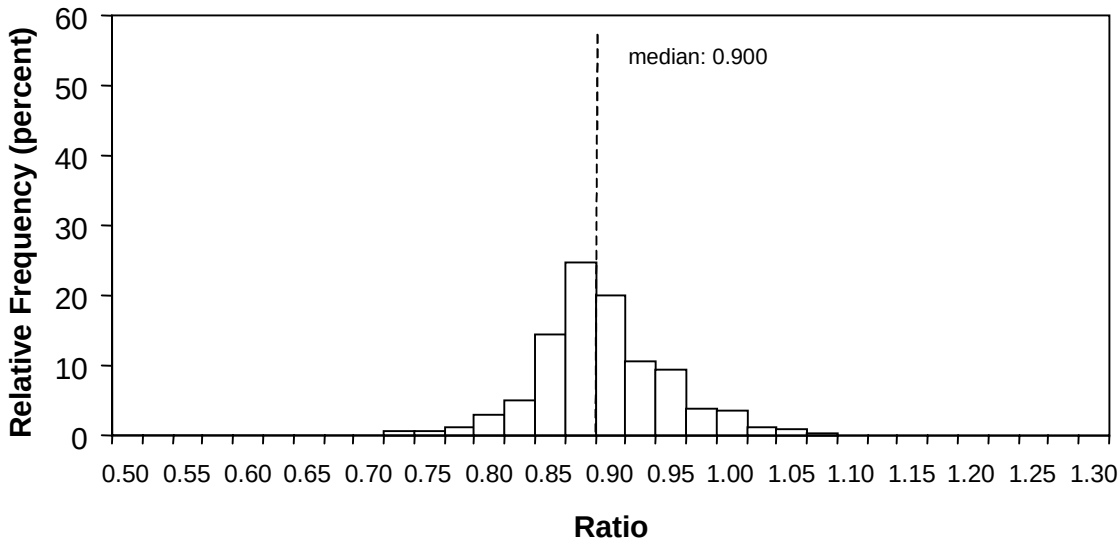
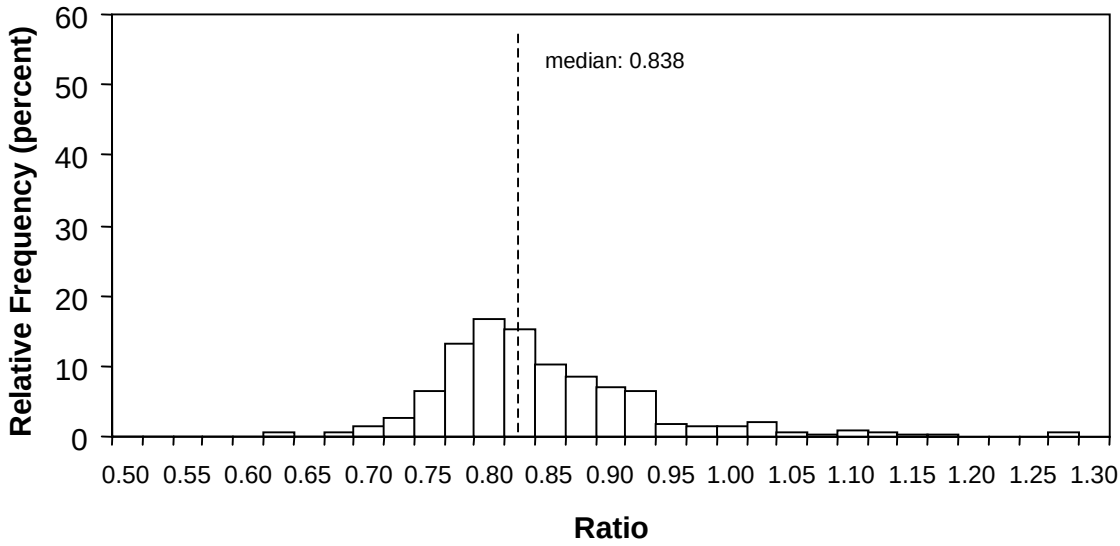



**Figure C-13b. Distribution of Monitor-Level Ratios for 95th Percentiles Ozone Concentration: 2010 Post-CAAA / 2010 Pre-CAAA**



## Estimating the Effects of the CAAA on Particulate Matter

Future-year concentrations of $PM_{10}$ and $PM_{2.5}$ corresponding to the Post-CAAA and Pre-CAAA scenarios were estimated through application of the RADM/RPM and REMSAD modeling systems. The former was used for the eastern U.S., while the latter was applied for the western U.S. Details of both RADM/RPM and REMSAD modeling are presented in this section. Included is an overview of each modeling system, and a description of the application procedures and modeling results. The calculation of PM air quality profiles using the combined modeling results from both models is also described.

For ease of reading, all figures follow the text in this section.

### *Overview of the RADM/RPM Modeling System*

RADM was developed over a ten-year period, 1984-1993, under the auspices of the National Acid Precipitation Assessment Program (NAPAP) to help address policy and technical issues associated with acid deposition. More recently, EPA created the Regional Particulate Model, expanding the Agency's atmospheric modeling capabilities. Functioning together, RADM and RPM help predict PM concentrations by generating estimates of secondary paticulates that comprise a significant portion of total PM.

RADM, a three-dimensional Eulerian grid-based model, is designed to provide a scientific basis for predicting regional air pollution concentrations and levels of acid deposition resulting from changes in precursor emissions. The concentration of a specific pollutant in a grid cell at a specified time is determined by the following factors:

- the emissions rate;
- the transport of that species by wind into and out of the grid in three dimensions;
- movement of the atmosphere via turbulent motion;
- chemical reactions that either produce or deplete the chemical;
- the change in concentration due to vertical transport by clouds;
- aqueous chemical transformation and scavenging; and
- removal by deposition.[3]

RPM is an extension of RADM. Like RADM, RPM is a three-dimensional Eulerian air quality model. Functioning in tandem with RADM, RPM predicts the chemistry, transport, and dynamics of the secondary aerosols of sulfate, nitrate, ammonium, and organics.[4] For this study, however, RPM organic aerosol estimates were not included in the final analysis because the model significantly underestimates organics and the reason for this systematic underestimation has not yet been characterized. The model's predictions of secondary sulfate, nitrate, and ammonium concentrations were used to develop particulate matter concentration estimates.

### *Application of RADM/RPM for the Eastern U.S.*

In this analysis, the RADM/RPM modeling system was used to estimate future year nitrate and sulfate concentrations, two major components of secondary PM. These model results were then used to generate adjustment factors, which in turn aided development of PM predictions for the eastern half of the United States. A summary of the model's application and results follows.

#### Modeling Domain

The domain of application for both RADM and RPM is eastern North America, from the Rocky

[3]A more detailed description of RADM is provided in R. Dennis, 1995.

[4]A more detailed description of the structure and basic features of RPM is given in F.S. Binkowski and U. Shankar, 1995.

Mountains eastward to Newfoundland, Canada and the Florida Keys. This expansive model area that includes part of Southern Canada allows RADM/RPM to accurately reflect the several-day residence times of sulfur and nitrogen species in the atmosphere and the resulting transport distances of 1,000 kilometers (km) or more that may be covered during that time. The 2,800 by 3,040 km model domain is divided into 80-km grid cells. Nested within this domain are a set of finer resolution 20-km grid cells, covering the geographic region extending eastward from central Illinois to the Atlantic Ocean and southward from Sudbury, Ontario to the upper section of North Carolina (Figure C-14). The model also consists of vertical layers that, in total, stretch 16 km above ground level.

## Simulation Periods

RADM/RPM model runs were conducted for 30 five-day periods. The 30 periods, which represent dominant transport regimes spanning four years, were randomly selected to develop annual averages. Later, to develop warm season (May through September) and cold season (October through April) averages, they were divided into these two seasonal groups. Annual warm and cold season averages were developed using a weighting scheme based on the frequency of occurrence of transport regimes. To avoid the influence of the model starting up and adjusting to a new set of conditions associated with each period, only results from the last three days of each period were used to estimate PM levels.

## Model Inputs

### RADM

Detailed emissions and meteorological data are required to run RADM. The emissions inventory for the model must account for both the timing and location of emissions. Accurate model predictions also depend on a host of meteorological inputs, most notably temperature, wind speed, and wind direction.

Separate emissions inventories were used as input in this analysis for each of the emissions scenarios: 1990 base year, 2000 Pre-CAAA, 2000 Post-CAAA, 2010 Pre-CAAA, and 2010 Post-CAAA.[5] These scenarios and their accompanying inventories, described in more detail in Appendix A, incorporate emissions from all five major source categories: industrial point sources, utilities, nonroad engines/vehicles, motor vehicles, and area sources. This inventory for each scenario contains hourly, day-specific emissions figures for every source category; area and mobile source data are provided at the county level, while utility and industrial point source emissions are given at the source classification code level.

Biogenic emissions were also included in the RADM input. This inventory was developed from version two of EPA's Biogenic Emissions Inventory System (BIES-2). BEIS-2 estimates biogenic emissions based on a variety of factors including biomass and emissions factors.

The meteorological inputs for RADM were derived using output from the Pennsylvania State University/National Center for Atmospheric Research (PSU/NCAR) mesoscale model (MM4). Using MM4 results, EPA generated essential grid-specific RADM input, including wind flow patterns, temperatures, and water vapor concentrations.

### RPM

RPM requires inputs similar to those described for RADM. This model uses a subset of RADM emissions data and the RADM meteorological fields. Additional RPM inputs include atmospheric water data generated by RADM and RADM-predicted levels of oxidants, nitric acid, and ammonia.

[5]See Pechan, June 1998 for a detailed description of the emissions scenarios developed for this analysis.

## RADM/RPM Simulation Results

### Model Performance

The assessment of model performance for particulate models is a difficult task due to a relative lack of data and information regarding the spatial distribution, composition, and size fractionation of airborne particulates. Development and evaluation of particulate measurement and modeling techniques are active areas of research. As a result, there are currently no standard approaches or model performance criteria for the evaluation of regional-scale particulate models.

Development of RADM began in the mid-1980's. The evolution of this model, along with its application and performance evaluation have all been documented extensively by NAPAP.[6] RADM continues to undergo periodic peer review, evaluations, and improvements.[7] In addition to the present study and the section 812 retrospective analysis, RADM has been used in other Agency studies of acid deposition[8] and in assessments of deposition of nitrogen to coastal estuaries.[9]

RPM was evaluated by comparing the model's 1990 base year seasonal nitrate and sulfate estimates with observed data measured by EPA's Clean Air Act Status and Trends Network (CASTNet). CASTNet is a network of monitors distributed throughout the Eastern U.S. that measures dry deposition of atmospheric sulfur and nitrogen compounds. RPM predictions for particulate sulfate and CASTNet data are provided in Table C-6. Examination of these ambient concentrations shows that RPM predicts the significant seasonal differences in sulfate production, although the model overestimates the annual average sulfate concentration by approximately 20 percent.

Table C-7 displays RPM and CASTNet seasonal average nitrate concentrations and ratios showing the fraction of total nitrate that is in particulate form. Comparison of the values in this table indicate that RPM accurately captures the ratio of particulate to total nitrate, but underestimates overall nitrate levels in the colder months and overestimates them during the warmer months. Averaged over the entire year, however, RPM results and CASTNet data are similar.

**Table C-6**
**Comparison of CASTNet and RPM Average Concentration of $SO_4$**

| Season | CASTNet $SO_4$ ($\mu g/m^3$) | RPM $SO_4$ ($\mu g/m^3$) |
|---|---|---|
| Warm | 7.8 | 9.1 |
| Cold | 3.7 | 3.6 |
| Annual | 5.4 | 6.6 |

[6]Chang, J. et al. 1987, Chang, J. et al. 1990, and Dennis, R. et al. 1990.

[7]Dennis, R. et al. 1993, McHenry J. and Dennis, R. 1994, and External Review Panel 1994.

[8]U.S. EPA, 1995.

[9]Dennis, R. 1997 and EPA 1997.

**Table C-7**
**Comparison of CASTNet and RPM**
**Average Concentrations and Fractions of $NO_3$**

| Season | CASTNet $NO_3$ ($\mu g/m^3$) | RPM $NO_3$ ($\mu g/m^3$) | CASTNet $NO_3$/t-$NO_3$ (ratio) | RPM $NO_3$/t-$NO_3$ (ratio) |
|---|---|---|---|---|
| Autumn | 1.39 | 1.25 | 0.42 | 0.42 |
| Winter | 1.67 | 1.01 | 0.44 | 0.44 |
| Spring | 0.85 | 1.07 | 0.24 | 0.24 |
| Summer | 0.42 | 0.57 | 0.14 | 0.10 |
| Annual | 1.06 | 1.06 | 0.31 | 0.27 |

## RADM/RPM Modeling Results

RADM/RPM generated estimates of nitrate and sulfate concentrations for the years 2000 and 2010 under both the Pre- and Post-CAAA scenarios. These two constituents are major components of secondary PM. As described in more detail later in this appendix, these RADM/RPM results were used to project 1990 observed nitrate and sulfate concentrations to future year levels. From these future year estimates, monitor-level $PM_{10}$ and $PM_{2.5}$ concentrations were calculated for 2000 and 2010.

Comparison of 1990 base year PM levels with future year Pre- and Post-CAAA estimates shows that under the Pre-CAAA scenario, concentrations of $PM_{10}$ and $PM_{2.5}$ are generally expected to increase from base year levels. Under the Post-CAAA scenario, both $PM_{10}$ and $PM_{2.5}$ concentrations are predicted to decrease throughout much of the U.S. in both 2000 and 2010, with greater decreases expected in 2010. The histograms in Figures C-21 through C-24 show the relationship between base year and future year PM estimates. In these figures, ratios greater than one indicate that the future year concentration is greater than the 1990 base year value, while ratios less than one indicate a lower value for the future. Figures C-25 and C-26 show the relationship between Pre- and Post-CAAA PM estimates. All of these histograms present data for the entire U.S., including RADM/RPM data for the East and REMSAD data for the West (see below).

## *Overview of the REMSAD Modeling System*

The Regulatory Modeling System for Aerosols and Deposition (REMSAD) programs have been developed to support a better understanding of the distributions, sources, and removal processes relevant to fine particles and other airborne pollutants, including soluble acidic components and toxics. Consideration of the different processes that affect primary and secondary (i.e., formed by atmospheric processes) particulate matter at the regional scale in different places is fundamental to advancing this understanding and to assessing the effects of proposed pollution control measures. These same control measures will, in most cases, affect ozone, particulate matter and deposition of pollutants to the surface.

The REMSAD system was initially focused on atmospheric aerosols and the deposition of toxic pollutants such as mercury from the air to the surface. Any modeling system for aerosols and deposition must be built on the foundation of an atmospheric transport and dispersion model. Many atmospheric dispersion models have been developed since the late 1970s and applied for various purposes. Urban and regional air quality models are generally based on the Eulerian approach. The REMSAD system is built on the foundation of the UAM-V regional air quality model, which includes a number of advantageous capabilities. The REMSAD aerosol and toxics

deposition module (ATDM) is capable of "nesting" a finer-scale subgrid within a coarser overall grid, which permits high resolution over receptor regions without an intolerable computing burden. The modeling system may thus be applied at scales ranging from a single metropolitan region to a continent containing multiple urban areas.

The REMSAD system consists of a meteorological data preprocessor, the core aerosol and toxic deposition model (ATDM), and postprocessing programs. The ATDM is a three-dimensional grid model designed to calculate the concentrations of both inert and chemically reactive pollutants by simulating the physical and chemical processes in the atmosphere that affect pollutant concentrations. The basis for the model is the atmospheric diffusion or species continuity equation. This equation represents a mass balance in which all of the relevant emissions, transport, diffusion, chemical reactions, and removal processes are expressed in mathematical terms. The model is usually exercised over a multi-day period, typically a full year.

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

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

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

Model inputs are prepared for meteorological and emissions data for the simulation days. Once the model results have been evaluated and determined to perform within prescribed levels, a *projected* emission inventory can be used to simulate possible policy-driven emission scenarios.

REMSAD provides gridded averaged surface and multi-layer instantaneous concentrations, and surface deposition output for all species and grids simulated. The averaged surface concentrations and depositions are intended for comparison with measurements and ambient standards. The instantaneous concentration output is primarily used to restart the model, and to examine model results in the upper levels. Concentrations of particulates are passed as input to a module that estimates atmospheric visibility. Wet and dry acidic deposition fluxes are calculated hourly and may be accumulated for any desired interval.

The particulate matter species modeled by REMSAD include a primary coarse fraction

(corresponding to particulates in the 2.5 to 10 micron size range), a primary fine fraction (corresponding to particulates less than 2.5 microns in diameter), and several secondary particulates (e.g., sulfates, nitrates, and organics). The sum of the primary fine fraction and all of the secondaries is taken to be roughly representative of $PM_{2.5}$. Table C-8 lists the simulated species written to the REMSAD output files.

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

Version 4.0 of the REMSAD modeling system (with simplified ozone chemistry) was employed for this study.

**Table C-8**
**REMSAD Output File Species**

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

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

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

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

## *Application of REMSAD for the Western U.S.*

For this study, the REMSAD modeling system was applied for the analysis of PM and visibility in western U.S. Although the modeling domain includes the entire U.S. (contiguous 48 states), only the results for the western U.S. were used to calculate the future-year PM concentration profiles. However, the results for the entire domain are presented here. The application procedures and modeling results are summarized in this section.

### Modeling Domain

The REMSAD modeling domain encompasses the contiguous 48 states. The domain extends from 126 degrees west longitude to 66 degrees west longitude, and from 24 degrees north latitude to 52 degrees north latitude. A grid cell size of 2/3 longitude by 1/2 latitude (approximately 56 by 56 km) was used across the grid, resulting in a 90 by 55 grid (4,950 cells) for each vertical layer. Eight vertical layers were used for the PM modeling and the first layer results were used to estimate future air quality for the surface monitoring sites. Although REMSAD covers the entire U.S., in this analysis only results for their 11 westernmost states are used.

### Simulation Periods

Four simulation periods or episodes were modeled. These episodes correspond to the four seasons of the year and consist of the first ten days of the months of May (spring), July (summer), October (fall), and December (winter).

### Model Inputs

The REMSAD modeling system also requires a variety of input files that contain information pertaining to the modeling domain and simulation period. These include gridded, day-specific emissions estimates and meteorological fields; initial and boundary conditions; and land-use information.

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

### Modeling Emission Inventories

The data and methodologies used to prepare the REMSAD modeling emission inventories for this study were consistent with those used for the photochemical modeling, but included primary particulates and other species as required for the particulate chemistry. Similar to UAM/UAM-V, REMSAD, requires detailed emission inventories, containing temporally allocated emissions for each grid cell in the modeling domain for each species being simulated. EPS 2.5e was used for the emissions processing. Note that this system has been specifically designed to accommodate regional-scale model applications of particulate matter and toxic species as well as ozone precursors.

The emissions scenarios for this study included 1990 base, 2000 Pre-CAAA, 2000 Post-CAAA, 2010 Pre-CAAA, and 2010 Post-CAAA scenarios. Each inventory includes typical season weekday area source emissions, typical summer or winter day utility emissions (as appropriate), typical season weekday non-utility point source emissions, and typical season day biogenic emissions.

The anthropogenic input emissions inventory data were provided by Pechan (1998). These included area and point source emissions data from the National Particulates Inventory (by county and for specific point sources); county-level vehicle miles traveled (VMT) estimates; mobile-source emission factors for VOC, $NO_x$, and CO; and PM emission estimates for mobile sources. Note that road dust and other primary particulates are included in the area-source emissions file.

Seasonal biogenic emission estimates for the domain were prepared using version 2 of the EPA's UAM Biogenic Emissions Inventory System (BEIS-2).

BEIS-2 (which estimates biogenic emissions based on various biomass, emission, and environmental factors) utilizes land-use information to determine the distribution of biogenic emissions.

Preliminary processing of the data prior to the application of the EPS 2.5e system was necessary. This consisted of generating the on-road mobile emissions and reformatting all data into AMS and AFS workfile format. Particulate matter pollutants from on-road mobile emissions were provided at county level and were broken down into 12 different urban and rural roadway classifications. To take advantage of the temporal information provided in the utility emissions data, seasonal AFS workfiles were generated separately for the summer and winter months.

All anthropogenic emissions inputs to the various models were preprocessed through the EPS 2.5e system. Point, area, and on-road mobile source emission data were processed separately to facilitate both data tracking for quality control and the use of the data in evaluating the effects of alternative control strategies on simulated air pollutant concentrations. Temporal and spatial allocation were performed as described in Section III.

Primary particulate and secondary particulate precursor emissions are basically derived from particulate matter species, i.e., $PM_{10}$, $PM_{2.5}$, and $NH_3$. Therefore a chemical speciation scheme that differs from that for VOC speciation is applied. Table C-9 provides the chemical speciation applied for REMSAD.

**Table C-9**

**Chemical Speciation Schemes Applied for REMSAD**

| |
|---|
| *VOC*: VOC |
| *$NH_3$*: $NH_3$ |
| *$NO_x$*: $NO_X$, NO, $NO_2$ |
| *PMC*: POA, PEC, $GSO_4$, $PNO_3$, PMcoars |
| *PM*: POA, PEC, $GSO_4$, $PNO_3$, Pmfine |

Emission inputs to the REMSAD for selected species, by component, are provided in Table C-10. The purpose of the tables is to quantify the contribution of each source category to total emissions. The species shown include primary particulates and other species that are important to secondary particulate formation. VOC, $NO_x$, and $SO_2$ emissions are estimated to increase under the Pre-CAAA scenario and to decrease under the Post-CAAA scenario. For $SO_2$, the decreases come from the utility sector and are offset by increases in the other components. $NH_3$ emissions increase for both scenarios and are slightly higher under the Post-CAAA scenario for both years, presumably due to increased use of natural gas fuel. $PM_{10}$ and $PM_{2.5}$ emissions (primary particulates) are similar for all scenarios.

The primary chemical process for PM applications in REMSAD is sulfate formation. In-cloud processes can account for the majority of atmospheric sulfate formation, especially in the wintertime when gas-phase chemistry is slow. The two most important pathways for in-cloud sulfate formation are the reactions of aqueous $SO_2$ with ozone and hydrogen peroxide ($H_2O_2$). At cloud pH below 4.5 (most common in the eastern U.S.), the ozone reaction is slow and the $H_2O_2$ reaction dominates. Since the $H_2O_2$ is often present at ambient concentrations below those of $SO_2$, formation of sulfate can be limited by the availability of $H_2O_2$, and thus can be quite nonlinear. The formation of $H_2O_2$ is tied to the overall atmospheric photochemical system, and responds to changes in ambient levels of VOC and $NO_x$. Because of this link, emission changes for VOC and $NO_x$ may have effects on ambient sulfate levels. In short, the emissions of ozone and PM precursors (i.e., $NO_x$ and VOC) will affect the oxidizing capacity of the troposphere which is represented primarily by the concentrations of radicals and hydrogen peroxide, and thus affect the rate of oxidation of the $NO_x$ and $SO_2$ to nitrate and sulfate.

In REMSAD, there is no relationship between VOC emissions and the production of secondary organic aerosol (SOA).

**Table C-10**
**Emission Totals by Component for each Scenario for the Entire U.S. (tpd)**

| | Base 1990 | 2000 Pre-CAAA | 2000 Post-CAAA | 2010 Pre-CAAA | 2010 Post-CAAA |
|---|---|---|---|---|---|
| **VOC** | | | | | |
| Area | 33,972 | 39,154 | 27,620 | 43,708 | 28,575 |
| Onroad Mobile | 18,659 | 16,454 | 10,683 | 18,776 | 8,804 |
| Point | 9,503 | 10,298 | 8,457 | 11,606 | 9,454 |
| Utility | 96 | 85 | 85 | 134 | 137 |
| **Total** | **62,229** | **65,991** | **46,845** | **74,224** | **46,970** |
| **NOx** | | | | | |
| | Base 1990 | 2000 Pre-CAAA | 2000 Post-CAAA | 2010 Pre-CAAA | 2010 Post-CAAA |
| Area | 13,766 | 15,659 | 15,252 | 17,697 | 15,794 |
| Onroad Mobile | 20,399 | 20,660 | 17,421 | 24,142 | 14,696 |
| Point | 7,964 | 8,694 | 5,645 | 9,803 | 5,985 |
| Utility | 20,188 | 22,787 | 11,170 | 24,808 | 10,319 |
| **Total** | **62,316** | **67,800** | **49,487** | **76,450** | **46,793** |
| **SO2** | | | | | |
| | Base 1990 | 2000 Pre-CAAA | 2000 Post-CAAA | 2010 Pre-CAAA | 2010 Post-CAAA |
| Area | 3,517 | 4,174 | 4,174 | 4,811 | 4,811 |
| Onroad Mobile | 1,555 | 1,730 | 924 | 2,109 | 1,121 |
| Point | 12,808 | 14,620 | 14,620 | 16,422 | 16,422 |
| Utility | 43,380 | 44,261 | 28,742 | 48,482 | 27,016 |
| **Total** | **61,260** | **64,786** | **48,460** | **71,823** | **49,369** |
| **NH3** | | | | | |
| | Base 1990 | 2000 Pre-CAAA | 2000 Post-CAAA | 2010 Pre-CAAA | 2010 Post-CAAA |
| Area | 10,230 | 13,189 | 13,189 | 15,710 | 15,710 |
| Onroad Mobile | 544 | 957 | 957 | 1,191 | 1,194 |
| Point | 667 | 742 | 742 | 842 | 1,015 |
| Utility | - | - | 91 | - | 608 |
| **Total** | **11,441** | **14,888** | **14,979** | **17,744** | **18,527** |
| **PM10** | | | | | |
| | Base 1990 | 2000 Pre-CAAA | 2000 Post-CAAA | 2010 Pre-CAAA | 2010 Post-CAAA |
| Area | 73,221 | 74,431 | 72,640 | 74,532 | 72,240 |
| Onroad Mobile | 972 | 799 | 706 | 814 | 563 |
| Point | 2,549 | 2,891 | 2,891 | 3,252 | 3,252 |
| Utility | 764 | 691 | 697 | 837 | 758 |
| **Total** | **77,507** | **78,812** | **76,933** | **79,435** | **76,813** |
| **PM2.5** | | | | | |
| | Base 1990 | 2000 Pre-CAAA | 2000 Post-CAAA | 2010 Pre-CAAA | 2010 Post-CAAA |
| Area | 16,717 | 17,438 | 17,147 | 18,174 | 17,637 |
| Onroad Mobile | 797 | 618 | 536 | 640 | 391 |
| Point | 1,625 | 1,840 | 1,840 | 2,066 | 2,066 |
| Utility | 288 | 247 | 250 | 332 | 305 |
| **Total** | **19,427** | **20,143** | **19,773** | **21,212** | **20,398** |

## Air Quality, Meteorological, and Land-Use Inputs

Initial species concentrations and lateral boundary conditions were specified to approximate background concentrations of the species; for the lateral boundaries the concentrations varied (decreased parabolically) with height. The background concentrations are listed in Table C-11.

**Table C-11**
**Background Species Concentration used for REMSAD Initial and Boundary Conditions.**

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

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

## REMSAD Simulation Results

### Model Performance

The assessment of model performance for particulate models is a difficult task due to a relative lack of data and information regarding the spatial distribution, composition, and size fractionation of airborne particulates. Development and evaluation of particulate measurement and modeling techniques are active areas of research. As a result, there are currently no standard approaches or model performance criteria for the evaluation of regional-scale particulate models. For this study, model performance for REMSAD was examined by comparing the simulated values of selected species with available data. This comparison is intended to provide an indication as to whether the simulated values represent the concentration levels and the range of concentrations indicated by the available observations.

Summaries of model performance were prepared by comparing the simulated values of PM with observed values representing seasonal averages. Comparisons were performed for the entire domain (entire U.S.), the western U.S., and the eastern U.S. Only the western U.S. results are presented here. Data from both the AIRS and IMPROVE PM monitoring networks were included in the evaluation. REMSAD-derived sulfate and nitrate concentrations were also compared to a small number of IMPROVE measurements.

Scatter plots for $PM_{10}$ are provided in Figures C-15 through C-18. For $PM_{10}$, there is a tendency for underestimation of the seasonal averages in the western U.S., in particular for the fall and winter simulation periods. Similar plots for $PM_{2.5}$, sulfate, and nitrate are available in (SAI, 1999) and show generally good agreement for these species.

These plots indicate that model performance varies throughout the western U.S. and throughout the year. A closer look at the comparison between the simulated and observed values indicates that the agreement is generally better for the IMPROVE sites and that most of the large underestimation occurs for the AIRS sites. The IMPROVE sites tend to be located in rural areas, while the AIRS sites tend to be located in urban areas. There are numerous possible explanations for the differences. One possibility is that one or more components of the urban emissions may not be accurately represented in the inventory. A second possibility is that the grid resolution (approximately 56 km) is not sufficient to resolve the urban-scale processes influencing particulate formation and transport. It is encouraging that generally good agreement is achieved for the limited number of sulfate and nitrate measurements. Overall, the model performance results suggest that the REMSAD modeling system (including the meteorological, air quality, and geographical inputs) provides a reasonable basis for the Section 812 prospective modeling.

## REMSAD Modeling Results

The REMSAD simulation results for the Pre- and Post-CAAA scenarios were used in this study to calculate factors for adjustment of observed data and estimation of future-year concentration levels. These were calculated by comparing the simulated concentrations corresponding to each future-year/scenario simulation with those for the base-year simulation (1990). These comparisons indicate that for both future years and both size categories, the Pre-CAAA simulation results are characterized by increases in PM, while the Post-CAAA results show both increases and decreases. Focusing on the western U.S., the increases occur over the larger urban areas and are likely attributable to increases in areas-source emissions of precursors.

While there are increases in primary particulate emissions for some portions of the west, most of the increases are attributable to secondary particles. Isopleth maps for these comparisons are available in (SAI, 1999).

Figures C-19 and C-20 illustrate the differences in seasonal average simulated PM concentration between the Pre- and Post-CAAA simulations for 2010 for the summer period. The differences are calculated as Post-CAAA minus Pre-CAAA, so that negative values indicate lower concentrations for the Post-CAAA scenario. The simulated values for the Post-CAAA scenario are lower than the corresponding Pre-CAAA values for both years. The magnitude and spatial extent of the decreases is greater for 2010 than for 2000 (not shown).

### *Calculation of PM Air Quality Profiles*

The calculation of PM profiles for 2000 and 2010 (for assessment of the effects of the CAAA) include the use of REMSAD results for the western U.S. and RADM/RPM results for the eastern U.S. As for ozone, this was accomplished using an approach that combines observed data and air quality modeling results to estimate the future-year concentrations. While the overall approach is similar to that for ozone (as described in Section III), there are some differences. The future-year air quality profile estimation methodology for PM, as applied to the analysis of the CAAA, is described in this section.

## Overview of the Methodology

The methodology for calculation of the adjustment factors differed slightly for the RADM/RPM and REMSAD applications. For RADM/RPM the modeling results were used to calculate adjustment factors for several PM component species; for REMSAD adjustment factors

for $PM_{10}$ and $PM_{2.5}$ were computed directly from the model output. The adjustment factors for each monitoring site were calculated (using the appropriately matched values) for several different concentration levels (i.e., the changes in concentration are dependent upon concentration level). The species concentrations for each monitoring site (estimated using the observations) were then modified using the site-specific (or grid-cell-specific) adjustment factors. For RADM/RPM, PM concentrations were then recalculated using the resulting component values.

For both models, the ratios were calculated on a seasonal basis and were used accordingly to adjust the observed values. Following adjustment of the observed data, statistical quantities, or "profiles", describing the PM distribution for each monitoring site were then calculated.

## Description of the Observation Dataset

One of the first tasks in calculating the future-year PM profiles was the creation of a dataset containing the observed concentrations for all monitoring sites located within the modeling domain for the year 1990.

The starting point for this analysis is a database retrieved from the EPA Aerometric Information System (AIRS) of measured ambient concentrations of TSP, $PM_{10}$, and $PM_{2.5}$ for the year 1990. Due to the limited number of measurements (usually taken once every six days), data for 1989 and 1991 were also used to supplement the 1990 database. Cross-estimation was performed when one of measurements was missing (i.e., $PM_{10}$ or $PM_{2.5}$). The PM component species (that make up secondary PM) were estimated based on a methodology developed by Langstaff and Woolfolk (1995) for the Section 812 retrospective modeling analysis. Size fractionation ($PM_{10}$ fraction of TSP and $PM_{2.5}$ fraction of $PM_{10}$) and apportionment of secondary PM species relied on a review of previous studies to provide general relationships used to estimate these components of particulate matter. The relationships used for this study depend only on broad geographic region (East, Central, West), time of year (quarter for $PM_{10}$ and season for $PM_{2.5}$), and whether the monitor is located in an urban or rural setting.

The geographical regions used throughout this analysis are presented in Table C-12. In addition to secondary composition fractions, ratios relating $PM_{2.5}$ to $PM_{10}$ were employed. The literature review conducted for establishing secondary particulate matter concentrations for the 1990 data and the sources of ratios and apportionment factors used in the equations below is discussed in some detail by Langstaff and Woofolk (1995).

It should be noted that there is considerable variability in the size and species composition of particulate matter, not only between different locations, but also from day to day in the same location. The average size fractions and speciation factors used for this study represent a rather sweeping simplification of the actual physical phenomena that are being modeled. However, this may be justified in the context of this study, due to data limitations and the fact that the results are aggregated to the annual level.

As mentioned earlier, cross-estimation of TSP, $PM_{10}$, and $PM_{2.5}$ was used to estimate values not present in the original AIRS database. The results of a linear regression of TSP versus $PM_{10}$ by region, quarter, and land-use were used to fill in either $PM_{10}$ or TSP, if the other was missing. After this, the results of a linear regression of $PM_{2.5}$ versus $PM_{10}$ by region, season, and land use were then used to fill in $PM_{2.5}$ values where missing. With both estimated and observed TSP, $PM_{10}$, and $PM_{2.5}$, the coarse PM concentration was calculated as well as the PM concentration greater than 10 microns.

$$PM_{>10} = TSP - PM_{10} \quad (1)$$

$$PM_{C} = PM_{10} - PM_{2.5} \quad (2)$$

**Table C-12**
**Geographical Regions of the U.S.**

| Central | East | West |
|---|---|---|
| Oklahoma | Indiana | Nevada |
| Missouri | Kentucky | Utah |
| Kansas | Ohio | Colorado |
| Nebraska | Michigan | New Mexico |
| Iowa | Virginia | Arizona |
| South Dakota | West Virginia | Texas |
| North Dakota | Pennsylvania | California |
| Minnesota | New York | Oregon |
| Wisconsin | Maryland | Washington |
| Illinois | New Jersey | Idaho |
| | Connecticut | Wyoming |
| | Rhode Island | Montana |
| | Massachusetts | |
| | Vermont | |
| | New Hampshire | |
| | Maine | |
| | Delaware | |
| | Washington, DC | |
| | Florida | |
| | Georgia | |
| | Alabama | |
| | Mississippi | |
| | Louisiana | |
| | Arkansas | |
| | Tennessee | |
| | North Carolina | |
| | South Carolina | |

$PM_{2.5}$ and coarse PM were partitioned into secondary particulate concentrations. As shown below, each equation illustrates how the secondary particulate concentrations are calculated from coarse and fine PM.

$$S = [PM_{2.5} * r_{s\,2.5}] + [PM_C * r_{s\,C}] \quad (3)$$

$$N = [PM_{2.5} * r_{n\,2.5}] + [PM_C * r_{n\,C}] \quad (4)$$

$$O = [PM_{2.5} * r_{o\,2.5}] + [PM_C * r_{o\,C}] \quad (5)$$

$$P = [PM_{2.5} * r_{p\,2.5}] + [PM_C * r_{p\,C}] \quad (6)$$

where

S = sulfate concentration
N = nitrate concentration
O = organic concentration
P = other particulate concentration
$PM_{2.5}$ = PM less than or equal to 2.5 microns in size
$PM_C$ = PM between 2.5 and 10 microns in size (coarse PM)
$r_{x\,2.5}$ = ratio of <=2.5 micron sulfate (x=s), nitrate (x=n), organic (x=o), and other particulate (x=P) to $PM_{2.5}$
$r_{x\,C}$ = ratio of 2.5-10 micron sulfate (x=s), nitrate (x=n), organic (x=o), and other particulate (x=P) to coarse PM

Note that $r_{xx}$ was based on a review of available data/literature and depends on geographic region,

time of year, and land-use characteristics of the monitoring site location.

The observed and estimated species concentrations were then input into a single AMP350-format datafile. From the information contained in this file, two SAS datasets were created: a concentration dataset and a monitor information dataset. The concentration dataset contains the daily concentrations for each monitor, with each record in the dataset representing a single monitor-day. The monitor information dataset contains monitor-specific information such as land-use and location.

Because PM monitors are typically operated on a one-in-six day monitoring schedule, calculating percentiles for the PM profiles using data for a single year can be very sensitive to the method used in the percentile calculation. This is especially true when a monitor record only needs to be 50 percent complete (i.e., contain at least 30 values) for a profile to be generated. To minimize dependence on the form of the percentile equation, the 1990 PM data were supplemented with that from the years 1989 and 1991. In using multiple years worth of monitoring data, it was discovered that the identifier (ID) corresponding to a monitor in a given physical location could change from one year to the next. Also, a monitor could have moved to a nearby location and been assigned a different ID. It was also possible that the monitor ID for a $PM_{10}$ monitor might be different from that of a TSP or $PM_{2.5}$ monitor despite the fact that their physical separation is zero. Because much of the profile work is dependent upon the monitor ID, this led to a vast increase in the reported number of operating monitors.

To accommodate these possibilities, monitors with different monitor ID's were considered the same monitor if their physical separation was less than or equal to 1 km. Monitoring data from the two monitors were combined. If data existed for both of the monitors on the same day, the daily data from the monitor with the higher ID was removed.

For particulate data, a monitor record was considered to be complete if data were available for 50 percent of the 24-hour observations for a given year (assuming a one-in-six day monitoring schedule). Although three years worth of data were used for the PM analysis, these data were considered to represent one year with respect to the completeness requirement. There were 2048 PM monitors with complete data.

## Calculation of Percentile-Based Adjustment Factors

For each future-year modeling scenario, grid-cell-species-season-specific adjustment factors were calculated using the speciated, daily-simulated concentrations from RADM/RPM and REMSAD. Because the species and seasons differed between the two models, the exact calculation of adjustment factors also differed. Nevertheless, the overall approach was the same. Individual monitoring sites were mapped onto the gridded output (to determine the grid cell in which each monitor was located) and the concentrations for the corresponding grid cells were used to calculate a set of adjustment factors for each species, season, and future-year modeling scenario. The adjustment factors were specified to be the ratio of the percentile concentrations for the future- and base-year simulations of a given species-season, where the percentile concentrations were calculated using data for the selected species and season concentrations:

$$\text{Adjustment Factor}_{i,species,season} = \frac{\text{xth Percentile Concentration}_{future\ year,species,season}}{\text{xth Percentile Concentration}_{base\ year,species,season}}$$

$$\{x_i\} = \{10, 30, 50, 70, 90\}$$

For calculation of the percentile concentrations, the empirical distribution function with averaging was employed. Because the concentrations for the lower percentiles can be rather small, a threshold value of 0.01 microgram/m$^3$ was set to keep the adjustment factors reasonable. In other words, all concentrations below 0.01 microgram/m$^3$ were reset to 0.01

microgram/m$^3$. If either the base year or the future ear percentile concentration was set to the minimum value, the adjustment factor was set equal to one. This percentile-based approach was selected due to the limitations of using a single adjustment to represent the change in the modeled PM species concentrations in moving from the base- to the future-year scenarios.

For RADM, adjustment factors were calculated for the sum of sulfate, nitrate, and ammonium. These were calculated for the entire year (i.e., only one "season"). For REMSAD, the adjustment factors were calculated for $PM_{10}$ and $PM_{2.5}$. These were calculated on a seasonal basis.

A SAS dataset containing the monitor-level adjustment factors was created for each future-year modeling scenario considered in this study for this year.

## Use of Adjustment Factors to Modify Observed Concentrations

Using the calculated adjustment factors for each future-year scenario and the monitor-level observations, a dataset containing modified $PM_{10}$ and $PM_{2.5}$ concentrations for each of the four future-year scenarios was created. Because each monitor has five adjustment factors per scenario, species, and season, it was first necessary to rank order the observed concentrations into five quintile-based groups (with ties being assigned to the higher group) with respect to the species and season definitions mentioned previously. Thus for RADM, the quintiles were calculated for the daily sum of the observed sulfate, nitrate, and ammonium concentrations over the entire year (ignoring that the data are actually for the years 1989, 1990, and 1991). For REMSAD the quintiles were calculated for the observed daily $PM_{10}$ and $PM_{2.5}$ over each of the four seasons. Once each of the observed concentrations was identified with a particular quintile group, the appropriate adjustment factor was selected and applied to calculate the future-year-scenario $PM_{10}$ and $PM_{2.5}$.

For RADM, the adjustment factor was applied using the following equations:

$$AdjNitrateSulfate_i = ObsNitrateSulfate_i * Adj.Factor_{k[ObsNitrateSulfatei],NitrateSulfate}$$
$$AdjOrganics_i = ObsOrganics_i * 1$$
$$AdjP_i = ObsP_i * 1$$

For example in the first equation, $\{ObsNitrateSulfate_i\}$ is the set of observed daily sums of the nitrate and sulfate concentrations (in micrograms/m$^3$) for a given monitor. The $k[ObsNitrateSulfate_i]$ subscript is the number of the quintile group to which $ObsNitrateSulfate_i$ belongs. $Adj.Factor_{k[ObsNitrateSulfatei],NitrateSulfate}$ is then the appropriate adjustment factor for $ObsNitrateSulfate_i$. The resulting set of adjusted daily sums of nitrate and sulfate concentrations, $\{AdjNitrateSulfate_i\}$, represents the future year estimates of the daily sum of nitrate and sulfate concentrations. In this case, $P$ represents other particulate components. For those monitors within the RADM domain, $PM_{10}$ and $PM_{2.5}$ concentrations were calculated by summing each of the above components.

For monitors within the REMSAD domain, the procedure for calculating the future-year $PM_{10}$ and $PM_{2.5}$ is more direct. Future-year concentrations of these two PM species are calculated using the observed/estimated $PM_{10}$ and $PM_{2.5}$ concentrations and the appropriate adjustment factors:

$$AdjPM10_i = ObsPM10_i * Adj.Factor_{k[ObsPM10i],PM10,season}$$
$$AdjPM2.5_i = ObsPM2.5_i * Adj.Factor_{k[ObsPM2.5i],PM2.5,season}$$

In the first equation, $\{ObsPM10_i\}$ is the set of observed daily $PM_{10}$ concentrations (in micrograms/m$^3$) for a given monitor. The $k[ObsPM10_i]$ subscript is the number of the quintile group (based on season) to which $ObsPM10_i$ belongs. $Adj.Factor_{k[ObsPM10i],PM10,season}$ is then the appropriate adjustment factor for $ObsPM10_i$. The resulting set of $PM_{10}$ and $PM_{2.5}$ concentrations, $\{ObsPM10_i\}$ and $\{ObsPM2.5_i\}$, therefore represents the future-year estimates of these PM species.

## Calculation of PM Profiles

$PM_{10}$ and $PM_{2.5}$ air quality profile databases were compiled for all simulations performed as part of the Section 812 prospective analysis. For each of the particulate species, these data bases contained the number, the arithmetic mean, the median, the annual second highest, and the 2.5 to 97.5 percentiles (in increments of five) of the daily (as available) concentrations. The profiles are reported at the monitor level and include 2048 site locations.

The histograms in Figures C-21a through C-24b illustrate the distribution of ratios for the annual average monitor-level $PM_{10}$ and $PM_{2.5}$ concentrations corresponding to the 2000 and 2010 simulations. In these figures, ratios greater than one indicate that the future-year/scenario concentration is greater than the base-year (1990) value, whereas ratios less than one indicate a lower value for the future-year.

The 2000 Pre-CAAA ratios for $PM_{10}$ (Figure C-21a) indicate that the annual average $PM_{10}$ concentrations corresponding to this scenario are higher in some areas and lower in other areas than the base-year (1990) values. The ratios generally range from approximately 0.95 to 1.1, but also include some higher values. In contrast, the ratios corresponding to the 2000 Post-CAAA simulation (Figure C-21b) are generally less than or equal to one, with most sites being assigned a ratio consistent with a small decrease in annual average $PM_{10}$ concentration. There are also some lower values.

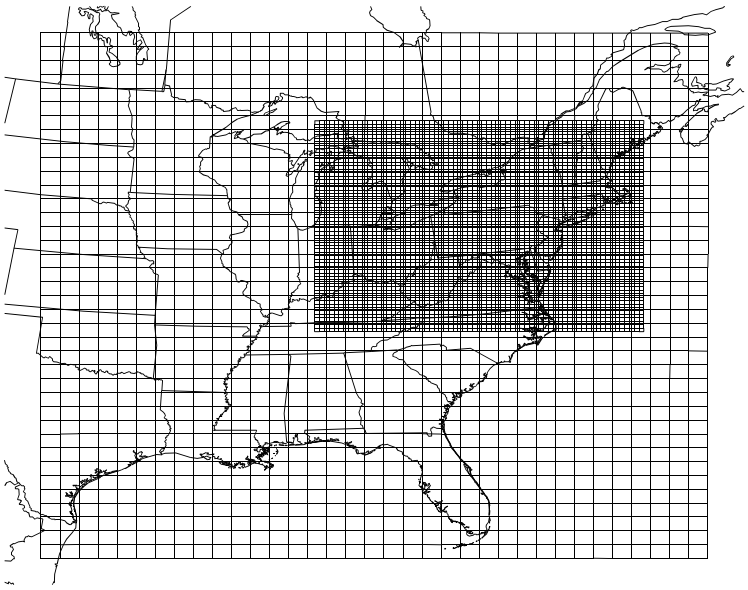
Figure C-22a and C-22b display the distribution of ratios of the future-year-scenario to base-year annual average $PM_{10}$ concentrations for 2010. Compared to the histogram plots for 2000, the ratios are higher for the Pre-CAAA scenario but similar for the Post-CAAA scenario. There is some indication that, by 2010, increases due to growth are limiting the effectiveness of the CAAA measures.

The 2000 Pre-CAAA ratios for $PM_{2.5}$ (Figure C-23a) indicate that the annual average $PM_{2.5}$ concentrations corresponding to this scenario are generally higher than (or equal to ) the base-year (1990) values. The ratios generally range from approximately 0.975 to 1.15. In contrast, the ratios corresponding to the 2000 Post-CAAA simulation (Figure C-23b) are generally less than one. In this case, the ratios range from approximately 0.925 to 1.075.

For 2010, the $PM_{2.5}$ ratios (Figures C-24a and C-24b), indicate increases for the Pre-CAAA scenario and mostly decreases for the Post-CAAA scenario. Again, compared to 2000, concentrations for 2010 are higher relative to the base year under the Pre-CAAA scenario and similar to or slightly lower relative to the base year under the Post-CAAA scenario.

For both future years (2000 and 2010), the ratios indicate that the Post-CAAA concentrations (annual average) are lower than the corresponding Pre-CAAA values. This is illustrated in Figures C-25a through C-26b. The smaller ratios for 2010 reflect larger differences between the Pre- and Post-CAAA scenarios.

**Figure C-14**
**80-km RADM Domain**



Note: Nested 20-km grid estimates were not used to generate final results, but were used in evaluating the reasonableness of results.

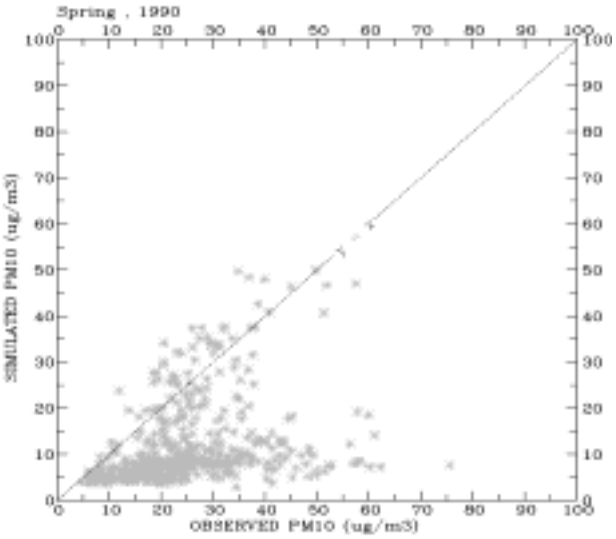



Figure C-15. Comparison of simulated and observed seasonal PM10 concentration (ug/m3) for REMSAD for the western U.S.: spring 1990

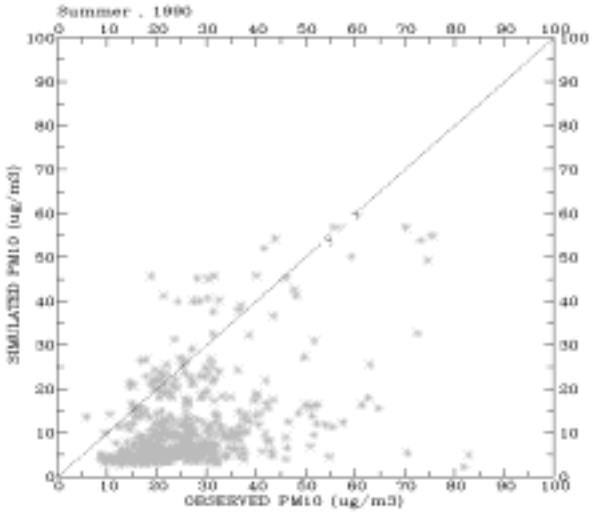



Figure C-16. Comparison of simulated and observed seasonal PM10 concentration (ug/m3) for REMSAD for the western U.S.: summer 1990

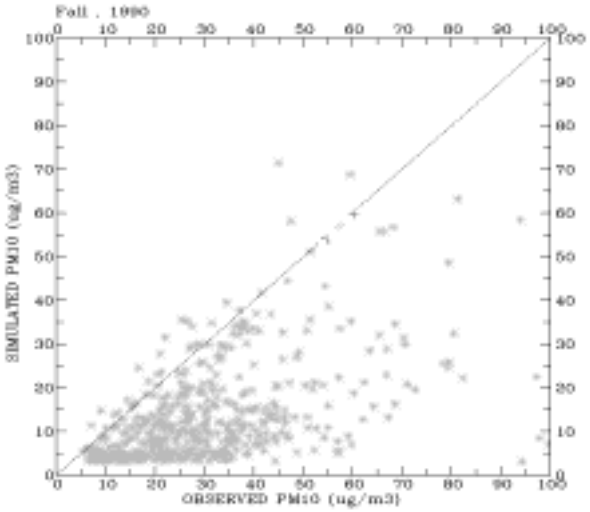



Figure C-17. Comparison of simulated and observed seasonal PM10 concentration (ug/m3) for REMSAD for the western U.S.: fall 1990

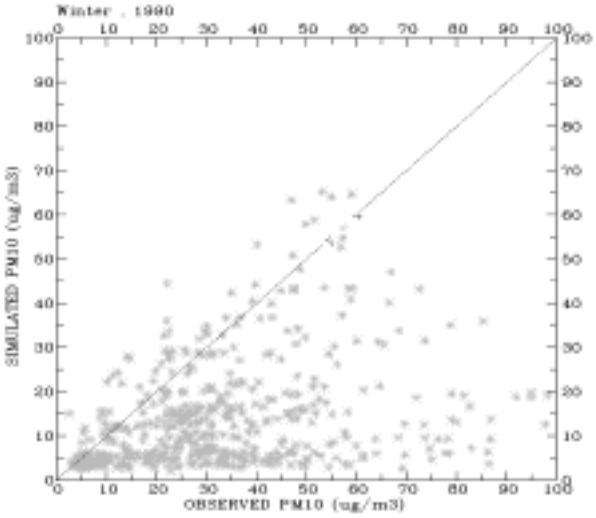



Figure C-18. Comparison of simulated and observed seasonal PM10 concentration (ug/m3) for REMSAD for the western U.S.: winter 1990

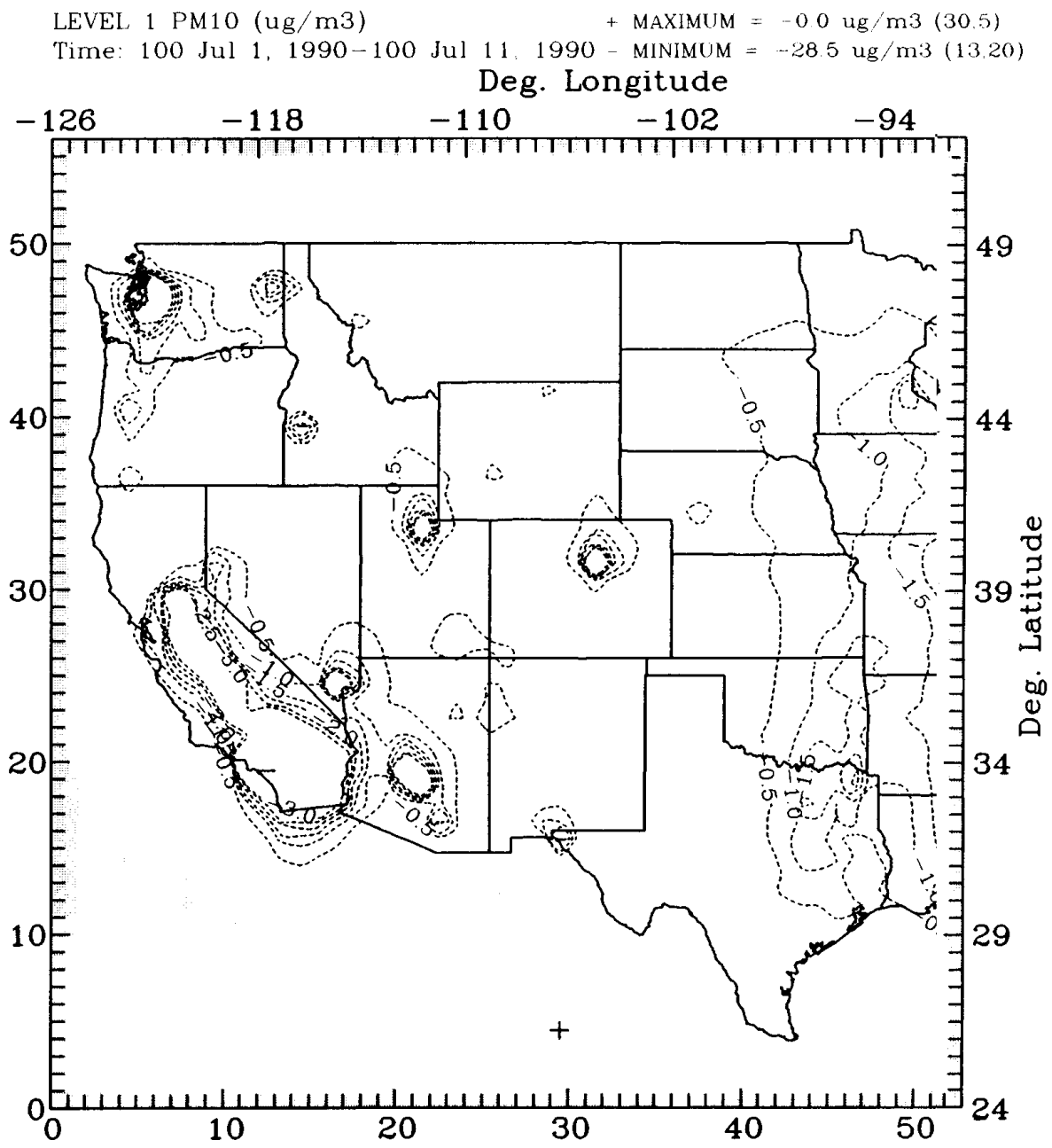



Figure C-19. Difference in seasonal average PM10 concentration (ug/m3) for the summer REMSAD simulation period (1-10 July 1990) for 2010: post-CAAA90 minus pre-CAAA90

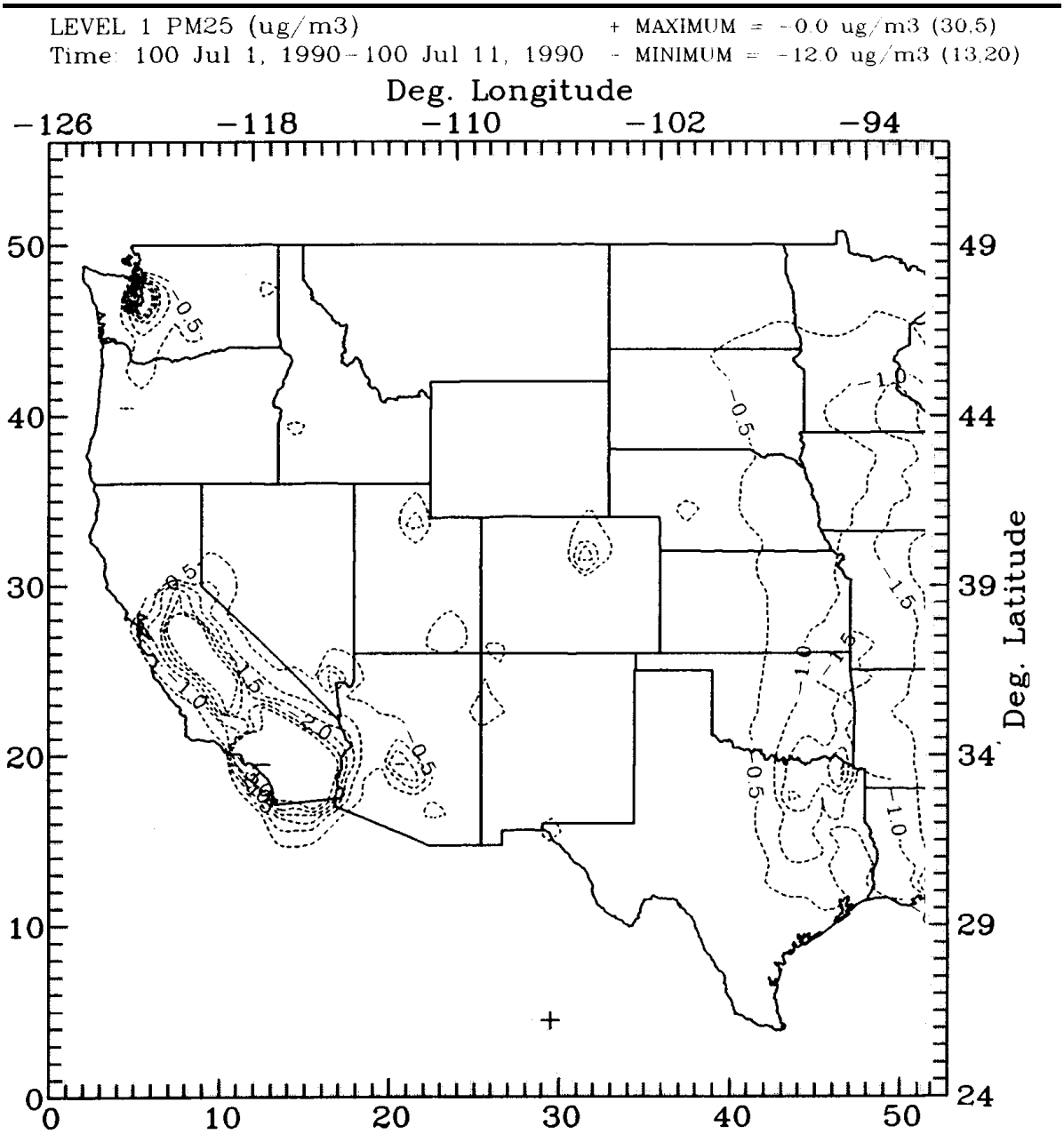



Figure C-20. Difference in seasonal average PM25 concentration (ug/m3) for the summer REMSAD simulation period (1-10 July 1990) for 2010: post-CAAA90 minus pre-CAAA90

**Figure C-21a. Distribution of Combined RADM/RPM- and REMSAD-Derived Monitor-Level Ratios for Annual Average $PM_{10}$ Concentration: 2000 Pre-CAAA / 1990 Base-Year**

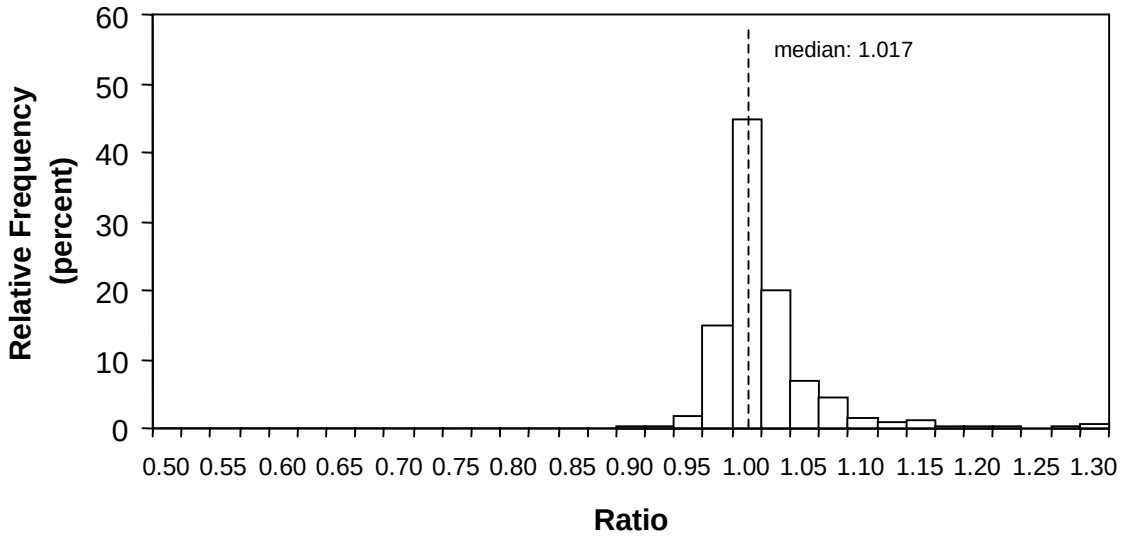



**Figure C-21b. Distribution of Combined RADM/RPM- and REMSAD-Derived Monitor-Level Ratios for Annual Average $PM_{10}$ Concentration: 2000 Post-CAAA / 1990 Base-Year**

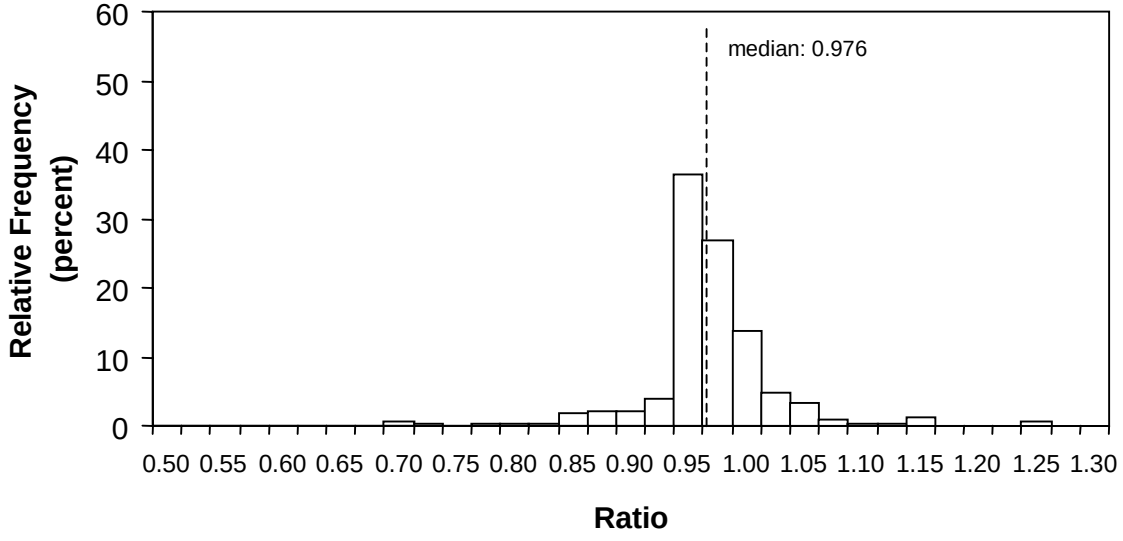


**Figure C-22a. Distribution of Combined RADM/RPM- and REMSAD-Derived Monitor-Level Ratios for Annual Average $PM_{10}$ Concentration: 2010 Pre-CAAA / 1990 Base-Year**

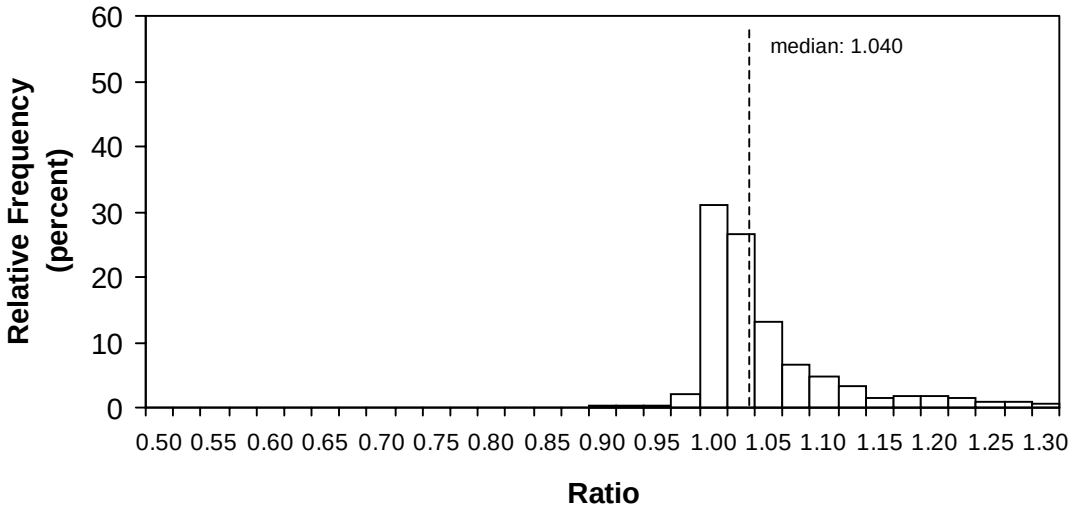



**Figure C-22b. Distribution of Combined RADM/RPM- and REMSAD-Derived Monitor-Level Ratios for Annual Average $PM_{10}$ Concentration: 2010 Post-CAAA / 1990 Base-Year**

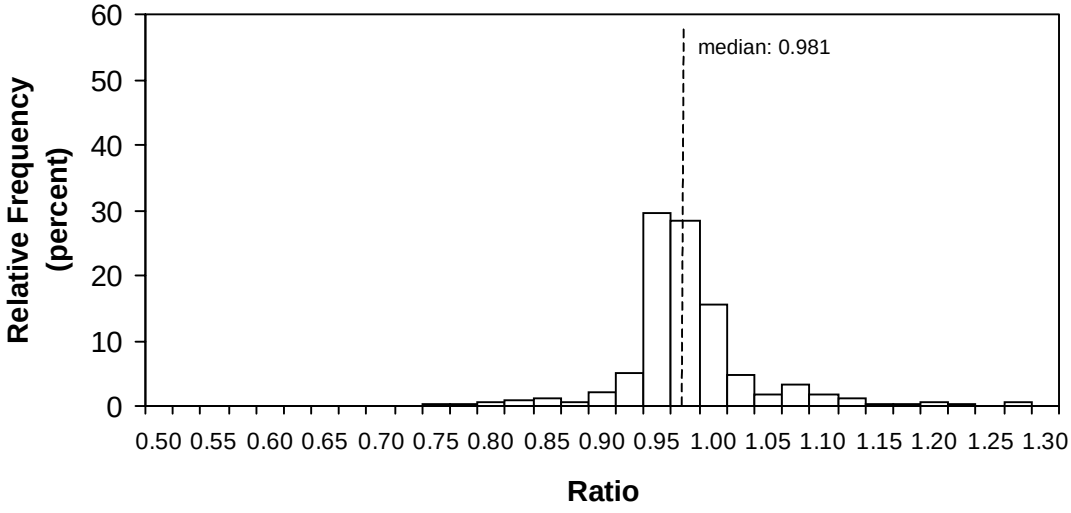


**Figure C-23a. Distribution of Combined RADM/RPM- and REMSAD-Derived Monitor-Level Ratios for Annual Average $PM_{2.5}$ Concentration: 2000 Pre-CAAA / 1990 Base-Year**

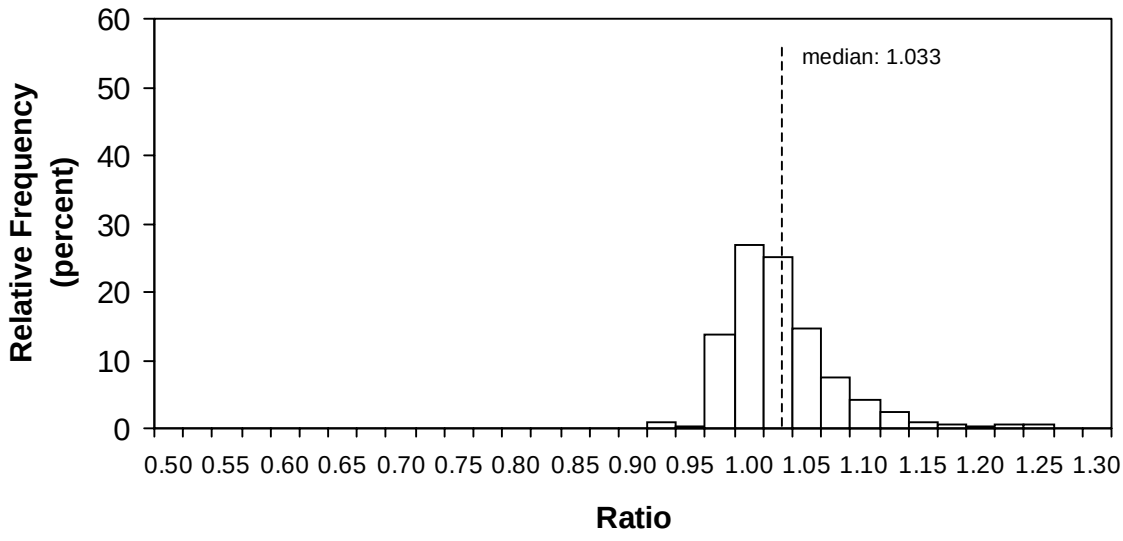



**Figure C-23b. Distribution of Combined RADM/RPM- and REMSAD-Derived Monitor-Level Ratios for Annual Average $PM_{2.5}$ Concentration: 2000 Post-CAAA / 1990 Base-Year**

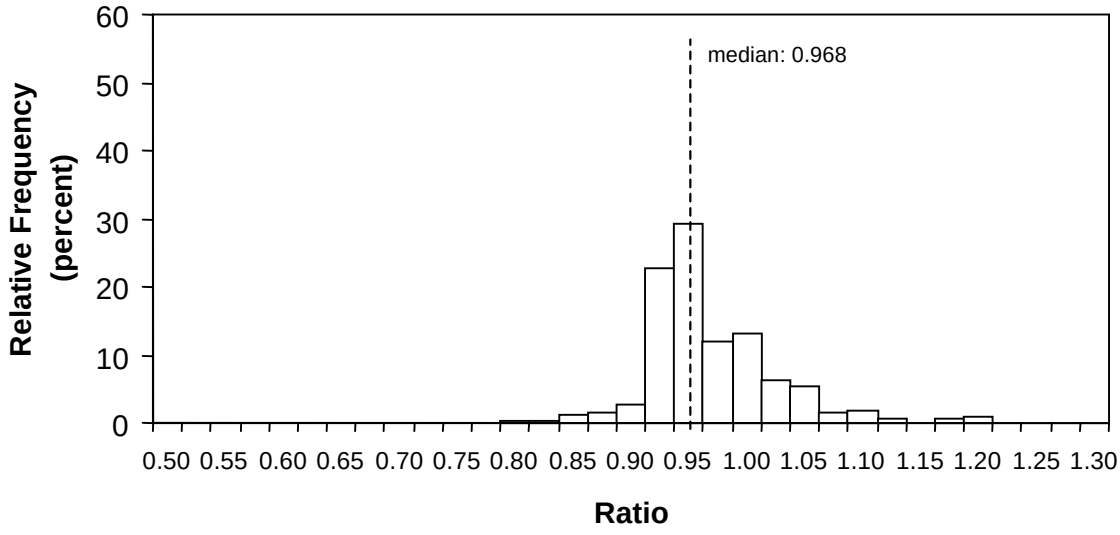


**Figure C-24a. Distribution of Combined RADM/RPM- and REMSAD-Derived Monitor-Level Ratios for Annual Average $PM_{2.5}$ Concentration: 2010 Pre-CAAA / 1990 Base-Year**

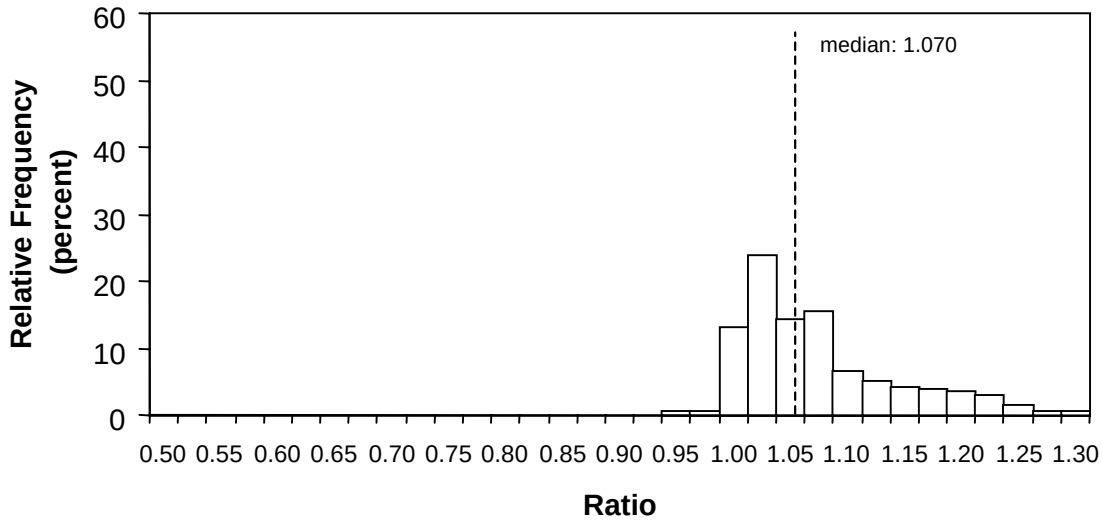



**Figure C-24b. Distribution of Combined RADM/RPM- and REMSAD-Derived Monitor-Level Ratios for Annual Average $PM_{2.5}$ Concentration: 2010 Post-CAAA / 1990 Base-Year**

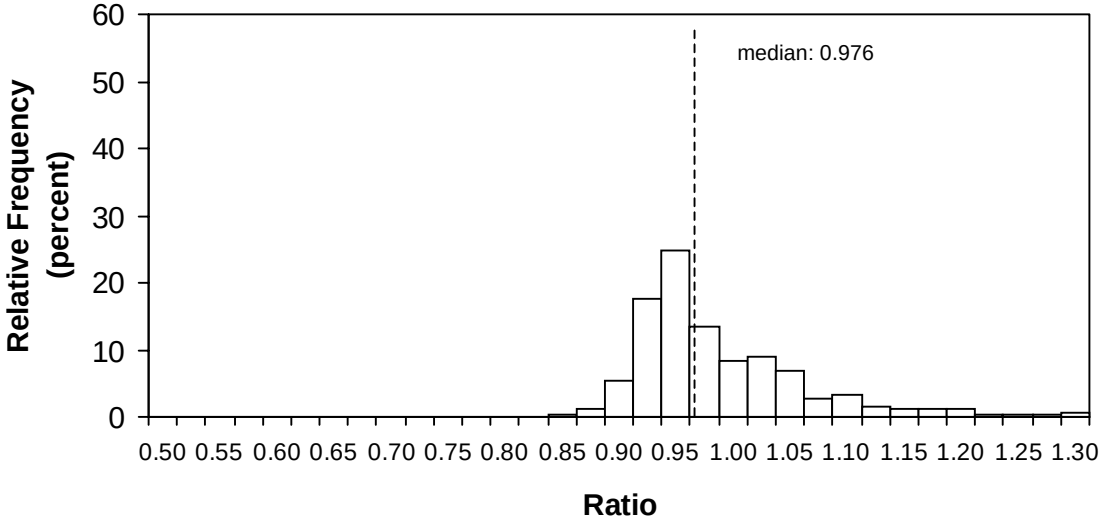


**Figure C-25a. Distribution of Combined RADM/RPM- and REMSAD-Derived Monitor-Level Ratios for Annual Average $PM_{10}$ Concentration: 2000 Post-CAAA / 2000 Pre-CAAA**

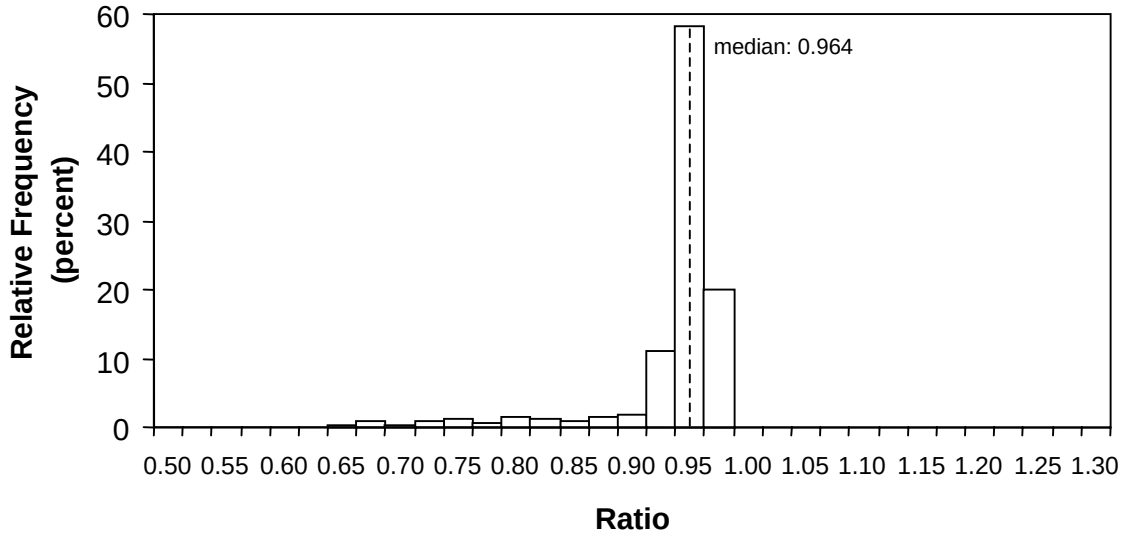



**Figure C-25b. Distribution of Combined RADM/RPM- and REMSAD-Derived Monitor-Level Ratios for Annual Average $PM_{10}$ Concentration: 2010 Post-CAAA / 2010 Pre-CAAA**

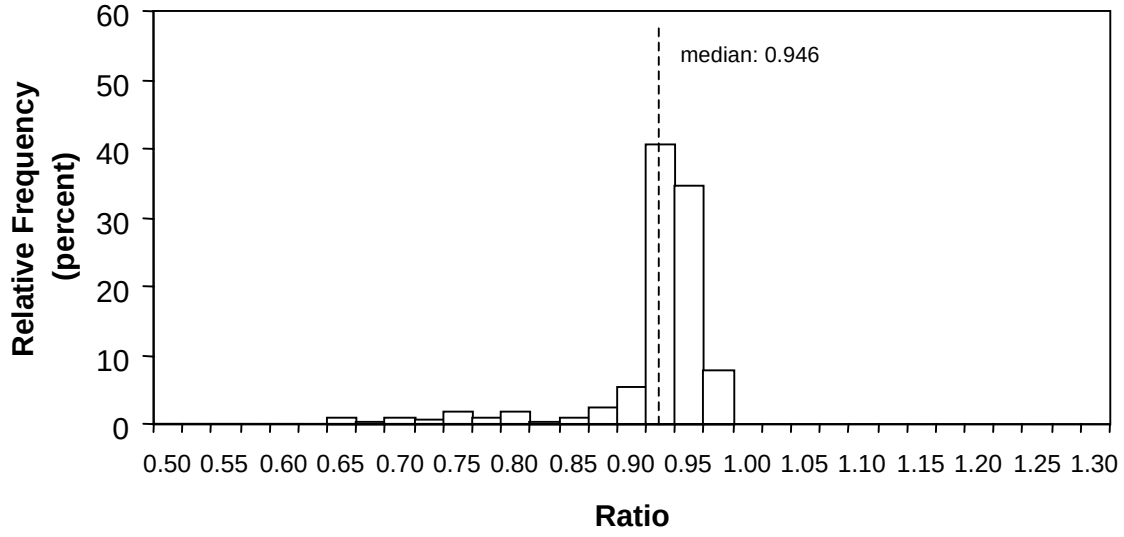


**Figure C-26a. Distribution of Combined RADM/RPM- and REMSAD-Derived Monitor-Level Ratios for Annual Average $PM_{2.5}$ Concentration: 2000 Post-CAAA / 2000 Pre-CAAA**

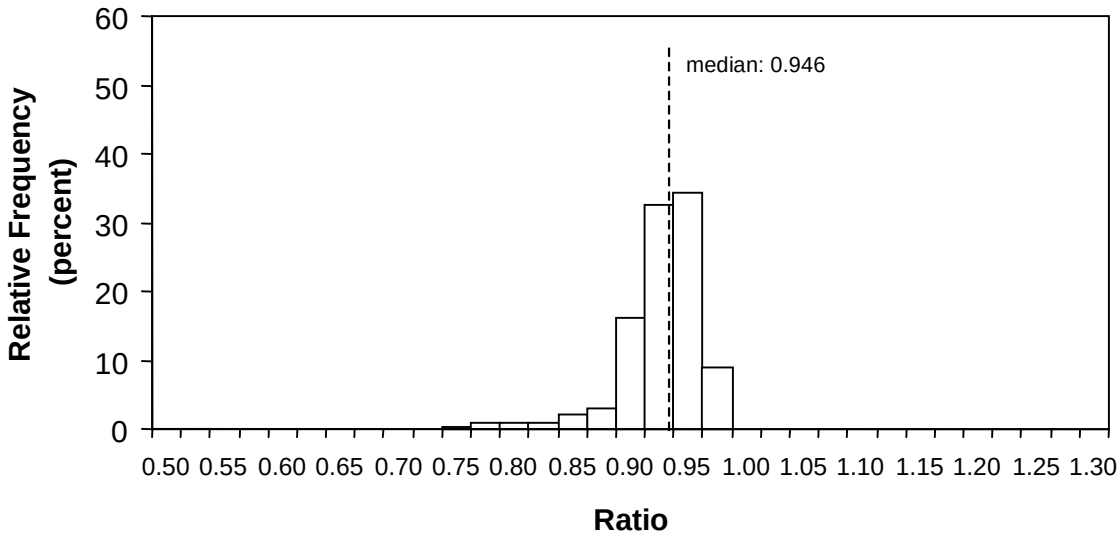
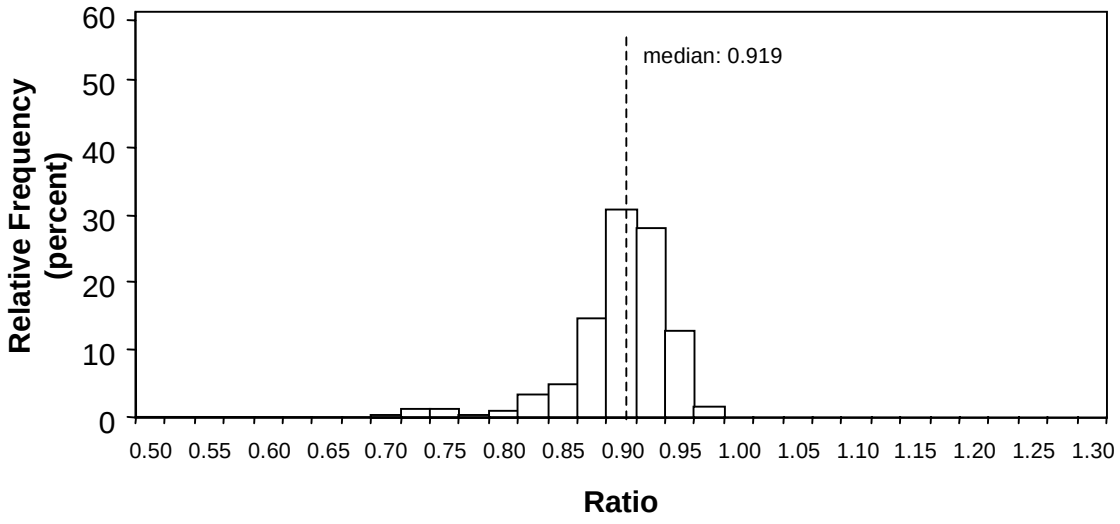



**Figure C-26b. Description of Combined RADM/RPM- and REMSAD-Derived Monitor-Level Ratios for Annual Average $PM_{2.5}$ Concentration: 2010 Post-CAAA / 2010 Pre-CAAA**



## Estimating the Effects of the CAAA on Visibility

Light traveling through the atmosphere is "absorbed" and "scattered" by gases and suspended particles. These distortion processes contribute to total atmospheric light extinction which, in turn, causes visibility degradation. To characterize and ultimately quantify the effect of changes in emissions on visibility, an understanding of the concentrations and types of gaseous particulate constituents in the air is necessary.

The influence of gaseous absorption on light extinction is almost negligible. Gaseous scattering has a larger impact, although this impact is generally not as significant as either particulate absorption or scattering. Together the influence of all four of these light distortion processes is expressed quantitatively as the light extinction coefficient, $b_{ext}$. In this analysis RADM/RPM and REMSAD are both used to calculate $b_{ext}$.

## RADM/RPM and Visibility

RADM/RPM estimates $b_{ext}$ in the eastern U.S., for each emissions scenario (1990 base year, 2000 Pre-CAAA, 2000 Post-CAAA, 2010 Pre-CAAA, and 2010 Post-CAAA), by combining the influences of particle scattering and absorption and incorporating the effect of scattering caused by water. The fine particles estimated by RADM/RPM (including their associated water) are secondary particulates: sulfates, nitrates, associated ammonium, and organics. Absorption by carbon particles is not included in the model's calculations, nor is extinction resulting from primary particles. By not including these latter influences, RADM/RPM may underestimate the effects of air pollution on visibility.

RADM/RPM, along with generating atmospheric light extinction values, calculates "visual range" and deciview (dV), both measures that quantify visibility. The former, VR, is related to the light extinction coefficient by the following equation:

$$VR(\text{meters}) = 3.912/b_{ext} ,$$

where $b_{ext}$ is in inverse meters. The latter measure of visibility, dV, and the related DeciView Haze Index are improved indicators of the clarity of the atmosphere. This index more accurately captures the relationship between air pollution and human's perception of visibility than does VR or $b_{ext}$ (Pitchford and Malm, 1994). A deciview is defined by the equation:

$$dV = 10\ln (b_{ext}/10) ,$$

where $b_{ext}$ is expressed in inverse megameters.

The DeciView Haze Index has a value of approximately zero when the light extinction coefficient is equal to the scattering coefficient for particle-free air. A roughly 10 percent increase in $b_{ext}$ translates to a one unit change in dV. Since the apparent change in visibility is related to a percent change in $b_{ext}$, equal changes in dV correspond to approximately equally perceptible changes in visibility. 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. An increase in the deciview level translates to degradation of visibility, while a decrease represents and improvement.

### *RADM/RPM Modeling Results*

For this analysis, under the 1990 base year and future year emissions scenarios, the annual mean daylight hour $b_{ext}$, VR, and dV were estimated for each RADM/RPM grid cell. A summary of 1990 and 2010 deciview levels for selected cities, metropolitan areas, and national parks is provided in Table C-13. These deciview estimates show that under the Pre-CAAA scenario visibility degradation is expected throughout much of the eastern U.S. Comparison of 1990 base year and 2010 Post-CAAA estimates, however, indicates that with the implementation of CAAA related measures, a perceptible improvement in visibility can be expected.

**Table C-13**
**Comparison of Visibility in Selected Eastern Cities, Metropolitan Areas, and National Parks**

| | | Mean Annual Deciview | | |
|---|---|---|---|---|
| **Area Name** | **State** | **1990 Base Year** | **2010 Pre-CAAA** | **2010 Post-CAAA** |
| Acadia NP | ME | 11.1 | 12.0 | 10.4 |
| Atlanta Metro Area | GA | 20.9 | 22.8 | 20.0 |
| Boston Metro Area | MA | 13.2 | 14.0 | 11.9 |
| Chicago Metro Area | IL | 17.5 | 19.1 | 17.0 |
| Columbus | OH | 16.5 | 17.7 | 15.1 |
| Detroit Metro Area | MI | 16.0 | 18.5 | 15.3 |
| Everglades NP | FL | 7.6 | 9.2 | 6.9 |
| Great Smoky Mtns. NP | TN | 20.4 | 22.3 | 19.6 |
| Indianapolis | IN | 20.1 | 21.1 | 19.0 |
| Little Rock | AR | 15.0 | 17.2 | 15.1 |
| Milwaukee Metro Area | WI | 15.6 | 18.4 | 15.3 |
| Minn.-St. Paul Metro Area | MN | 10.1 | 12.4 | 10.3 |
| Nashville | TN | 20.4 | 21.5 | 19.0 |
| New York City Metro Area | NY/NJ | 15.2 | 18.0 | 13.9 |
| Pittsburgh Metro Area | PA | 15.8 | 16.9 | 14.2 |
| St. Louis Metro Area | MO | 16.5 | 17.8 | 16.0 |
| Shenandoah NP | VA | 16.5 | 17.8 | 15.2 |
| Syracuse | NY | 12.4 | 13.2 | 11.5 |
| Washington, DC Metro Area | DC/VA/MD | 17.5 | 19.2 | 16.3 |

*For cities, metro areas, or national parks not contained by a single RADM/RPM grid cell, the visibility measure presented in this table is a weighted average of the mean annual deciview level from each of the grid cells that together completely contain the selected area. Weighting is based upon the spatial distribution of an area over the various grid cells.

## REMSAD and Visibility

REMSAD was used to estimate the effect of changes in emissions on visibility for the western U.S. This model calculates light extinction coefficients based upon estimates of the gridded ground-level concentrations of the following species – sulfate (NH4S+GSO4+ASO4), nitrate (NH4N+PNO3), NO2, SOA, POA, PEC, PMfine and PMcoarse (refer to Table C-8 for a description of these species abbreviations). The contribution from each of these species is adjusted based on the extinction efficiency of each and, in the case of sulfate, nitrate and SOA, an adjustment dependent on the relative humidity. The total extinction coefficient is then given by:

$$b_{ext} = 10.+ 0.17*NO2 + f_{so4}(RH)*sulfate + f_{no3}(RH)*Nitrate + f_{soa}(RH)*SOA +6.2*POA + 10.5*PEC + PMFINE + 0.6*PMCOARSE$$

where the constant value of 10.0 is the contribution to the scattering coefficient for particle-free air (Rayleigh scattering). REMSAD generated $b_{ext}$ values are then converted to deciviews.

### *REMSAD Modeling Results*

Visibility estimates and change in visibility were calculated for each of the future-year scenarios for use in the effects analysis. Figure C-27 illustrates 1990 base year deciview levels for the western U.S. This map shows that visibility is poorer in the region of California extending from San Francisco southward to Los Angeles, the Pacific Northwest, and larger metropolitan areas such as Denver, CO; Albuquerque, NM; and Phoenix, AZ. Most noticeable is the comparatively high deciview level in the Los Angeles region.

Figures C-28 and C-29 illustrate the difference between 2010 Pre-CAAA and 1990 base year estimates and the difference between 2010 Post-CAAA and 1990 base year estimates, respectively. The first of these maps shows that under the Pre-CAAA scenario visibility is expected to remain unchanged between 1990 and 2010 throughout much of the West and actually improve in coastal Oregon and along the western Idaho border. In the larger urban areas, however, perceptible visibility degradation is predicted. Visibility improvement in and around western cities, especially in California, is predicted under the Post-CAAA scenario. Figure C-29 captures these changes and shows that in 2010 improvements in visibility are not expected to be restricted to just the larger urban areas; compared to 1990 base year estimates, Post-CAAA deciview levels are also predicted to be lower throughout much of Washington, Oregon, and Nevada and in sizeable sections of Arizona, Idaho, Utah, and Wyoming.

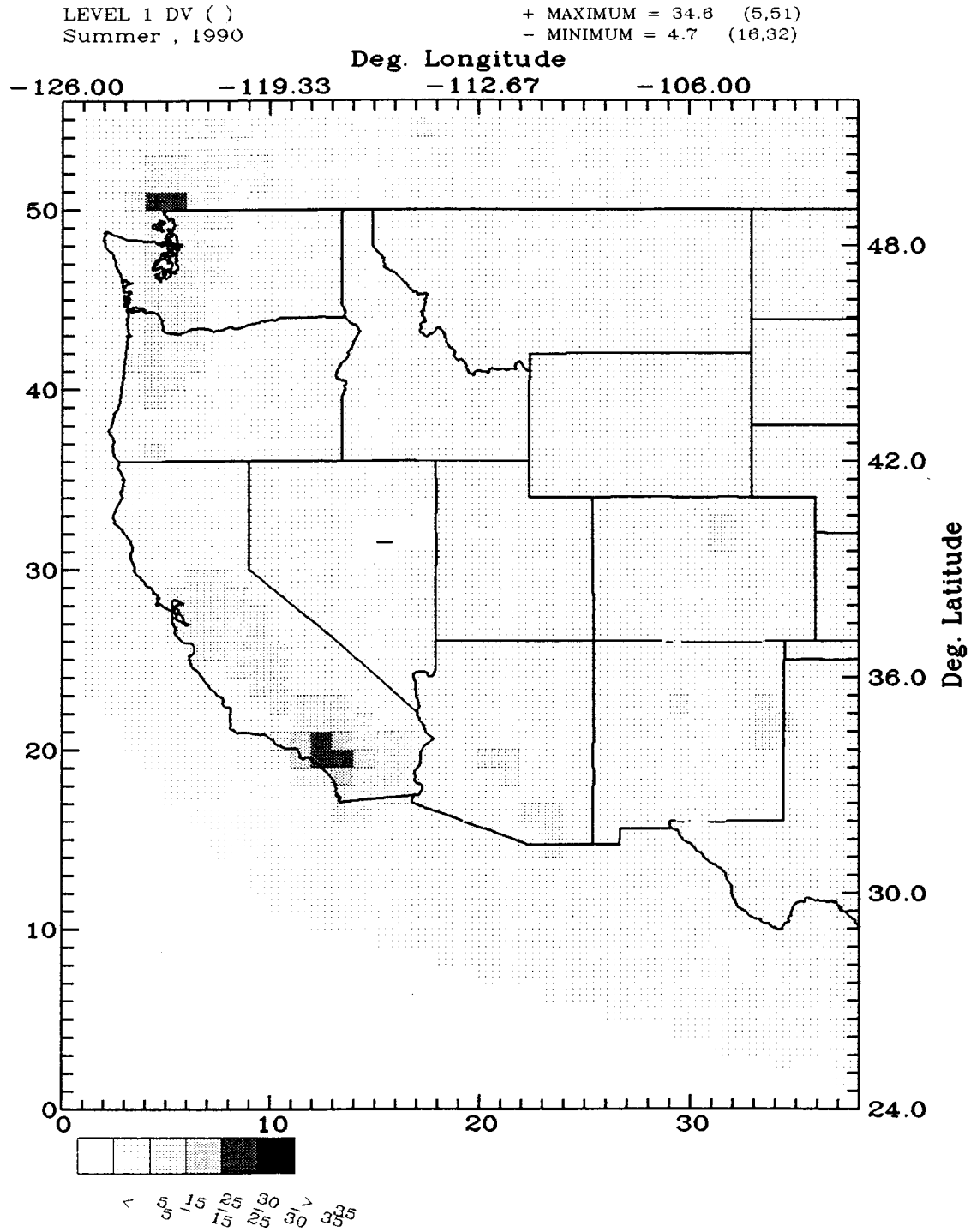



Figure C-27. Seasonal Average Deciview for the summer REMSAD simulation period (1-10 July 1990): base 1990 (western U.S. only)

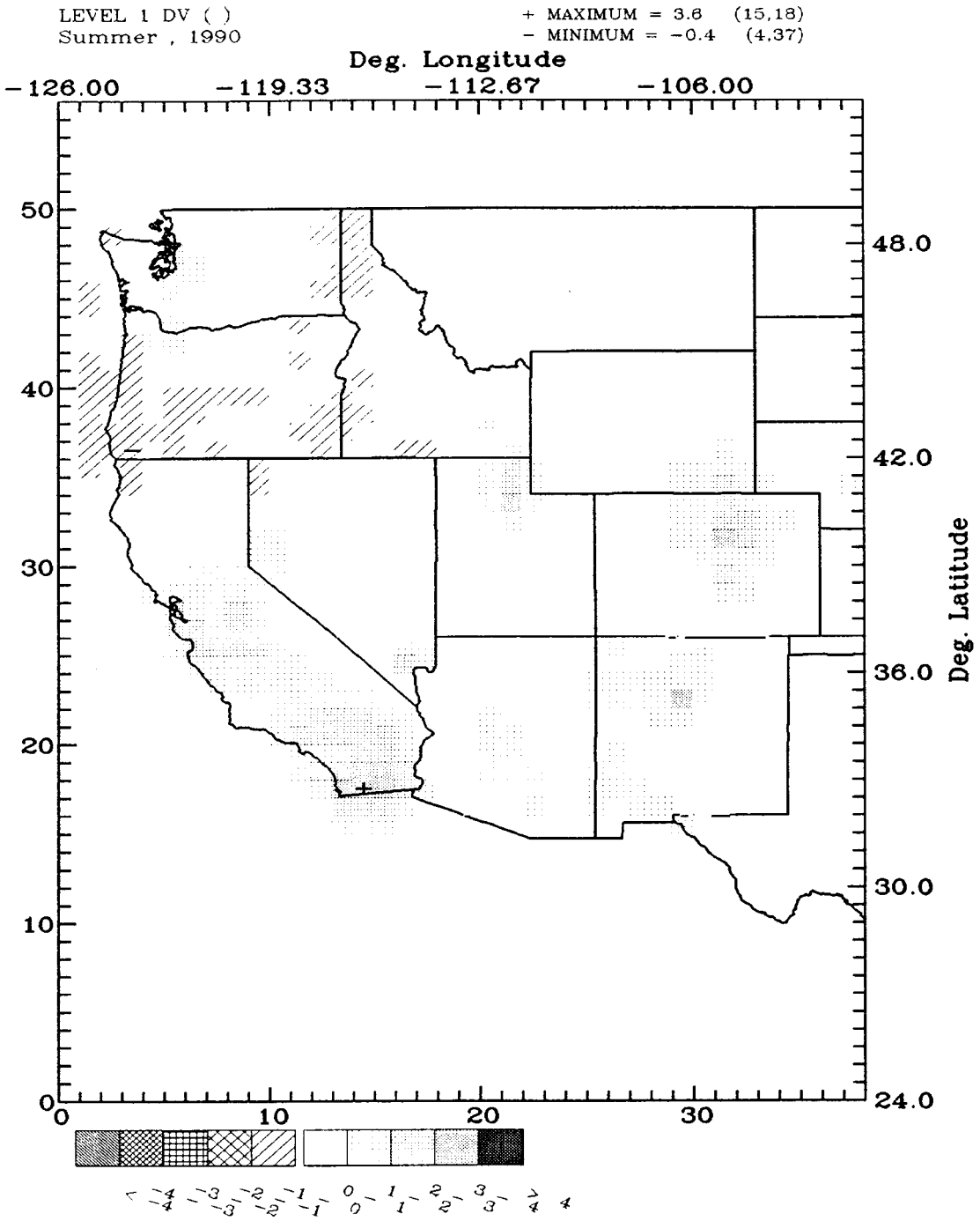



Figure C-28. Difference in seasonal average Deciview for the summer REMSAD simulation period (1-10 July 1990): 2010 pre-CAAA90 minus base 1990 (western United States only)

## Acid Deposition

The acid deposition modeling efforts for this analysis focused on estimating the change in ambient concentrations of selected pollutants as a result of changes in emissions. The need to focus on relative changes, rather than absolute predictions, is especially acute when 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 U.S. EPA's National Exposure Research Laboratory:

> *"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 compounds (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 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."*[10]

[10] Dennis, R. RADM Report (1995), p. 1.

The complexity and nonlinearity of the relationships between localized emissions of precursors, such as $SO_2$ and VOCs, and subsequent regional scale air quality and deposition effects are so substantial that advanced modeling is required to accurately estimate the broad-scale impact of changes in emissions on acid deposition. For this analysis, EPA used the Regional Acid Deposition Model (RADM) to estimate acid deposition in the eastern United States.

### *Overview of the RADM Modeling System*

RADM, a three-dimensional Eulerian grid-based model also used in the PM analysis, estimated nitrogen and sulfur deposition for the 1990 base year and each of the future year emissions scenarios. Estimates, expressed in kg/ha, were developed for 2000 and 2010 and calculated for each 80-km RADM grid cell. It is important to note, however, that ammonia deposition, a significant contributor to total nitrogen deposition, was held constant for each of the model runs. This was because livestock farming and other activities that drive ammonia formation and deposition were essentially unaffected by the CAAA-related control programs. A more detailed description of RADM, its domain, and its inputs is provided earlier in this appendix..

### *RADM Modeling Results*

Figures C-30 and C-31 show the 1990 base-year deposition estimates for sulfur and nitrogen respectively. Predictions for both pollutants under the Pre- and Post-CAAA scenarios are displayed in Figures C-32 through C-35. Comparison of the three maps showing sulfur deposition and comparison of the three maps showing nitrogen deposition reveals that for both pollutants annual deposition under the Pre-CAAA scenario is expected to increase between 1990 and 2010. Year 2010 Post-CAAA sulfur and nitrogen deposition projections, however, are not only lower than 2010 Pre-CAAA projections, but also below 1990 base year levels. Together, these maps indicate that between 1990 and 2010 average annual

acid deposition is expected to decrease as a result of the Clean Air Act Amendments.

Noticeable in each of the figures, especially those mapping nitrogen, is an area of high deposition along the Virginia-North Carolina border. This "hot spot" is above Person County, NC, a region with one large and one very large utility plant.[11] Emissions from these plants, particularly $NO_x$, likely are the source of the high deposition in this area. Person County exhibits the highest base year and future year Pre- and Post-CAAA acid deposition estimates in the entire RADM domain.

Comparison of 2010 Pre- and Post-CAAA emissions in Person County shows that $NO_x$ emissions are expected to be lower in 2010 as a result of the CAAA. This change in emissions, however, translates to a change in acid deposition that is not captured by the maps provided in this section. 2010 Post-CAAA nitrogen and sulfur deposition estimates for this county are 27.2 and 78.0 kg/ha respectively. These figures represent a decrease in nitrogen deposition of 14.0 kg/ha and a decrease in sulfur deposition of 4.5 kg/ha from 2010 Pre-CAAA levels. Compared to the base year, the 2010 Post-CAAA nitrogen deposition estimate for Person County is 4.1 kg/ha lower than 1990 levels, the 2010 Post-CAAA sulfur deposition prediction, however, is 12.9 kg/ha higher.

[11] Under the 2010 Post-CAAA scenario the Mayo (large) and Roxboro (very large) utility plants are predicted to emit 9,400 and 30,100 tons of $NO_x$ per year respectively.

**Figure C-29**
**Annual Sulfur Deposition**
**1990 Base Case Scenario**

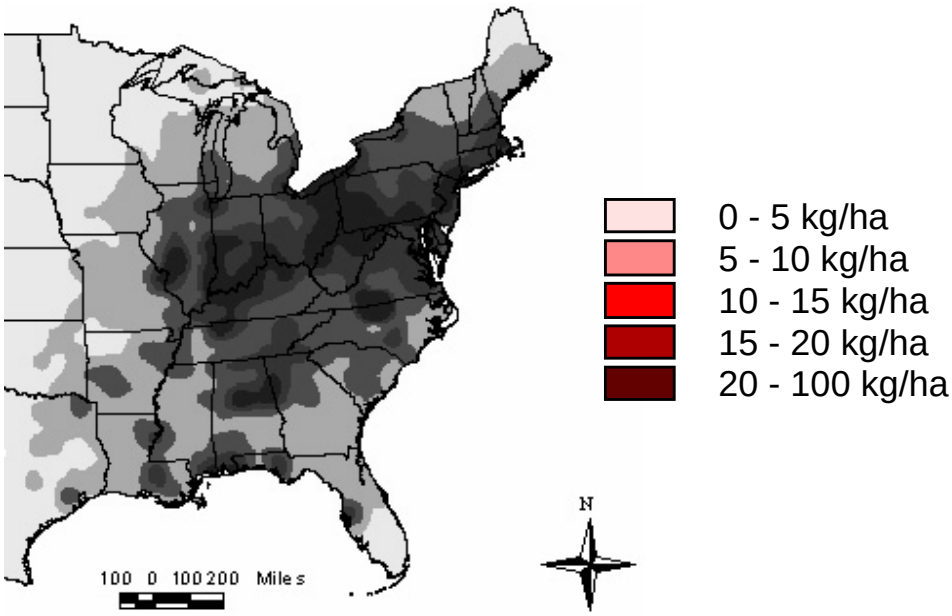


**Figure C-30**
**Annual Nitrogen Deposition**
**1990 Base Case Scenario**

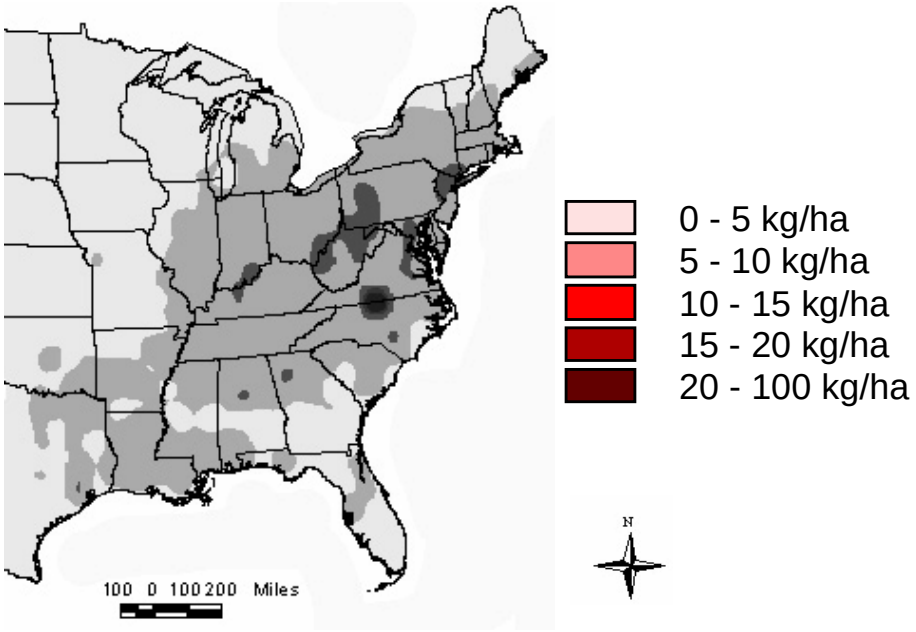


**Figure C-31**
**Annual Nitrogen Deposition**
**2000 Pre CAAA Scenario**

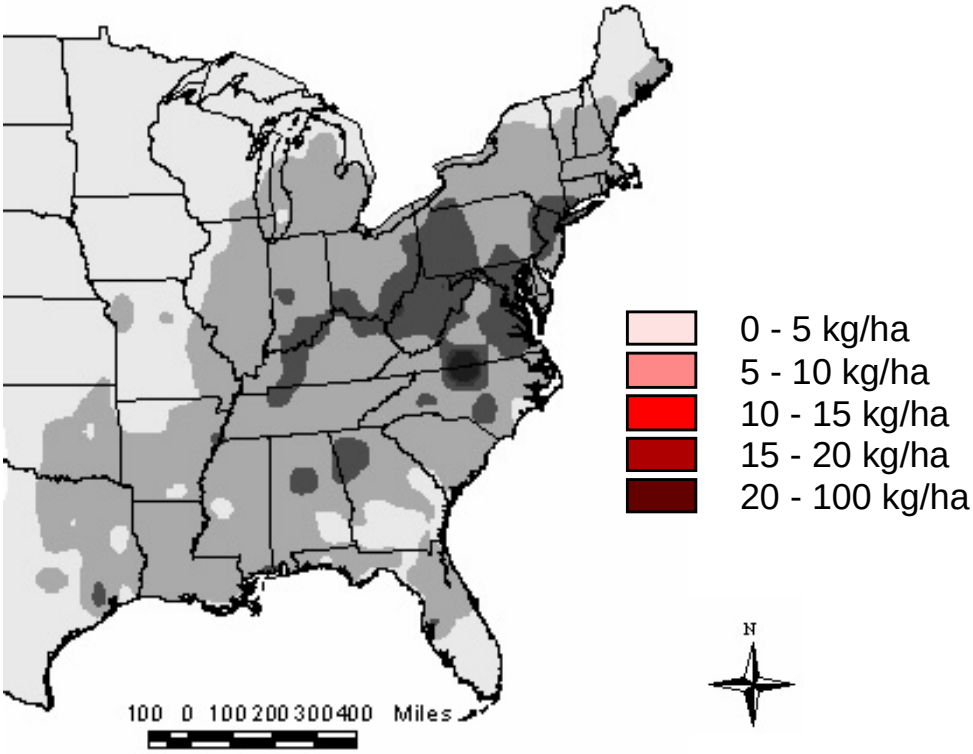


**Figure C-32**
**Annual Sulfur Deposition**
**2010 Post CAAA Scenario**

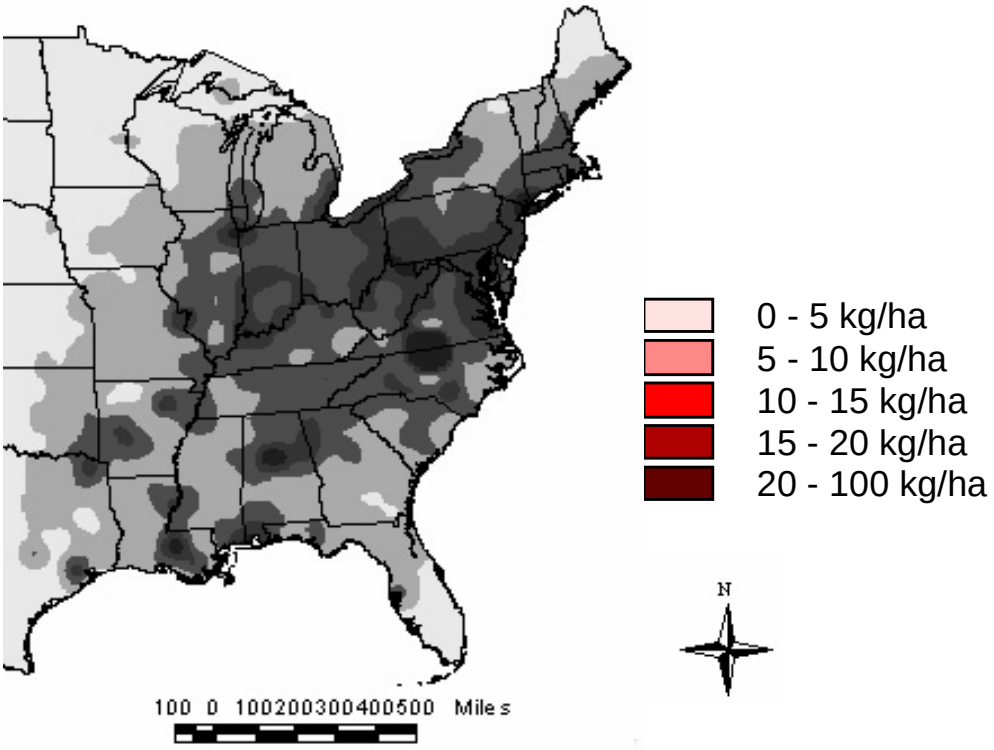


**Figure C-33**
**Annual Nitrogen Deposition**
**2010 Pre CAAA Scenario**

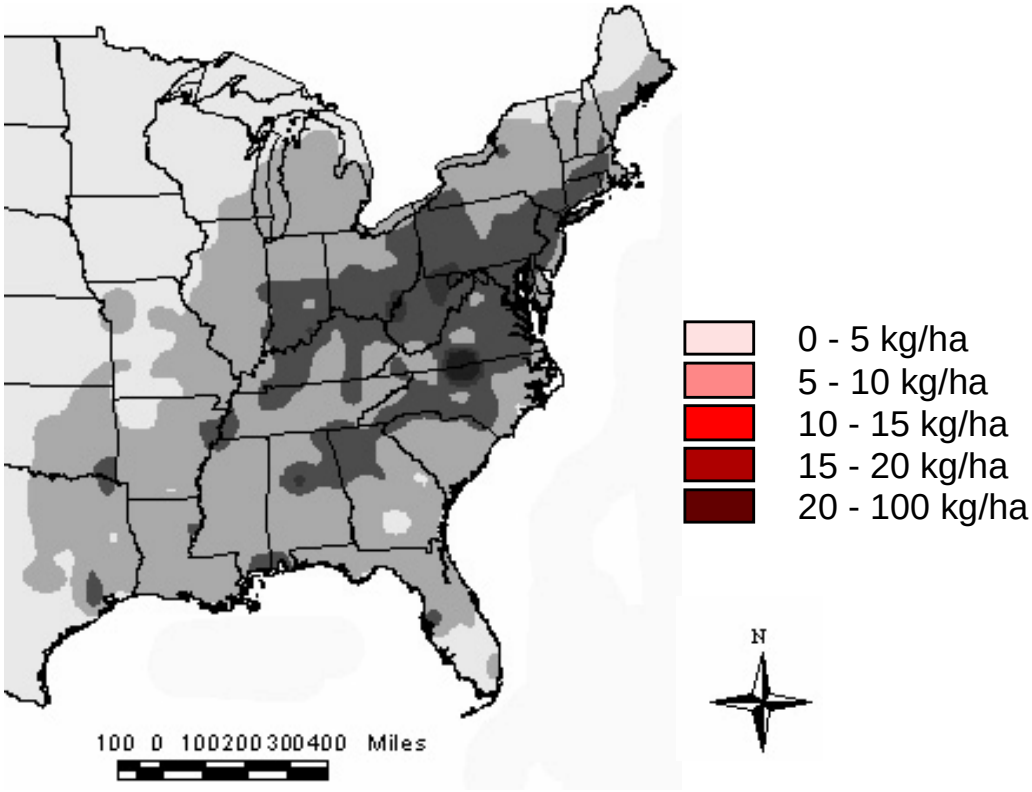
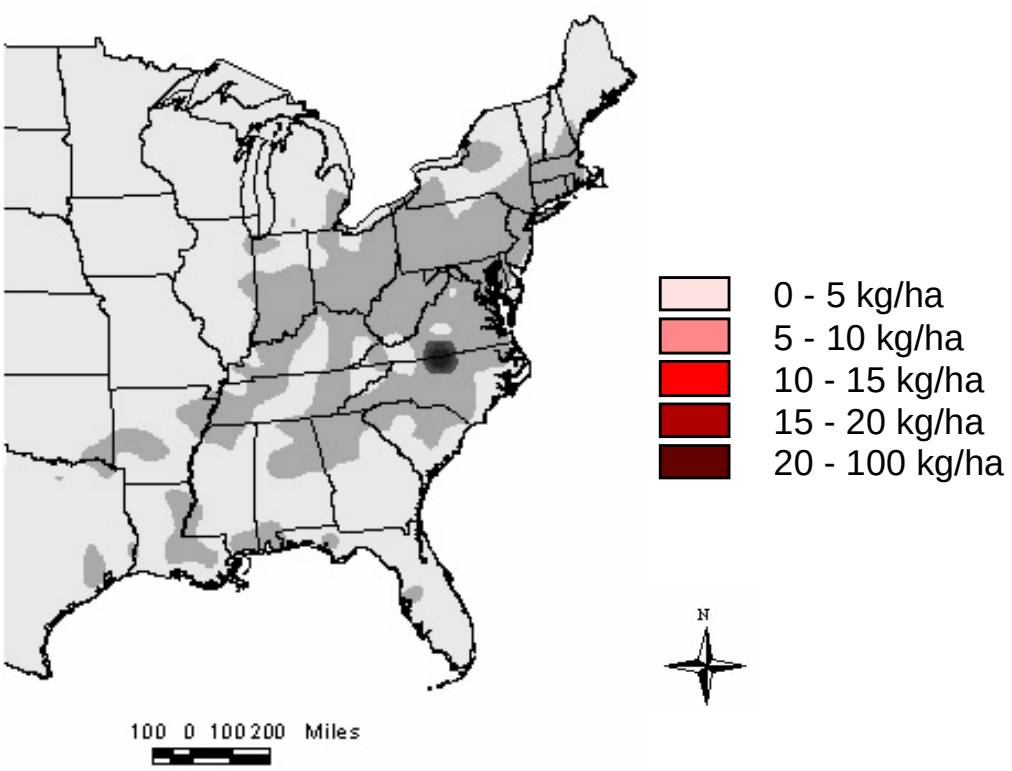


**Figure C-34**
**Annual Nitrogen Deposition**
**2010 Post CAAA Scenario**



## Estimating the Effects of the CAAA on Sulfur Dioxide, Oxides of Nitrogen, and Carbon Monoxide

Future-year Pre- and Post-CAAA ambient $SO_2$, NO, $NO_2$, and CO concentrations were estimated by adjusting 1990 concentrations using future-year to base-year emissions ratios. The methodology for calculating and applying these ratios is described below. The resulting future-year concentration also are discussed in this section; histograms are used to illustrate the relationship between Post- and Pre-CAAA emissions estimates.

### *Methodology for Estimating Future-Year $SO_2$, NO, $NO_2$, and CO Concentrations*

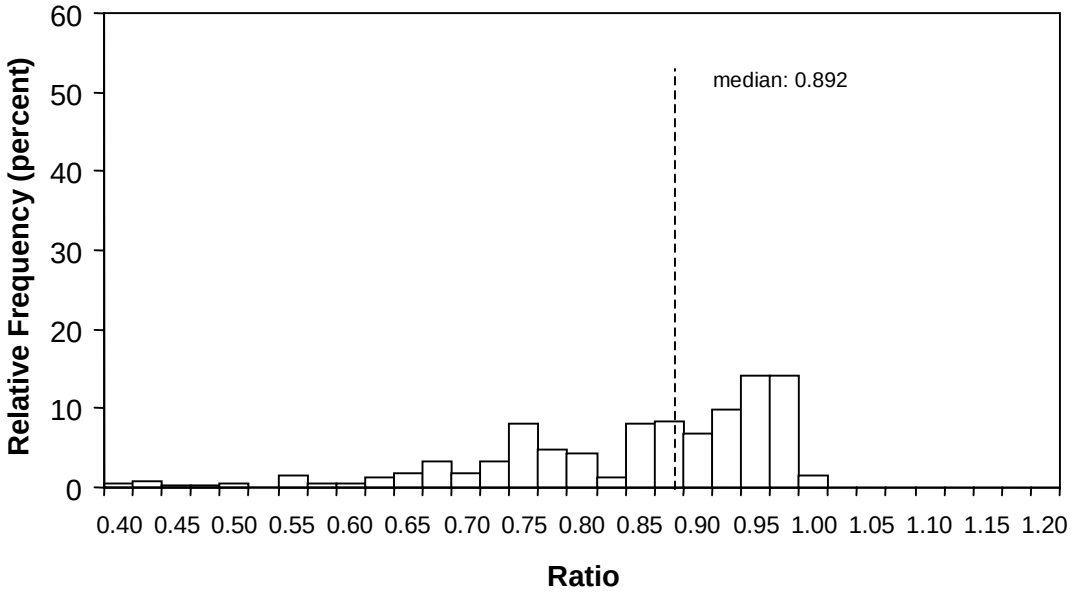
To estimate future-year $SO_2$, NO, $NO_2$, and CO concentrations, adjustment factors were calculated using grid cell specific REMSAD emissions data (Douglas et al., 1999). REMSAD's domain encompasses the 48 contiguous states and is divided into 4,950 grid cells, each measuring approximately 56 km by 56 km. As part of the model's input, gridded emission inventories containing seasonal Pre- and Post-CAAA $SO_2$, NO, $NO_2$, and CO emissions estimates were prepared. These same emissions estimates used as REMSAD input in other parts of this prospective analysis, were also used to calculate $SO_2$, NO, $NO_2$, and CO adjustment factors.

Before emission-based ratios (adjustment factors) were calculated, two separate inventories maintained individually for REMSAD modeling purposes, one containing elevated point source emissions data and the other containing emissions data for low-level sources, were combined. Each stack corresponding to an elevated point source was assigned to a grid cell based on location. Emissions from elevated point sources were then added to the low-level emissions corresponding to the grid cell in which the stack is located. In this manner, a file containing total emissions for each grid cell was prepared. This was done for each season for the 1990 base year and 2000 and 2010 Pre- and Post-CAAA scenarios.

Once the emissions inventory was prepared, emission-based ratios for $SO_2$, NO, $NO_2$, and CO were generated. For each RADM grid cell, adjustment factors were calculated comparing future-year (2000 and 2010) emissions under each projection scenarios to base-year (1990) emissions. Separate sets of ratios were developed for each season.

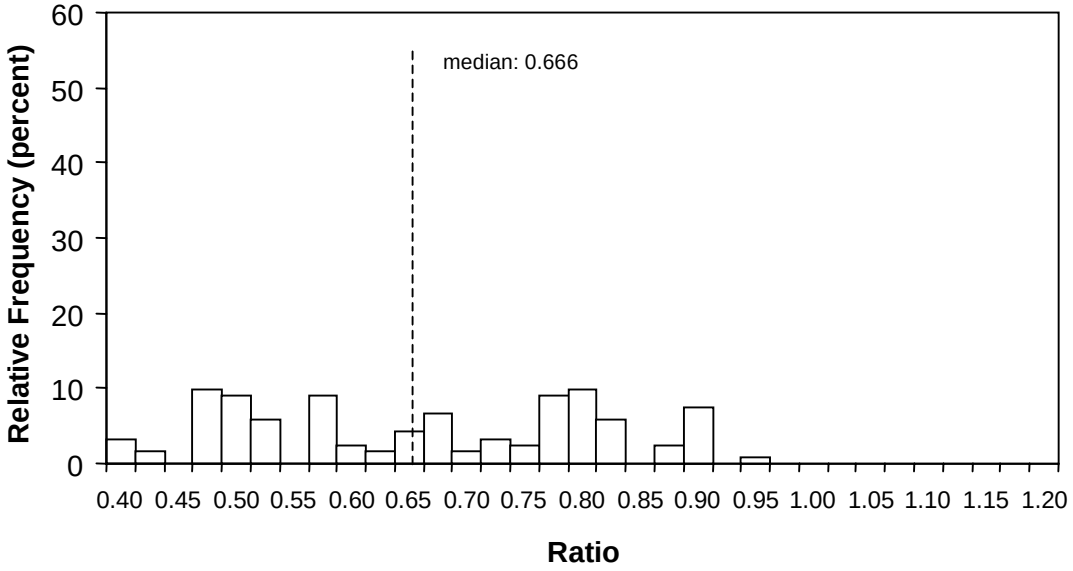
Following the calculation of emission-based ratios, future-year concentrations were then estimated by applying these ratios to observed 1990 base-year monitor concentrations. For REMSAD grid cells without 1990 monitor concentration data interpolation was used to estimate base-year concentrations. Adjustment factors for the grid cell were then applied to the interpolated values.

**Figure C-35. Distribution of Monitor-Level Ratios of Summer $SO_2$ Emissions: 2010 Post-CAAA / 2010 Pre-CAAA**



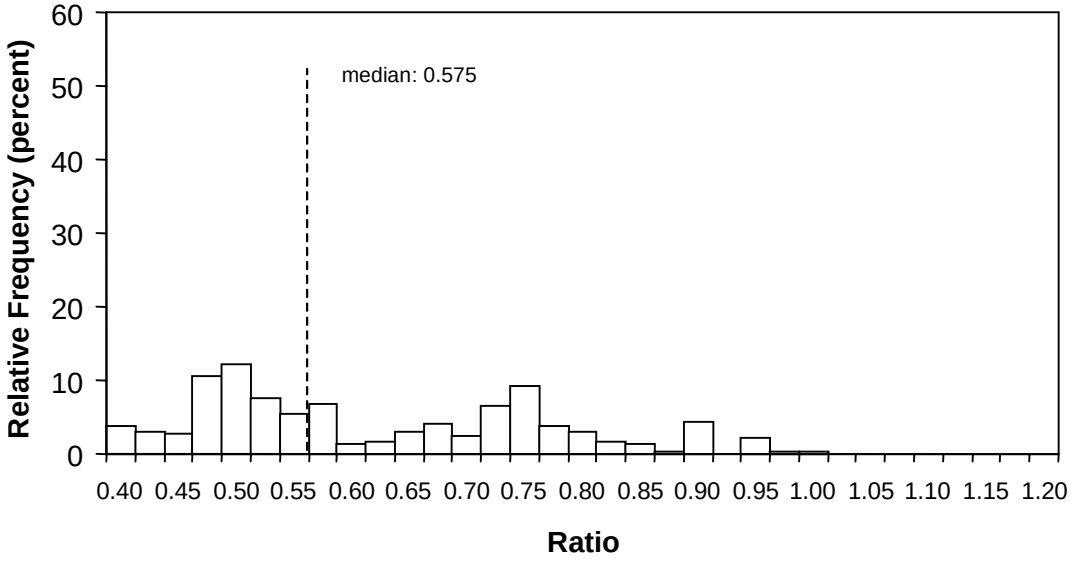

Note: 2.4 percent of the distribution of ratios is less than 0.40.

**Figure C-36. Distribution of Monitor-Level Ratios of Summer NO Emissions: 2010 Post-CAAA / 2010 Pre-CAAA**



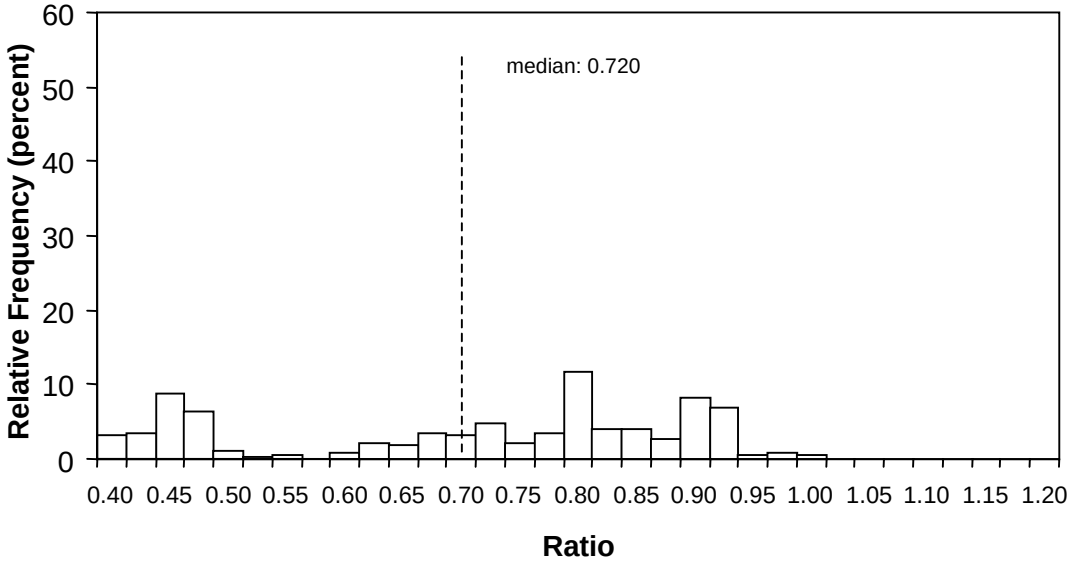

Note: 3.3 percent of the distribution of ratios is less than 0.40.

**Figure C-37. Distribution of Monitor-Level Ratios of Summer $NO_2$ Emissions: 2010 Post-CAAA / 2010 Pre-CAAA**




Note: 2.7 percent of the distribution of ratios is less than 0.40.

**Figure C-38. Distribution of Monitor-Level Ratios of Summer CO Emissions: 2010 Post-CAAA / 2010 Pre-CAAA**




Note: 15.7 percent of the distribution of ratios is less than 0.40.

## Emission-Based Ratios for $SO_2$, NO, $NO_2$, and CO

Emission-based ratios were calculated for each grid cell, regardless of whether or not the cell contained a monitoring site. The figures included in this section however, represent the distribution of ratios for actual monitoring site locations. These distributions reveal the relationship between future-year and base-year concentrations. A ratios greater that one indicates an increase in ambient concentration from the base-year, while a ratio less than one indicates a decrease.

Our results indicate that compared to the base-year, future-year concentrations of $SO_2$, NO, $NO_2$, and CO tend to increase under the Pre-CAAA scenario, while Post-CAAA concentrations for all four pollutants except $SO_2$ tend to decrease. For example, the median 2010 Pre-CAAA emission-based ratio for $SO_2$ is roughly 1.35, indicating an increase in median 2010 Pre-CAAA $SO_2$ concentration of approximately 35 percent from the 1990 base-year. The median ratios for NO, $NO_2$, and CO are roughly 1.13, 1.17, and 1.05 respectively. Under the Post-CAAA scenario we estimate that in 2010 NO, $NO_2$, and CO concentrations will tend to be approximately 25 and 30 percent below base-year levels. The median 2010 Post-CAAA emission-based ratios for these three pollutants are roughly 0.74, 0.70, and 0.76 respectively. We estimate that $SO_2$, concentrations, however, will increase in many areas of the U.S. The median adjustment ratio for this pollutant is approximately 1.21.

## Comparison of the Pre- and Post-CAAA Ratios

Comparison of Pre- and Post-CAAA emission-based adjustment factors also helps illustrate the effect of the 1990 Amendments on ambient pollution concentrations. The ratio of 2010 Post-CAAA adjustment factors to 2010 Pre-CAAA adjustment factors shows the impact of the 1990 Amendments on ambient concentrations relative to the baseline scenario. Ratios less than one indicate that we estimate that future-year concentrations of $SO_2$, NO, $NO_2$, and CO are lower under the Post-CAAA scenario than under the Pre-CAAA scenario.

Figures C-35 through C-38 show the distribution of 2010 Post-CAAA to 2010 Pre-CAAA ratios for summertime $SO_2$, NO, $NO_2$, and CO respectively. These figures illustrate the regional variation in the influence of the 1990 Amendments on ambient concentrations of these pollutants. Although we estimate concentrations in some areas will increase under the Post-CAAA scenario relative to Pre-CAAA estimates, the median summertime 2010 Post- to Pre-CAAA ratios for $SO_2$, NO, $NO_2$, and CO are 0.90, 0.67, 0.58, and 0.72 respectively. These values, each less than one, indicate that the central tendency for summertime 2010 Post-CAAA concentration estimates of these four pollutants is to be lower than 2010 Pre-CAAA estimates.

Table C-14 displays the median values of the distribution of Post- to Pre-CAAA ratios for the summer months described above and the remaining three seasons. Just as for the summer; spring, autumn, and winter median values are less than one. Averaged over all four seasons, we estimate a median reduction in $SO_2$, NO, $NO_2$, and CO concentrations of approximately 9, 33, 40, and 25 percent respectively. RACT requirements, tailpipe emissions standards, and $NO_x$ emissions trading account for the bulk of the reduction in NO and $NO_2$ concentrations. Title I nonattainment area controls and Title II motor vehicle provisions are responsible for much of the change in CO concentrations, while regulation of utility and motor vehicle emissions account for majority of the decrease in $SO_2$ concentrations.

**Table C-14**
**Median Values of the Distribution of Ratios of 2010 Post-CAAA/Pre-CAAA Adjustment Factors**

| | $SO_2$ | NO | $NO_2$ | CO |
|---|---|---|---|---|
| Spring | 0.904 | 0.669 | 0.598 | 0.790 |
| Summer | 0.892 | 0.666 | 0.575 | 0.720 |
| Autumn | 0.916 | 0.677 | 0.614 | 0.756 |
| Winter | 0.924 | 0.686 | 0.626 | 0.692 |

**Table C-15**
**Background Concentrations used to Prepare the $SO_2$, NO, $NO_2$, and CO Profiles**

| Pollutant | Background Concentration |
|---|---|
| $SO_2$ | 0 |
| NO | 0 |
| $NO_2$ | 0 |
| CO | 0.2 ppm |

## Attributes and Limitations of the Modeling Analysis Methodology

The Section 812 prospective modeling analysis utilized a set of modeling tools and input databases that for the most part had been developed, tested, and evaluated as part other modeling studies (e.g., OTAG, SIP modeling analyses, etc.). This provided a cost-effective means of conducting a national-scale modeling exercise. The models used for the study are among the most widely used and evaluated tools for ozone and PM modeling, and have been used for previous regulatory applications. The modeling was performed in manner that is consistent with established practice and EPA guidelines regarding air quality model applications.

Although appropriate techniques were used for the analysis of each pollutant, use of separate models/techniques for the analysis of ozone, PM, and the other criteria pollutants does not allow a fully integrated analysis of the effects of each. Consequently, the results do not reflect all potential interactions between pollutants (e.g., ozone and PM). Ongoing research involving the development and testing of integrated modeling tools (by EPA and other organizations) may provide the opportunity for fully integrated future Section 812 prospective modeling efforts.

Analysis of the effects on the national scale (the CAAA applies to the entire nation) required the use of several different domains with varying grid resolution as well as the use of relatively coarse resolution for many areas of the country for the grid-based modeling effort. The use of relatively coarse grid resolution (12 km and greater) is a potentially important source of uncertainty with respect to the modeling results. Previous studies have found that the response of the UAM-V modeling system to emission reductions is affected by grid resolution (Douglas et al., 1996). Thus, the use of grid-cell specific adjustment factors to modify site-specific data may introduce some uncertainty into the future-year estimates.

There are always uncertainties associated with the use of modeling results to estimate future-year air quality. These derive from inaccuracies in the model inputs and/or model formulation and were manifested in this study in the evaluation of model performance. While good model performance was

achieved for most model applications, ozone concentrations were underestimated within the Los Angeles domain and PM concentrations were underestimated during the fall and winter simulation periods in the REMSAD application. RADM/RPM, used as part of the PM and visibility analyses, showed a tendency to overestimate annual average sulfate concentrations and warm season nitrate concentrations. Annual average nitrate predictions generated by RADM/RPM, however, matched air quality monitor data.

The acid deposition estimates included in the present analysis are limited to the eastern states within the RADM domain. Deposition in the western U.S. was not modeled for this study. Although acid deposition is a problem primarily for the eastern U.S., 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 associated with the Clean Air Act Amendments.

The approach used in this study to estimate future air quality (the combined use of observed data and modeling results) may, compared to a more standard air quality model application (e.g., a model application for attainment demonstration purposes), tend to minimize the effects of many of the uncertainties mentioned in this section. The reason for this is that the modeling results are used in a relative sense, rather than an absolute sense. This may enhance the reliability of the future-year concentration estimates, especially in the event that the uncertainty inherent in the absolute concentration values is greater than that associated with the response of the modeling system to changes in emissions.

The ratios for adjusting the observed data are calculated using modeling results for a limited number of simulation days and it is assumed, using this methodology, that the ratios can be used to represent longer time periods. This approach permits the estimation of seasonal and annual concentration distributions. Nevertheless, the use of the model-based ratios in adjusting data for an entire season or year may result in some over- or underestimation of the benefits of the simulated control measures, depending upon whether the simulation results for the modeled days are sufficiently representative of the meteorological and air quality conditions that occurred during 1990.

Finally, there are numerous ways in which the adjustment factors could be calculated and applied. The approach used in this study was designed to make the best use of the information and level of detail present in both the observations and the modeling results (e.g., use of decile and quintile based ratios for ozone and PM, respectively). The specific assumptions employed in the application of the methodology, however, may affect the resulting air quality profiles and should be carefully considered in the subsequent use and interpretation of the results.

## Conclusions and Recommendations for Further Research

The results from the air quality modeling component of the Section 812 prospective analysis indicate that for both future years (2000 and 2010), the measures and programs associated with the CAAA are expected to result in lower concentrations of ozone, PM, and the other criteria pollutants compared to a future-year scenario without such programs. The degree of improvement in air quality varies among the criteria pollutants and the various portions of the country included in the modeling analysis. The results also differ between the two future years, such that the improvements are greater and more widespread for 2010.

The modeling analysis relied on a set of modeling tools and input databases that (for the most part) had been developed, tested, and evaluated as part other modeling studies. It also made use of several of the most widely used and comprehensively tested tools for ozone and PM modeling. The modeling was performed in a manner that is consistent with established practice and EPA guidelines regarding air quality model applications. However, as noted in the

previous section of this report, there are several features of the modeling analysis that could be improved upon, especially considering recent advances in the development of integrated modeling tools and techniques. Recommendations for future air quality modeling efforts to support the Section 812 prospective analyses include:

- Selection of modeling episode periods using an integrated episode selection procedure (e.g., Deuel and Douglas, 1998) such that the modeling periods are representative of the historical meteorological and air quality conditions and can be used to represent seasonal and annual ozone, PM, and visibility metrics

- Reconfiguration of the modeling domain(s) such that a consistent use of high-resolution grids over urban areas with complex meteorological or emissions-based features are accommodated.

- Review and update of the input data and input preparation techniques to include, for example, updated (more recent) emissions estimates (anthropogenic and biogenic), higher-resolution meteorological inputs, enhanced estimates of future land-use patterns (reflecting growth of urban areas, changes in the interstate transportation networks, etc.).

- Use of an integrated modeling tool for the simultaneous analysis of the effects of emissions changes on ozone, PM, and other pollutants (several tools, including MODELS-3 and UAM-VPM, are currently undergoing development and testing). A comprehensive evaluation of model performance will be required.

- Continued review and enhancement (as appropriate) of the methodology for the combined use of observed data and modeling results.

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# Human Health Effects of Criteria Pollutants

Appendix D

## Introduction

In response to the mandate of section 812 of the Clean Air Act Amendments of 1990 (CAAA), EPA identified and estimated the quantifiable health benefits Americans should enjoy in 2000 and 2010 due to improved air quality resulting from the CAAA. The results suggest that the CAAA will result in significant reductions in mortality, respiratory illness, heart disease, and other adverse health effects, in addition to those reported in EPA's (1997) retrospective analysis of the Clean Air Act. In that analysis, the Agency found that significant health benefits accrued between 1970 and 1990, especially as a consequence of the reductions in ambient particulate matter (PM).

This appendix presents an overview of EPA's approach for modeling the human health effects of the CAAA. It outlines the principles used to guide the human health benefits analysis, describes methods used to quantify criteria air pollutant exposure nationwide, and discusses issues that arise in using health effect information. Following this overview, the appendix presents the modeling results, reported as estimates of avoided incidences of adverse health effects.

## Health Effects Modeling Approach

In the section 812 retrospective analysis, EPA (1997) developed an approach for quantifying the effects of reduced pollutant exposure in the 48 continental states and the District of Columbia, with particular focus on those effect categories for which monetary benefits could be estimated. The study design adopted for this analysis follows a similar approach, using a sequence of linked analytical models to estimate benefits. The first step is an analysis of the likely implementation activities undertaken in response to the CAAA. These forecasted activities provided a basis for modeling criteria pollutant emissions under the two scenarios considered (the Pre-CAAA scenario and the Post-CAAA scenario), as documented in Appendix A. The emissions estimates were input into the air quality models (Appendix C), and ambient pollutant concentrations estimated by the air quality models were input into the health benefits model, the focus of this appendix.

The health benefits model relies on two inputs: (1) forecasted changes in pollutant exposures across the study period, and (2) concentration-response (C-R) functions that quantify the relationship between the forecasted changes in exposure and expected changes in specific health effects.We discuss the inputs used for the 48 continental states and the District of Columbia below.[1]

### *Quantifying Changes in Pollutant Exposures*

Quantifying changes in pollutant exposures in this analysis relies on two inputs: (1) forecasts of ambient pollution levels at the available air quality monitors in the 48 contiguous states, and (2) extrapolations from the available air quality monitors (which are not uniformly distributed across the U.S.) to a population grid system of eight km by eight km cells that covers the 48 contiguous states and the District of Columbia.

[1] These inputs could also be used to estimate exposure in the border regions of Mexico and Canada that might have improved air quality in the Post-CAAA scenario.

## Forecasting 2000 and 2010 Pollution Levels at Ambient Air Quality Monitors

When quantifying adverse human health effects, the section 812 prospective analysis estimated 2000 and 2010 ambient concentrations for both the Pre-CAAA and Post-CAAA scenarios for the following pollutants and air quality parameters:

- Particulate matter, less than 10 microns in diameter ($PM_{10}$)
- Particulate matter, less than 2.5 microns in diameter ($PM_{2.5}$)
- Ozone ($O_3$)
- Nitrogen dioxide ($NO_2$)
- Sulfur dioxide ($SO_2$)
- Carbon monoxide (CO)

The sixth criteria pollutant, lead (Pb), is not included in this analysis since airborne emissions of lead were virtually eliminated by pre-1990 CAA programs. The methods used to estimate the concentrations of these pollutants at monitors are described in Appendix C.

## Extrapolating Forecasts at Air Quality Monitors to Population Grid Cells

The next step is to extend forecasts for a limited number of air quality monitors to estimate population exposure at all locations in the continental United States, using the Criteria Air Pollutant Modeling System (CAPMS). CAPMS divides the United States into eight kilometer by eight kilometer grid cells and estimates the changes in incidence of adverse health effects associated with given changes in air quality in each grid cell. The national incidence change (or the changes within individual states or counties) is then calculated as the sum of grid-cell-specific changes. To calculate changes in population exposure in a grid cell, CAPMS requires data on the population in the grid-cell and the change in air quality.

First, grid-cell-specific population counts for 1990 are derived from U.S. Census Bureau block level population data (Wessex, 1994). Future year grid-cell population estimates are then extrapolated from 1990 grid-cell population levels using the ratio of future-year and 1990 state-level population estimates provided by the U.S. Bureau of Economic Analysis (1995). CAPMS assumes that all grid cell populations in a state grow at the same rate as the state population as a whole (where a grid cell is defined as being in a state if the grid cell centroid is in the state).

Second, CAPMS requires estimates of two air quality regimes at CAPMS grid cell centers: baseline (in this case, 1990) air quality levels and regulatory alternative air quality levels in future years (in this case, 2000 and 2010). Air quality inputs to CAPMS for pre- and Post-CAAA scenarios must use the averaging time required by the C-R functions being used.[2] For example, a C-R function relating mortality to annual median $PM_{2.5}$ concentrations requires that annual median $PM_{2.5}$ concentrations be available at CAPMS grid cell centers. Although the input $PM_{2.5}$ data must be in the form of daily averages, the monitors need not be at CAPMS grid cell centers. Given any set of location-specific air quality data, CAPMS interpolates the corresponding air quality values at each CAPMS grid cell center.

To reduce computational time when estimating the change in health effects associated with daily pollution levels, CAPMS approximates a year's (or season's) worth of daily pollutant concentrations at each monitor by 20 "bins" of pollutant concentrations. Each bin represents five percent of the daily pollutant concentrations in the period of interest, and is set at the midpoint of the percentile range it represents. For n = 20 and a year's worth of observations, the first bin represents the first (lowest) five percent of the distribution of 365 pollutant concentrations at the given location, and is set at the 2.5th percentile value; the second bin represents the next five percent of the distribution of daily values,

[2]The development of C-R functions is discussed later in this appendix.

and is set at the 7.5th percentile value, and so on. Each of the twenty bins therefore represents 18.25 (=365/20) days. Interpolation of air quality levels at CAPMS grid cell centers is based on these input location-specific bins, so that the annual incidence changes in each grid cell are calculated for twenty pollutant concentrations (the 20 bins of air quality) rather than for 365 pollutant concentrations. The resulting incidence change is then multiplied by 18.25 to reconstruct an entire years' worth of incidence change in the CAPMS grid cell.

As shown in Figure 1, actual ambient pollution data is only available from limited monitor sites. Data must be extrapolated to unmonitored locations, in order to estimate the impact of air pollution on the health and welfare effects considered in this analysis. The available air monitoring data were extrapolated from all available monitor locations to a grid of eight km by eight km population grid-cells throughout the contiguous 48 states, using a Voronoi neighbor averaging (VNA) spatial interpolation procedure.[3]

The VNA procedure interpolates air quality estimates from the set of surrounding air quality monitors to the center of each population grid-cell. The VNA procedure is a generalization of planar interpolation. Rather than arbitrarily limiting the selection of monitors, VNA identifies the set of monitors that best "surrounds" the center of each grid-cell by identifying which monitor is closest (considering both angular direction and horizontal distance) in each direction from the grid-cell center. Each selected monitor will likely be the closest monitor for multiple directions. The set of monitors found using this approach forms a polygon around the grid-cell center.

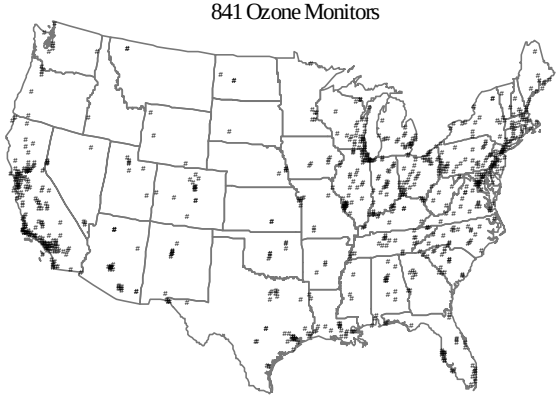
For each grid cell, CAPMS calculates the distance to each member of a set of monitors surrounding that grid cell. Monitors close to the grid cell are assumed to yield a more accurate air quality description of that grid cell, and are given a larger weight when calculating the average air quality for that grid cell. Conversely, monitors that are further away receive less weight. After determining the final set of surrounding monitors, the grid cell's air quality level is calculated as an inverse, distance-weighted average of the air quality levels at the selected monitors.

Air quality estimates generated using this VNA method are likely to be most uncertain at population grid cell locations far removed from the nearest monitor. For example, if a grid cell encompasses a relatively unpolluted rural area, but the nearest (albeit distant) monitors are measuring air quality in industrialized urban areas, the VNA method described above will overestimate the pollution level for that grid cell. As a result, this monitor-based VNA extrapolation method is used only at grid cells located within 50 kilometers of an air pollution monitor.

At distances greater than 50 kilometers from a monitor, additional information is needed to improve the estimates of air quality in unmonitored areas. A modified VNA method incorporating both monitor data and air quality modeling predictions is employed at these grid cell locations. In addition to the distance-weighted averaging of monitor concentrations, this modified extrapolation method incorporates a spatial adjustment factor that reflects the ratio of model-derived air quality predictions at the target and source locations. The addition of the modeling results helps account for differences in geography, meteorology, land use and other factors affecting air pollution levels between the target and source areas. Additional details on both VNA extrapolation methods can be found in Abt Associates (1999).

[3]For locations within 50 kilometers of a monitor, the interpolation method is the same as that used by Abt Associates (1998) for the $NO_x$ SIP call analysis; previously termed the "convex polygon" method, it is more accurately described as Voronoi neighbor averaging (VNA) spatial interpolation, which will be used throughout this document.

841 Ozone Monitors

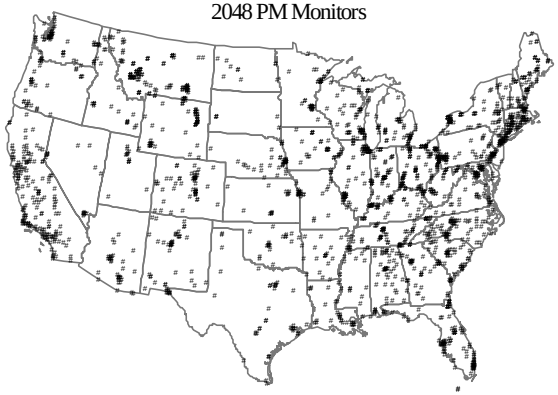


761 SO2 Monitors

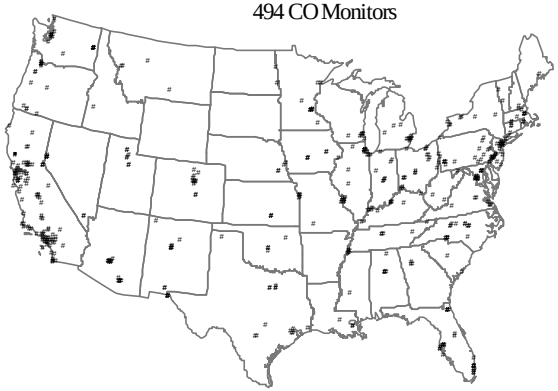
348 NO2 Monitors

**Figure D-1**
**Location of Air Quality Monitors**
**Section 812 Analysis**

2048 PM Monitors



494 CO Monitors



### Quantifying Human Health Effects of Exposure

To calculate point estimates of the changes in incidence of a given selection of adverse health and welfare effects associated with a given set of air quality changes, CAPMS performs the following steps for each grid cell: (1) Interpolation of the air quality in the baseline scenario and in the control scenario at each CAPMS grid cell center for each pollutant. (2) Calculation of the changes in air quality from baseline to control scenario in the CAPMS grid cell. The changes in air quality are calculated as the differences between the baseline bins and the corresponding control scenario bins. (3) Identification of the selected C-R functions being used, and the required baseline incidence rates and the relevant grid cell population. (4) Calculation of the change in incidence of each adverse health effect for which a C-R function has been identified. The resulting annual incidence change for each grid cell is then summed with those of the other grid cells, to calculate the estimated change in incidence nationwide.

## Types of Health Studies

Research on the health effects of air pollution strongly suggests that reductions in the incidence of adverse health effects are a significant benefit of air pollution control. The available human health studies that could serve as the basis of the section 812 prospective assessment can be categorized into chamber studies and epidemiology studies. Chamber studies involve examination of human responses to controlled conditions in a laboratory setting, while epidemiological studies investigate the association between exposure to ambient air pollution and observed health effects in a study population. The relative advantages of reliance on each type of research are described below.

### Chamber Studies

Chamber 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 exposure. These measurements can include general biomedical information (e.g., pulse rate and blood pressure), physiological effects specifically induced by the pollutant (e.g., altered 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, which increases respiration and the amount of pollution introduced into the lungs.

One advantage of chamber studies is that they can potentially establish cause-effect relationships between pollutants and certain human health effects. In addition, repeated experiments altering the pollutant level, exercise regime, and type 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, chamber 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 chamber studies as the basis for a comprehensive benefits analysis. Chamber studies are most appropriate for examining acute symptoms caused by exposure to a pollutant for a few hours. While this permits examination of some important health effects from air pollution, such as broncho-constriction in asthmatic individuals caused by sulfur dioxide, it precludes studying effects caused by long term exposure. Another drawback is that health effects measured in some well-designed chamber studies are selected on the basis of the ability to measure precisely an effect, for example forced expiratory volume, rather than a larger symptom. Some of these measurable but relatively minor health effects, such as reduced lung function, have an unclear impact on future medical condition and lifestyle, although some research discussed later has addressed this question.

Ethical considerations in experiments involving humans also impose important limits on the potential scope of chamber research. Chronic effects cannot be

investigated because people cannot be kept in controlled conditions for an extended period of time, and because chronic effects are generally irreversible. Participation is generally restricted to healthy subjects, or at least excludes people with existing health conditions that compromise their safe inclusion in the study. This can result in a lack of direct evidence about populations of most concern, such as people who already have serious respiratory diseases. Ethical considerations also limit experimental pollutant concentrations to relatively modest exposure levels, and confine studies to examining only mild health effects that are believed to do no permanent damage. Obviously, for ethical reasons, evidence from chamber studies cannot be obtained on the possible relationship between pollution and mortality, heart attack, strokes, or cancer.

The information derived from chamber research raises some questions as to how well it applies to the general population and their activity patterns and pollution exposures. (1) The dose-response functions developed from chamber research are specific to the population participating in the study. Chamber studies typically study a small population -- certainly much smaller than those typically evaluated in epidemiological studies (discussed below) -- so there are concerns that the results may not apply to the much larger and likely more diverse general population. (2) Chamber studies evaluate only a certain number of activity patterns (e.g., exercise), and cannot replicate the diverse pattern of activity seen in the course of a day. (3) Chamber studies cannot easily replicate the varied pollution levels to which people are exposed during the course of their day at work, on the freeway, at home, and other places.

As discussed below in the section on health effects study selection, the generalizability of results is an important factor in this analysis. Studies that use a large, diverse group of subjects are easier to apply to the general population than studies using smaller, narrowly defined group of subjects. This does not, however, rule out studies that focus on asthmatics, children, or the elderly, since these groups may be particularly sensitive to air pollution. Similarly, studies that use exposure regimes and exercise levels similar to what large groups of the population experience are easier to apply in a benefits model than are less representative studies.

## *Epidemiological Studies*

Epidemiological studies present the results of a statistical analysis of the relationship between ambient pollution exposure and adverse health effects. The data for these studies includes ambient air quality monitoring data and adverse health effects data such as mortality incidence (e.g., National Center for Health Statistics, 1994), hospital admissions (e.g., Graves and Gillum, 1997), questionnaires (e.g., Adams and Marano, 1995), and diaries that are kept by study participants over a period of time (e.g., Ostro et al., 1991).

At least to some extent, these estimated relationships implicitly take into account complex real-world human activity patterns (including actions to avoid air pollution), spatial and temporal differences in air pollution distributions, and possible synergistic effects of multiple pollutants. Epidemiological studies typically involve a large number of people and may not suffer as much from the extrapolation problems common to chamber studies, which often have a limited number of subjects. In addition, observable health endpoints are measured, unlike chamber studies, which often monitor endpoints that do not result in observable health effects (e.g. forced expiratory volume).

Two types of epidemiological studies are considered for inclusion in this analysis: individual-level cohort studies and population-level ecological studies. Cohort-based studies track individuals over a certain period of time, with periodic evaluation of the individuals' exposure and health status. Cohort studies can either follow a group of initially disease-free individuals forward in time (a prospective cohort) or gather historical data on exposure and disease for a given group (a retrospective cohort). 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 shorter time period. An important feature of cohort studies is that information is collected about each individual that may include other variables that could be correlated with both the exposure and the disease outcome, such as smoking or income. If investigators do not identify and control for these variables, called confounders, in

a study, they may produce a spurious association between air pollution and adverse health effects.

A second type of study used in this analysis is a population-level ecological study. These studies assess 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. There are two types of such studies: cross-sectional and time-series studies. Using data at a point in time from a variety of locations, cross-sectional studies examine the relationship between pollution exposure and adverse health effects, while trying to control confounding variables. Cross-sectional studies are not as desirable as prospective cohort studies, in part, because of their failure to control for important covariates such as smoking.[4] Rather than look at variety of locations at one point in time, a time series analysis studies a single location and typically examines the relationship between daily changes in ambient pollution level and daily changes in adverse health effects. An important 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, and is thus similar to a cohort study (Schwartz, 1997, p. 372). Other factors that change over time can also affect health (tobacco, alcohol and illicit drug use, access to health care, employment, and nutrition). However, since such potential confounding factors are unlikely to vary from day to day in the same manner as air pollution levels, these factors are unlikely to affect the magnitude of the association between air pollution and daily variations in human health responses.

Drawbacks to epidemiological studies include difficulties associated with adequately characterizing exposure to individuals (that tends to lead to a downward bias in the estimated pollution-health effect relationship), and uncontrolled confounding variables, that can potentially lead to spurious conclusions. In particular, air pollutants are often highly correlated, so it is difficult to determine which may be associated with an adverse effect. In addition, epidemiological studies, by design, are unable to definitively prove a causal relationship between an exposure and a given health effect; they can only identify associations or correlations between exposure and the health outcome. However, given the major advantage of epidemiological studies -- relatively severe health effects may be observed in a large, more heterogeneous population -- epidemiological studies are used as the basis for determining the majority of health effects and C-R functions in this analysis. Chamber studies are used if there are health effects observed in chamber studies not observed in epidemiological studies, such as shortness of breath in young asthmatics induced by $SO_2$ (e.g., Linn et al., 1987).

[4]Criticisms of cross-sectional studies are considered in Evans et al. (1984), Lipfert and Wyzga (1995), and others.

## Selection of C-R Functions

This section describes the methods used to derive the C-R functions used in this analysis to quantify the effect of CO, $NO_2$, $SO_2$, $O_3$, and PM on people's health. It discusses the general issues that arise with the choice and use of C-R functions, and the issues specific to C-R functions for mortality and morbidity.

### *C-R Function General Issues*

#### Derivation of C-R Functions

For expository simplicity, the following discussion focuses on PM C-R functions, although it applies to all of the health effects and pollutants considered in the 812 prospective analysis. In what follows, the health effect estimated is simply denoted as y, and is estimated at a single location (population cell), where a change in PM air quality ($\Delta$PM) corresponds to a change in the health endpoint ($\Delta$y). The calculation of $\Delta$y depends on a C-R function, derived typically from an epidemiological study.

There are a variety of epidemiological studies in the science literature, making it important to understand the nuances of each study before developing a C-R function. Different epidemiological studies may have estimated the relationship between PM and a particular health endpoint in different locations. The C-R functions estimated by these studies may differ from each other in several ways. They may have different functional forms; they may have measured PM concentrations in different ways;

they may have characterized the health endpoint, y, in slightly different ways; or they may have considered different types of populations. Some studies have assumed that the relationship between y and PM is best described by a linear form, where the relationship between y and PM is estimated by a linear regression in which y is the dependent variable and PM is one of several independent variables, while other studies have assumed that the relationship is best described by a log-linear form (i.e., the relationship between the natural logarithm of y and PM is estimated by a linear regression).[5] Some studies of the relationship between ambient PM concentrations and mortality have excluded accidental deaths from their mortality counts; others have included all deaths. One study may have measured daily (24-hour) average PM concentrations, while another study may have used two-day averages. Finally, one study may have considered changes in the health endpoint only among members of a particular subgroup of the population (e.g., individuals 65 and older), while other studies may have considered the entire population in the study location.

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

$$y = Be^{\beta \cdot PM} \ , \qquad \textbf{(1)}$$

or, equivalently,

$$\ln(y) = \alpha + \beta \cdot PM \ , \qquad \textbf{(2)}$$

where the parameter B is the incidence of y when the concentration of PM is zero, the parameter β is the coefficient of PM, ln(y) is the natural logarithm of y, and $\alpha = \ln(B)$.[6] If the functional form of the C-R relationship is log-linear, the relationship between ΔPM (= $PM_{baseline}$ - $PM_{after\ change}$) and Δy is:

$$\Delta y = y - y_{after\ change} = -y \cdot \left[e^{-\beta \cdot \Delta PM} - 1\right], \qquad \textbf{(3)}$$

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

$$RR_{\Delta PM} = \frac{y_{after\ change}}{y} = e^{-\beta \cdot \Delta PM} \qquad \textbf{(4)}$$

Epidemiological studies often report a relative risk for a given ΔPM, rather than the C-R coefficient, β. The coefficient can be derived from the reported relative risk and ΔPM, however, by solving for β in equation (4):

$$\beta = -\frac{\ln(RR)}{\Delta PM}. \qquad \textbf{(5)}$$

The linear relationship is of the form:

$$y = \alpha + \beta \cdot PM \ , \qquad \textbf{(6)}$$

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

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

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

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

$$\Delta y = \beta \cdot \Delta PM \ . \qquad \textbf{(7)}$$

If the C-R function is linear, equation (7) may be used to estimate Δy associated with ΔPM, assuming the measurement of ΔPM is consistent with the PM measurement used in the health effects study from which the C-R function was derived. If the function is log-linear, the baseline incidence for y and an appropriate measure for ΔPM may be used in equation (3).

A few epidemiological studies, estimating the relationship between certain morbidity endpoints and air pollution, have used functional forms other than linear or log-linear forms. Of these, logistic regressions are the most common. The details of the models used in these studies are given in the papers reporting the methods and results of the studies.

## Thresholds

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

Thresholds may *also* be incorporated by a policy analyst using a C-R function derived from the original study, even if the original study did not assume a threshold. A threshold may be set at any point, although some points may be considered more obvious candidates than others. One possible assumption is that there is a threshold at the non-anthropogenic background level of the pollutant. Another possibility is there is a threshold at the lowest observed level in the study that estimated the C-R function. Another might be a relevant standard for the pollutant.

One method to conduct policy analysis assuming a threshold model is to simply truncate the C-R function at the threshold (i.e., to exclude any physical effect changes associated with, say, PM levels below the designated threshold). This method uses the original C-R function, but calculates the change in PM as [max(T, baseline PM) - max(T, regulatory alternative PM)], where T denotes the designated threshold. An alternative method is to replace the original C-R function with a "hockey stick" model that best approximates the original function that was estimated using actual data. A typical hockey stick C-R function is horizontal up to a designated threshold PM level, T, and is linear with a positive slope for PM concentrations greater than T. This is just the following variation on equation (2) above:

$$\ln(y) = \alpha \quad for\ PM \le T\ , \qquad \textbf{(8)}$$

$$= \alpha + \beta \cdot PM \quad for\ PM > T\ ,\ \ where\ \beta > 0\ . \qquad \textbf{(9)}$$

The specification of such a 'hockey stick' model, while theoretically preferable to a simple truncation model, requires re-analysis of the underlying data from the original health effect study. Such primary re-analysis is beyond the scope of the section 812 analysis. Alternatively, if a simple truncation model is used, application of the resulting C-R function would likely result in a significant underestimate of the health effects avoided by reductions in pollutant exposures above the assumed threshold.

The possible existence of an effect threshold is a very important scientific question and issue for policy analyses such as the section 812 analysis. However, there is currently no scientific basis for selecting a particular threshold for the effects considered in this

analysis, if a threshold is defined as a level characterized by an absence of observable effects. Therefore, this analysis assumes there are no thresholds for modeling health effects. However, the potential impact of alternative threshold assumptions for PM-related premature mortality is explored as a key sensitivity analysis.

## Pooling Studies

When only a single study has estimated the C-R relationship between a pollutant and a given health endpoint, the estimation of a population cell-specific incidence change is straightforward. For some endpoints, however, C-R functions have been estimated by several studies, often in several locations. In this case, if the input components (e.g., functional forms, pollutant averaging times, study populations) are all the same (or very similar), a pooled, "central tendency" C-R function can be derived from the multiple study-specific C-R functions.

One potential method of pooled analysis is simply averaging the coefficients from all the studies. This has the advantage of simplicity, but the disadvantage of not taking into account the measured uncertainty of each of the estimates. Estimates with great uncertainty surrounding them are given the same weight as estimates with very little uncertainty.

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

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

In some cases, studies reported several estimates of the C-R coefficient, each corresponding to a particular year or particular study area. For example, Ostro and Rothschild (1989b) report six separate regression coefficients that correspond to regression models run for six separate years. This analysis combined the individual estimates using a meta-analysis on the six years of data.

## Pollution Exposure Measure

The study on which an acute exposure C-R function is based may have used pollution concentrations averaged over several days. Those studies that use multi-day averages are in effect using a smoothed data set, comparing each day's adverse health effects to recent average exposure rather than simply exposure on the same day. This does not have much effect on the estimated adverse health effects, especially when the C-R function has a linear or nearly linear functional form. For example, if the functional form were linear and based on a five-day pollution average, then the estimated effects over the course of the year would be essentially the same between using daily pollution observations in the C-R function or a two-day average. This is analogous to summing up five numbers (6,4,8,4,8=30) or taking their average and multiplying by five (6*5=30); in each case the answer is 30. This analysis uses daily pollution levels in cases where there are multi-day averaging times.

## Regional Variation

Whether the C-R relationship between a pollutant and a given health endpoint is estimated by a single function from a single study or by a pooled function of C-R functions from several studies, that same C-R relationship is applied everywhere in the current benefits analysis. Although the C-R relationship may in fact vary somewhat from one location to another (for example, due to differences in population susceptibilities or differences in the composition of PM), location-specific C-R functions are available only for those locations in which studies were conducted. A single function applied everywhere may result in overestimates of incidence changes in some locations and underestimates of incidence changes in other locations. It is not possible, however, to know the extent or direction of the overall bias in the total incidence change introduced by application of a single C-R function everywhere.

## PM Size and Composition

Current research suggests that particle size, and perhaps particle composition, matters when estimating the health impacts of PM. Particulate matter is a heterogeneous mix that varies over time and space, and may include solid or liquid compounds, including organic aerosols, sulfates, nitrates, metals, elemental carbon, and other material. Fine PM is generally viewed as having a more harmful impact than coarse PM, although it is not clear to what extent this may differ by the type of health effect or the exposed population. While one cannot necessarily assume that coarse PM has no adverse impact on health, it seems reasonable to prefer the use of $PM_{2.5}$ as a proxy for the impact of PM. Due to the relative abundance of studies using $PM_{10}$, however, and the reasonably good correlation between $PM_{2.5}$ and $PM_{10}$ in urban areas, in many cases this analysis also uses $PM_{10}$ studies to estimate the impact of PM. Similarly, at this stage of knowledge, it is not clear what composition distinctions to make, if any, when estimating the impact of PM. The C-R functions used in this analysis relate adverse health effects to an undifferentiated mass of particles (e.g., $PM_{10}$); they do not relate effects to individual PM components.

## Baseline Incidence Rate

Some C-R functions (those expressed as a change relative to baseline conditions) require baseline incidence data associated with ambient levels of pollutants. Baseline 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. County-specific estimates of baseline mortality incidences used in this analysis were obtained from the National Center for Health Statistics (1994). The National Center for Health Statistics also provided much of the information on national incidence rates. However, for some studies, the only available baseline incidence information comes from the studies themselves; in these cases, the baseline incidence in the study population is assumed to represent the baseline incidence nationally.

## Population

Many studies focus on a particular age cohort. The age group chosen is often a matter of convenience (e.g., extensive Medicare data may be available for the elderly population) and not because the effects are necessarily restricted to the specific age group, even though their incidence may vary considerably over an individual's life span. Nevertheless, to avoid overestimating the benefits of reduced pollution levels, this analysis applies the given C-R 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, such as in the estimation of worker productivity. In these cases, application of dose-response functions has been restricted to populations of individuals with these same characteristics. As discussed in more detail below, however, by assuming that the C-R relationships should only be applied to those subpopulations matching the original study population, the present analysis may be significantly underestimating the whole population benefits of reductions in pollutant exposures.

## C-R Function Selection Criteria

A number of considerations arose in selecting and applying concentration-response (C-R) functions for the section 812 prospective assessment. These considerations are summarized in Table D-1 below. Because concentration-response functions are the means of relating changes in pollutant levels to changes in health endpoints, they are a critical component of a benefits analysis. While a study may be superior with regard to one consideration (e.g., number of pollutants considered), it may be inferior with regard to another consideration (e.g., number of observations). The selection of C-R functions for the benefits analysis was guided by the goal of achieving a balance between comprehensiveness and scientific defensibility. The issues considered are discussed below in some detail.

**Table D-1**
**Summary of Considerations Used in Selecting C-R Functions**

| Consideration | Comments |
|---|---|
| Peer reviewed research | Peer reviewed research is preferred to research that has not undergone the peer review process. |
| Study type | Among studies that consider chronic exposure (e.g., over a year or longer) prospective cohort studies are preferred over cross-sectional studies (a.k.a. "ecological studies") because they control for important confounding variables that cannot be controlled for in cross-sectional studies. If the chronic effects of a pollutant are considered more important than its acute effects, prospective cohort studies may also be preferable to longitudinal time series studies because the latter type of study is typically designed to detect the effects of short-term (e.g. daily) exposures, rather than chronic exposures. |
| Study period | Studies examining a relatively longer period of time (and therefore having more data) are preferred, because they have greater statistical power to detect effects. More recent studies are also preferred because of possible changes in pollution mixes, medical care, and life style over time. |
| Study population | Studies examining a relatively large sample are preferred. Studies of narrow population groups are generally disfavored, although this does not exclude the possibility of studying populations that are potentially more sensitive to pollutants (e.g., asthmatics, children, elderly). However, there are tradeoffs to comprehensiveness of study population. Selecting a C-R function from a study that considered all ages will avoid omitting the benefits associated with any population age category. However, if the age distribution of a study population from an “all population” study is different from the age distribution in the assessment population, and if pollutant effects vary by age, then bias can be introduced into the benefits analysis. |
| Study location | U.S. studies are more desirable than non-U.S. studies because of potential differences in pollution characteristics, exposure patterns, medical care system, and life style. |
| Pollutants included in model | Models with more pollutants are generally preferred to models with fewer pollutants, though careful attention must be paid to potential collinearity between pollutants. Because PM has been acknowledged to be an important and pervasive pollutant, models that include some measure of PM are highly preferred to those that do not. |
| Measure of PM | $PM_{2.5}$ and $PM_{10}$ are preferred to other measures of particulate matter, such as total suspended particulate matter (TSP), coefficient of haze (COH), or black smoke (BS) based on evidence that $PM_{2.5}$ and $PM_{10}$ are more directly correlated with adverse health effects than are these more general measures of PM. |
| Economically valuable health effects | Some health effects, such as forced expiratory volume and other technical measurements of lung functioning, are difficult to value in monetary terms. These health effects are not quantified in this analysis. |
| Non-overlapping endpoints | Although the benefits associated with each individual health endpoint may be analyzed separately, care must be exercised in selecting health endpoints to include in the overall benefits analysis because of the possibility of double counting of benefits. Including emergency room visits in a benefits analysis that already considers hospital admissions, for example, will result in double counting of some benefits if the category “hospital admissions” includes emergency room visits. |

## Peer-Review of Research

Whenever possible, peer-reviewed research rather than unpublished information has been used. Research that has been reviewed by the EPA's own peer review processes, such as review by the Clean Air Scientific 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.

Studies published (or accepted for publication) in peer reviewed journals but not reviewed by CASAC have also been considered for use in the section 812 prospective assessment, and have been used if they are determined to be the most appropriate available studies. Indications that EPA intends to submit research to the CASAC (such as inclusion in a draft Criteria Document or Staff Paper) are considered further evidence that specific journal-published research is acceptable for use in this analysis.

Air pollution health research is a very active field of scientific inquiry, and new results are being produced regularly. Many research findings are first released in university working papers, dissertations, government reports, non-reviewed journals and conference proceedings. Some research is often 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.

## Study Type and Quality

Epidemiological studies of the relationship between air pollutants and health endpoints can generally be categorized as (1) "ecological" cross-sectional, (2) prospective cohort, or (3) longitudinal time series studies. The first two types of study are concerned with longer exposure periods, such as a year or over several years, while the third type is concerned with short-term exposures over one or more days. Among studies that consider longer exposure periods, or chronic exposure, prospective cohort studies are preferable to "ecological" cross-sectional studies, because they control for important confounding variables which cannot be controlled for in "ecological" cross-sectional studies. If the effects of chronic exposures are considered more significant than acute effects, prospective cohort studies may also be preferable to longitudinal time series studies because the latter type of study is typically designed to detect the effects only of daily exposures, rather than chronic exposures.

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. For example, a study that considers only air pollution, omitting other variables associated with a health outcome, could incorrectly conclude that a reduction in air pollution is exclusively responsible for a reduction in the health outcome. Potential confounders include weather-related variables (e.g., temperature) and ambient pollutants other than those being studied.

## Study Population

Many of the studies relevant to quantifying the benefits of air pollution reductions have focused on subpopulations that may or may not be representative of the general population. Extrapolating results from studies on nonrepresentative subpopulations to the general population introduces uncertainties into 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 C-R functions only to those subpopulations with the characteristics of the study population. While this approach has merit in minimizing uncertainty, it can result in a severe underestimate of benefits if similar effects are likely to occur in the rest of the population. For these reasons, studies that examine broad, representative populations may be preferable to studies with narrower scope, because they allow application of the functions to larger numbers of persons. There are, however, tradeoffs to comprehensiveness of study population.

Selecting a C-R function from a study that considered all ages will avoid omitting the benefits associated with any population age category. However, if the age distribution of the study population from an "all population" study is different from the age distribution in the assessment population, and if pollutant effects vary by age, then bias can be introduced into the benefits analysis.

## Study Period

Studies examining a relatively longer period of time are preferable because they have more data and therefore have greater statistical power to detect effects. In addition, more recent studies are preferable to older studies because of possible changes in pollution mixes, medical care, and life style over time. This latter issue is effectively a benefits transfer issue. Differences across time between the study period and the assessment period introduce uncertainties into the benefits analysis, because it is not known to what extent the C-R relationship estimated during the study period will be the same during the assessment period.

## Study Location

Studies conducted in locations that are different from the assessment location are generally less desirable because of the introduction of possible benefits transfer problems. The characteristics of a population (e.g., the proportion of the population that is particularly susceptible to pollution, or the behavior patterns of the population) and/or the pollution mix to which it is exposed may differ notably between the study location and the assessment location. As with differences in time periods, these differences in location introduce uncertainties into the benefits analysis, because it is not known to what extent the C-R relationship estimated in the study location is the same in the assessment location. For that reason, studies conducted in the United States or Canada are preferable for this benefits analysis to studies conducted, for example, in Europe or in developing countries. In addition, studies that include a wide range of areas are preferred. Studies focusing on a single city are not as desirable as studies that focus on multiple cities.

The preference for studies that focus on a range of areas, in the U.S. and Canada, is driven by a concern that there may be significant regional variation in the estimated C-R functions. There has not, however, been enough research to establish regional specific values.

## Pollutants Included in the Model

In many cases, several pollutants in a "pollutant mix" are correlated with each other -- that is, their concentrations tend to change together. Although there may be an association between an adverse health effect and this mix, it may not be clear which pollutant is causally related to the health effect -- or whether more than one pollutant is causally related. Using separate regressions (from single pollutant models) for each pollutant may overstate the effect of each pollutant alone. Models that consider pollutants simultaneously are therefore preferred, though careful attention must be paid to potential collinearity between pollutants. Because PM has been acknowledged to be an important pollutant, models that include some measure of PM are highly preferred to those that do not.

## Measure of Particulate Matter

Different epidemiological studies examining the health effects associated with particulate matter (PM) have used different measures of PM. Some have used $PM_{10}$ while others have used $PM_{2.5}$. The number of studies using $PM_{2.5}$ as the indicator of PM is substantially more limited than the number using $PM_{10}$ because of the relative sparseness of $PM_{2.5}$ monitor data. A number of studies have used total suspended particulate matter (TSP), British Smoke, coefficient of haze (COH) and other measures of particulate matter. There is some evidence that the relationship between fine particulates ($PM_{2.5}$) and health effects may be stronger than that between other measures of PM and health effects. If this is true, then studies that use measures of PM that more closely approximate the fine fraction of PM (such as $PM_{10}$) are preferable to those that use other measures.

## Economically Valuable Health Effects

A number of the health endpoints examined in the literature are difficult to value in monetary terms. These effects include forced expiratory volume and other technical measurements of lung functioning. It is not clear how to assign an economic value to such effects, as their impact on future medical condition and lifestyle are not well understood. One method to value these "clinical" measures is to estimate their association with adverse health effects that *are* valued.

Ostro et al. (1989a) reanalyzed data from four controlled ozone exposure studies, and found a statistically significant relationship between forced expiratory volume in one second ($FEV_1$) and the probability that an individual will report a mild, moderate or severe respiratory symptom. In this case, one could estimate ozone benefits by first calculating the change in $FEV_1$ associated with a given change in ozone concentration, converting this to a change in respiratory symptoms, and then valuing the respiratory symptom change. In a separate study, Neas and Schwartz (1998) found that certain measures of reduced pulmonary functioning are significant predictors of mortality. This result, however, would be difficult to use to calculate air pollution benefits, because they looked at the relationship between declines in lung function and mortality, and they did not estimate the impact of air pollution on this decline; separate work would be required to estimate the impact of air pollution on lung function.

The main concern when translating a clinical measure such as $FEV_1$ to an economically valuable one such as acute respiratory symptoms is that epidemiological work may already be available from which one can directly estimate a C-R function. To estimate acute respiratory symptoms directly (from an epidemiological study) and indirectly through the clinical measure, would double-count the effect. Another concern is that using the indirect method adds a layer of uncertainty because one must first translate the estimated clinical measure to the estimated economically valuable measure.

## Non-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 or upper respiratory symptoms). Measures of restricted activity provide a final example of overlapping health endpoints. Estimates are available for pollution-related restricted activity days, mild restricted activity days, and activity restriction resulting in work loss. While the benefits analysis estimates the benefits associated with individual endpoints, it takes care in deciding which endpoints to include in an estimate of total benefits, in order to avoid double-counting of benefits from overlapping endpoints.

### *Mortality*

Health researchers have consistently linked air pollution with excess mortality. Prospective cohort and cross-sectional studies have found a relationship between mortality over the course of a year or more with pollution levels measured over the course of a year or several years. In addition, a number of so-called "short-term" mortality studies have linked daily variations in mortality with daily pollution levels.

The EPA Clean Air Council (U.S. EPA, 1999, p. 11) recommends using the prospective cohort study by Pope et al. (1995), rather than short-term mortality studies. Although short-term studies lend substantial support to the hypothesis that there is a relationship between PM and mortality, they focus only on the acute effects associated with daily peak exposures. In contrast, the Pope et al. study was designed to capture the effect of exposure over many years, however it may be less able to capture the short-term impact of peak exposures. This creates an overlap of unknown size between the mortality estimates based on short-term studies and Pope et al. Capturing the chronic impact, however, is judged more important than

missing the impact of an unknown number of deaths occurring shortly after short-term peak exposures. For this reason, the Pope et al. study is preferred. A second prospective cohort study by Dockery et al. (1993) is also used to estimate the impact of PM on mortality. However, the Dockery et al. study used a smaller sample of individuals from fewer cities than the study by Pope et al., and is therefore presented only as an illustrative calculation that is consistent with Pope et al. (1995); the Pope et al. estimate is used in the primary analysis.[7]

The total mortality effect estimated by the Pope et al. (1995) and the Dockery et al (1993) studies does not necessarily occur in the same year as the estimated exposure. However, the exact relationship between the time of exposure and mortality is not well known. In the primary analysis, we assume that mortality occurs over a five year period, with 25 percent of the deaths occurring in the first year, 25 percent in the second year, and 16.7 percent in each of the third, fourth, and fifth years. We also perform an analysis of the sensitivity of benefits valuation to the lag structure by considering a range of assumptions about the timing of mortality (see Appendix H). It is important to keep in mind that changes in the lag assumptions do not change the total number of estimated deaths, but rather the timing of those deaths.

At least some evidence has been found linking each of the criteria pollutants with mortality. This raises concerns that the mortality-related benefits of air pollution reduction may be overstated if separate pollutant-specific estimates, some of which may have been obtained from models excluding the other pollutants, are aggregated. In addition, there may be important interactions between pollutants and their effect on mortality.

[7]The Pope et al., 1995 study estimated a C-R coefficient using median PM concentration data; however, mean pollutant concentration is the measure of central tendency commonly used in other health studies. We will explore the possibility of re-estimating the PM mortality C-R function using mean concentration data in future 812 prospective analyses.

The Pope et al. (1995) study included only PM, so it is unclear to what extent it may be including the impacts of ozone or other gaseous pollutants. Because of concern about overstating of benefits and because the evidence associating mortality with exposure to particulate matter is currently stronger than for other pollutants, only the benefits of PM-related mortality avoided are included in the total benefits estimate. The benefits associated with ozone reductions are estimated but are not included in the estimate of total benefits. The relationship between CO and mortality is briefly considered, but the evidence reviewed does not point to a clear link between the two.

## Statistical Lives Saved Versus Statistical Life-Years Saved

Considerable attention has been paid to using life-years lost as an alternative to lives lost as a measure of pollution-related premature mortality. This analysis uses both approaches to estimating pollution-related premature mortality.

The actual number of years any particular individual is going to live cannot be known. Instead, one estimates the *expected*, or statistical average, number of "life-years lost". The number of life-years lost may be expressed as the average number of life-years lost for all of the people who are exposed (the *ex ante* measure), or as the average number of life-years lost for the people who died from exposure (the *ex post* measure).

The *ex ante* estimate of life-years lost depends on the individual having been exposed to a pollutant, *not* on the individual having died prematurely from that exposure. Suppose, for example, that a 25 year old has a life expectancy of 50 more years in the absence of PM exposure and only 48 more years in the presence of exposure. The exposed 25 year old would, on average, have her life expectancy shortened by two years. That is, two expected life-years would be lost by every *exposed* individual.

The *ex post* estimate of life-years lost depends on the individual actually having died from exposure to pollution. 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; this can be calculated from age- and gender-specific life expectancy tables. Suppose that the life expectancy of 25 year olds is 75 -- 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 *affected* 25 year old (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 PM change, the relative risk assumed to be associated with the change in PM, and the length of exposure. 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.[8] 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 one 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 over which the average is calculated, however, can make a crucial difference in the reported average life-years lost. 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 who died divided by the number of exposed individuals. 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 *ex ante* and *ex post* measures of life-years lost take the same total number of life-years lost by the exposed population and divide them by different denominators. The *ex ante* measure divides the total life-years lost by the total population exposed; the *ex post* measure 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 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 ages 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 (96,947 individuals) were first calculated under the assumptions in the World Health Organization (WHO) 1996 report. 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 effects observed throughout the fifteen year exposure period can be summed and attributed (for mathematical convenience) to the 15$^{th}$ year of exposure; that individuals at the beginning of

[8] 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.

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, while the expected life-years lost per affected individual (i.e., for each of the 7,646 expected PM-related deaths) is 16.44 years.

## Ozone and Mortality

The literature investigating the relationship between ozone and mortality has been rapidly evolving over the last several years. Of the 31 time-series epidemiology studies identified in the literature that report quantitative results on a possible association between daily ozone concentrations and daily mortality, 25 were published or presented since 1995. These studies were conducted in various urban areas throughout the world: sixteen in the United States or Canada, nine in Europe, two in Australia, and four in Latin America. Seventeen of the studies report a statistically significant relationship between ozone and mortality, with the more recent studies tending to find statistical significance more often than the earlier studies.

While the growing body of epidemiological studies suggests that there may be a positive relationship between ozone and premature mortality, there is still substantial uncertainty about this relationship. Because the evidence linking premature mortality and particulate matter is currently stronger than the evidence linking premature mortality and ozone, it is important that models of the relationship between ozone and mortality include a measure of particulate matter as well. Because of the lack of monitoring data on fine particulates or its components, however, the measure of particulate matter used in most studies was generally either $PM_{10}$ or TSP or, in some cases, Black Smoke. If a component of PM, such as $PM_{2.5}$ or sulfates, is more highly correlated with ozone than with $PM_{10}$ or TSP, and if this component is also related to premature mortality, then the apparent ozone effects on mortality could be at least partially spurious.

Even if there is a true relationship between ozone and premature mortality, after taking particulate matter into account, there would be a potential problem of double counting in this analysis if the ozone effects on premature mortality were added to the PM effects estimated by Pope et al., 1995, because, as noted above, the Pope study does not include ozone in its model. Because of this, the potential ozone-mortality relationship is not included in the primary analysis. Instead the benefits associated with ozone reductions are estimated in a sensitivity analysis. The results of this sensitivity analysis should be reviewed with the appropriate caution, however, in view of the above-noted uncertainties surrounding a potential ozone-mortality relationship.

To synthesize the results of multiple studies on the relationship between ozone and premature mortality, a modified meta-analysis method was used. Because of differences in the averaging times used in the underlying studies (some use daily average ozone levels, while others use 1-hour daily maximum values), the meta-analysis approach was applied to the predicted mortality incidence changes estimated by each of the studies rather than to the coefficients of ozone in the C-R functions.

A study was included in the meta-analysis if it (1) is in or has been accepted by a peer-reviewed publication; (2) reports quantitative results for daily mortality and ozone (rather than for other measures such as total oxidants); (3) considers the entire population (rather than only a subset of the population) in the study location; (4) considers the whole year (rather than only a season or seasons); (5) considers all non-accidental or total mortality; (6) considers only one location (rather than a pooling of results across multiple locations); and (7) reports results from a copollutant model, that includes PM or some proxy for PM in the model with ozone, as well as some measure of temperature and season. The selection of a single result from among multiple ozone results reported in the same study was facilitated by the following three additional selection criteria: (8) PM ($PM_{10}$ or $PM_{2.5}$) is preferable to other measures of particulate matter; (9) more pollutants in the model is preferable to fewer pollutants; and (10) Poisson

regression is preferred to other specifications.[9] Nine studies were chosen using these criteria. To minimize benefits transfer problems, the meta-analysis was limited to the four of these nine studies that were conducted in the United States. Table D-2a briefly describes the four studies included in the meta-analysis.

[9] Almost all the models in the literature used Poisson regression. This final criterion was therefore included to impose consistency, if there was no other means by which to select a model from among several models in a study.

**Table D-2a**
**Studies and Results Selected for Meta-Analysis of the Relationship between Daily Mortality and Exposure to Ambient Ozone in the United States**

| Study | Study Location/ Duration | Copollutants in model | $O_3$ Concentration Measure (ppb) | Relative Risk and 95% CI for a 25 ppb Increase in $O_3$ |
|---|---|---|---|---|
| Ito and Thurston (1996) [a] | Cook County, Illinois 1985-1990 | $PM_{10}$ | average of same day and previous day 1-hr maxima | 1.016 (1.004 — 1.029) |
| Kinney et al. (1995) | Los Angeles County 1985-1990 | $PM_{10}$ | daily 1-hr max | 1.000 (0.989 — 1.010) |
| **The following studies were used to generate a single distribution for Philadelphia:** | | | | |
| Moolgavkar et al. (1995) | Philadelphia 1973-1988 | TSP, $SO_2$ | daily avg | 1.015 (1.004 — 1.026) |
| Samet et al. (1997) | Philadelphia 1974-1988 | TSP, $SO_2$, $NO_2$, Lagged CO | 2-day avg | 1.024 (1.008 — 1.039) |

[a] Relative risks derived from the ozone coefficient and standard error from the copollutant model were provided by personal communication with Dr. Kazuhiko Ito.

## Carbon Monoxide and Mortality

Research work presents some evidence that CO may be significantly linked to mortality, although it is not clear to what extent CO may have an effect independent of PM. Burnett et al. (1998) studied mortality in association with CO, $NO_2$, $O_3$, $SO_2$, coefficient of haze, TSP, sulfates and estimated $PM_{2.5}$ and $PM_{10}$ from 1980-1994 in metropolitan Toronto. In models that included the day of the week, weather, CO and one of the other pollutants, they found that daily average CO and all of the PM measures contributed a significant fraction of the daily number of non-accidental deaths. The measure for coefficient of haze had the strongest impact on the relative risk for CO. The relative risk associated with a 1.4 ppm change (i.e., 95$^{th}$ to the 5$^{th}$ CO percentile) was 1.070 in the single pollutant model; with the addition of COH, it fell to 1.043 (Burnett et al., 1998, Table 2). Nevertheless, the impact of CO is still quite large, and it is reported to occur in all seasons, age, and disease groupings. The model with the best fit included CO and TSP. With both CO and TSP in the model and using the mean levels of the pollutants reported for Toronto, CO contributed, on average, 4.7% of daily non-accidental deaths and TSP contributed 1% (Burnett et al., 1998, p. 689).

A review of three articles suggests that Burnett et al.'s results may not be consistent with other published results (Table D-2b).[10] In a model with CO and $PM_{10}$, Kinney et al. (1995, Figure 3) reported a relative risk of 1.05 for a 10 ppm CO increase (with a 95% confidence interval of 0.98-1.12). This is not statistically significant at the usual significance level of 5%, and the implied relative risk (1.007) for a 1.4 ppm change is about six times smaller than that reported by in Burnett et al's two-pollutant model.[11] Saldiva (1995, Table 4) reported a positive and significant CO regression coefficient in a model with just CO. Estimated at the mean, this suggests a relative risk of 1.039 per 1.4 ppm of CO, or about half the size of that reported in Burnett et al.'s single pollutant model (RR = 1.070).[12] Saldiva et al. also reported a model with CO along with all of the other measured

[10] A fourth study, by Gwynn, Burnett, and Thurston, cited as being submitted for publication, was not considered here.

[11] The underlying coefficient equals the logarithm of the relative risk divided by the change in pollution.

[12] The regression coefficient, β, = 1.69 (Saldiva et al., 1995, Table 4) and the mean mortality rate per day = 62.6 (1995, Table 1). Estimated mortality after reducing CO by 1.4 ppm = 60.23 deaths per day. The relative risk = (62.9/60.23) = 1.039.

pollutants: $PM_{10}$, $SO_2$, $NO_x$, and $O_3$. In this model, the $PM_{10}$ coefficient remained significant and unchanged from its single-pollutant model value, but the CO coefficient dropped substantially and became insignificant (1995, Table 4). Touloumi et al. (1996, Table 4) estimated a single pollutant model with a reported relative risk of 1.05 for a 7.6 $mg/m^3$ rise in CO. Assuming a conversion of 1 ppm = 1.145 $mg/m^3$ (U.S. EPA, 1991, Table 3-1), this suggests a relative risk (1.015) that is about five times smaller than the relative risk (1.070) in Burnett et al.'s single pollutant model value.

In 1991, the EPA (1991, p. 1-12) concluded that the results of CO epidemiological work "is suggestive, but not conclusive evidence" that CO may lead to sudden death in persons with coronary artery disease. Since that time, studies by Morris et al. (1995) and Schwartz and Morris (1995) reported that ambient CO concentrations increase the likelihood of hospitalization for cardiovascular disease. It is not unlikely that a certain fraction of these admittances will die, and thus indirectly one might estimate the impact of CO on mortality. However, there does not appear to be a study from which one may develop with confidence a C-R function to directly estimate CO-related mortality.[13] The results from Burnett et al. (1998) suggest that CO may have an effect on mortality independent of other pollutants, but it is premature to base an estimate of CO-related mortality with the relative risk published in their study.

[13]This difficulty may be related in part to the highly variable CO concentrations that are typically found in an urban area.

**Table D-2b**
**Selected Studies and Results for Carbon Monoxide and Mortality**

| Study | Location and Period | Population | Endpoint | Pollutants | Main Findings | Comment |
|---|---|---|---|---|---|---|
| Burnett et al. (1998) | Toronto, Canada 1980-1994 | All ages, metropolitan Toronto | non-accidental mortality | CO,$NO_2$, $SO_2$, $O_3$, $SO_4$, TSP, COH, $PM_{10}$, $PM_{2.5}$ | Significant CO effect found in all two pollutant models. Controlling for CO, significant effect found for $SO_4$, TSP, COH, $PM_{10}$, and $PM_{2.5}$. | Association with cardiac-related mortality is stronger, but CO is also significantly related to non-cardiac mortality. $PM_{10}$ and $PM_{2.5}$ estimated from $SO_4$, TSP, and COH. |
| Kinney et al. (1995) | Los Angeles County 1985-1990 | All ages | non-accidental mortality | CO, $O_3$, $PM_{10}$ | In single pollutant models, CO significant, and $PM_{10}$ and $O_3$ are marginally significant. In model with CO and $PM_{10}$, both CO and $PM_{10}$ are not significant. | Magnitude of single pollutant CO relationship drops modestly with inclusion of $PM_{10}$. |
| Saldiva et al. (1995) | Sao Paulo, Brazil 1990 to 1991 | Elderly (+65 years) | mortality from natural causes | CO, $O_3$, $PM_{10}$, $SO_2$, $NO_x$ | CO significant in single pollutant model. CO not significant in model with all other pollutants. | |
| Touloumi et al. (1996) | Athens, Greece 1987-1991 | All ages | total mortality | CO, $SO_2$, black smoke | CO, $SO_2$, and black smoke significant in single pollutant models. | Deaths during a one month summertime heat wave were excluded from analysis |

## Post-Neonatal Mortality

In a recent study of four million infants in 86 U.S. metropolitan areas, Woodruff et al. (1997) linked $PM_{10}$ exposure in the first two months of an infant's life with the probability of dying between the ages of 28 days and 364 days. In addition to the work by Woodruff et al., recent work in Mexico City (Loomis et al., 1999), the Czech Republic (Bobak and Leon, 1992), Sao Paulo (Pereira et al., 1998; Saldiva et al., 1994), and Beijing (Wang et al., 1997) provides additional evidence that particulate levels are significantly related to infant or child mortality, low birth weight or intrauterine mortality (Table D-3).

Conceptually, neonatal or child mortality could be added to the premature mortality predicted by Pope et al. (1995), because the Pope function covers only the population over 30 years old. Predicted neonatal mortality could not be added to the premature mortality predicted by the daily (short-term exposure) mortality studies, however, because these studies cover all ages. The EPA Clean Air Council recently advised the Agency not to include post-neonatal mortality in this analysis because the study is of a new endpoint and the results have not been replicated in other studies (U.S. EPA, 1999, p. 12). The estimated avoided incidences of neonatal mortality are estimated and presented as a sensitivity analysis, but are not included in the aggregate benefits analysis results.

**Table D-3**
**Studies and Results Selected for Adverse Effects in Fetuses, Infants, and Young Children**

| Study | Location and Period | Population | Endpoint | Pollutants | Main Findings |
|---|---|---|---|---|---|
| Bobak and Leon (1992) | 45 of 86 administrative districts in the Czech Republic 1986-1988 | neonates (0-1 month); post-neonates (1-12 months) | all-cause mortality; respiratory mortality | TSP, $SO_2$, $NO_x$ | Controlling for $SO_2$ and $NO_x$, TSP linked to all-cause and respiratory post-neonatal mortality; weaker, insignificant effect found for neonatal. Controlling for TSP and $SO_2$, $NO_x$ marginally significant for all-cause and respiratory post-neonatal mortality; no effect for neonatal mortality. No effect found for $SO_2$. |
| Loomis et al. (1999) | southwestern Mexico City 1/93-7/95 | infants <1 year old | all cause mortality | $PM_{2.5}$, $O_3$, $NO_2$, $SO_2$ | $PM_{2.5}$ and $NO_2$ significant in single pollutant models. $PM_{2.5}$ and $NO_2$ both not significant in two pollutant model; $PM_{2.5}$ coefficient changed little from single pollutant; $NO_2$ coefficient dropped substantially. $O_3$ not significant. $SO_2$ not analyzed since ambient levels were negligible. |
| Pereira et al. (1998) | Sao Paulo, Brazil 1/91-12/92 | fetuses over 28 weeks of pregnancy age | intrauterine mortality | $PM_{10}$, $O_3$, $NO_2$, $SO_2$, CO | In single pollutant models, $NO_2$, $SO_2$, and CO significantly related to intrauterine mortality. $PM_{10}$ and $O_3$ not significant. Considering all pollutants simultaneously, $NO_2$ is the only significant pollutant. |
| Ritz and Yu (1999) | Los Angeles, CA 1989-1993 | gestational age 37-44 weeks | low birth weight | CO | Average CO exposure in the last trimester associated with low birth weight. |
| Saldiva et al. (1994) | Sao Paulo, Brazil 5/90-4/91 | children <5 | respiratory mortality | $PM_{10}$, $O_3$, $NO_x$, $SO_2$, CO | $NO_x$ significantly related to respiratory mortality. No effect found for the other pollutants. |
| Wang et al. (1997) | Beijing, China 1988-1991 | gestational age 37-44 weeks | low birth weight | TSP, $SO_2$ | TSP and $SO_2$ exposure in the final trimester significantly related to low birth weight. Both pollutants highly correlated (r=0.92). |
| Woodruff et al. (1997) | 86 metropolitan areas in the U.S. 1989-1991 | post-neonates (1-12 months) | all-cause mortality; respiratory mortality | $PM_{10}$ | $PM_{10}$ exposure in the first two months of life significant for all-cause mortality. $PM_{10}$ significant for respiratory mortality in average birth-weight infants, but not low birth-weight infants. |
| Xu et al. (1995a) | Beijing, China 1988 | 25,370 pregnant women | pre-term delivery | TSP, $SO_2$ | TSP and $SO_2$ exposure significant for pre-term delivery. |

## Chronic Illness

There are a limited number of studies that have estimated the impact of air pollution on chronic bronchitis (Table D-4). An important hindrance is the lack of health data and the associated air pollution levels over a number of years. Schwartz (1993) and Abbey et al. (1995; 1993) provide evidence that PM exposure over a number of years gives rise to the development of chronic bronchitis in the U.S., and a recent study by McDonnell et al. (1999) provides evidence that ozone exposure is linked to the development of asthma in adults. These results are consistent with research that has found chronic exposure to pollutants leads to declining pulmonary functioning (Abbey et al., 1998; Ackermann-Liebrich et al., 1997; Detels et al., 1991).

Schwartz (1993) examined survey data collected from 3,874 adults ranging in age from 30 to 74, and living in 53 urban areas in the U.S. The survey was conducted between 1974 and 1975, as part of the National Health and Nutrition Examination Survey, and is representative of the non-institutionalized U.S. population. Schwartz (1993, Table 3) reported chronic bronchitis prevalence rates in the study population by age, race, and gender. Non-white males under 52 years old had the lowest rate (1.7%) and white males 52 years and older had the highest rate (9.3%). The study examined the relationship between the prevalence of reported chronic bronchitis and annual levels of TSP, collected in the year prior to the survey.

Abbey et al. (1995; 1993) are part of a series of studies of an ongoing prospective cohort tracking research project that began in 1977. These two studies on the development of chronic respiratory illness are based on a ten year follow-up examination of adult Seventh-Day Adventists living in California. Abbey et al. (1993) examined 3,914 adults, and estimated the relationship between annual mean ambient TSP, ozone and $SO_2$ and the presence of certain chronic respiratory symptoms (including airway obstructive disease (AOD), chronic bronchitis, and asthma) that were not present at the beginning of the study. TSP was significantly linked to new cases of AOD and chronic bronchitis, but not to asthma or the severity of asthma. Ozone was not linked to the incidence of new cases of any endpoint, but ozone was linked to the severity of asthma. No effect was found for $SO_2$. Abbey et al. (1995) examined the relationship between estimated $PM_{2.5}$ (annual mean from 1966 to 1977), $PM_{10}$ (annual mean from 1973 to 1977) and TSP (annual mean from 1973 to 1977) and the same chronic respiratory symptoms in a sample population of 1,868 Californian Seventh-Day Adventists. In this single-pollutant study, there was a statistically significant $PM_{2.5}$ relationship with development of chronic bronchitis, but not for AOD or asthma; $PM_{10}$ was significantly associated with chronic bronchitis and AOD; and TSP was significantly associated with all cases of all three chronic symptoms.

The McDonnell et al. (1999) study used the same cohort of Seventh-Day Adventists, and examined the association between air pollution and the onset of asthma in adults between 1977 and 1992. Males who did not report doctor-diagnosed asthma in 1977, but reported it in 1987 or 1992, had significantly higher ozone exposures, controlling for other covariates; no significant effect was found between ozone exposure and asthma in females. No significant effect was reported for females or males due to exposure to PM, $NO_2$, $SO_2$, or $SO_4$.

We estimate the changes in the new cases of chronic bronchitis using the studies by Schwartz (1993), Abbey et al. (1993), and Abbey et al. (1995); also, we estimate the onset of asthma in adult males using the work by McDonnell et al. (1999). The Schwartz study is somewhat older and uses a cross-sectional design; however, it is based on a national sample, unlike the Abbey et al. studies which are based on a sample of California residents who were non-smokers. We first pool the estimates from the two studies by Abbey et al. – since they are based on the same sample population and simply use different measures of PM – and then pool this estimate with that from Schwartz.

The Abbey et al. (1995; 1993) studies are based on the incidence of new cases of chronic bronchitis,

however, Schwartz (1993) is based on the *prevalence* of chronic bronchitis, not its *incidence*. To use Schwartz's study and still estimate the change in incidence, there are at least two possible approaches. The first is to simply assume that it is appropriate to use the baseline *incidence* of chronic bronchitis in a C-R function with the estimated coefficient from Schwartz's study, to directly estimate the change in incidence. The second is to estimate the percentage change in the prevalence rate for chronic bronchitis using the estimated coefficient from Schwartz's study in a C-R function, and then to assume that this percentage change applies to a baseline incidence rate obtained from another source. (That is, if the prevalence declines by 25 percent with a given decrease in PM, then baseline incidence drops by 25 percent with the same drop in PM). This analysis uses the latter approach, and estimates the change in incidence by first estimating the percentage change in prevalence.

**Table D-4**
**Summary of Selected Studies for Chronic Illness**

| Study | Location and Period | Population | Endpoint | Pollutants | Main Findings | Comment |
|---|---|---|---|---|---|---|
| Abbey et al. (1993) | California initial survey: 1977 final survey: 1987 | 3,914 Seventh Day Adventists | AOD; chronic bronchitis; asthma | TSP, $O_3$, $SO_2$ | TSP linked to new cases of AOD and chronic bronchitis, but not to asthma or the severity of asthma. $O_3$ not linked to the incidence of new cases of any endpoint, but $O_3$ was linked only to the severity of asthma. No effect found for $SO_2$. | Emphysema, chronic bronchitis, and asthma comprise AOD. |
| Abbey et al. (1995) | California initial survey: 1977 final survey: 1987 | 1,868 Seventh Day Adventists | AOD; chronic bronchitis; asthma | $PM_{2.5}$ | $PM_{2.5}$ related to new cases of chronic bronchitis, but not to new cases of AOD or asthma. | $PM_{2.5}$ estimated from visibility data. |
| Chapman et al. (1985) | 4 Utah communities 1976 | 5,623 young adults | persistent cough and phlegm | $SO_2$, $SO_4$, $NO_3$, TSP | Persistent cough and phlegm is higher in the community with higher $SO_2$, $SO_4$, and TSP concentrations. | |
| McDonnell et al. (1999) | California initial survey: 1977 final survey: 1992 | 3,091 Seventh Day Adventists | asthma | $O_3$, $PM_{10}$,, $SO_4$, $SO_2$, $NO_2$ | Single pollutant models: $O_3$ significantly linked to new asthma cases in males, but not in females; other pollutants not significantly linked to new asthma cases in males or females. Two pollutant models estimated for ozone with another pollutant; little impact found on size of ozone coefficient. | Average pollution level from 1973-1992 used. Prior to 1987, $PM_{10}$ estimated from TSP. |
| Portney and Mullahy (1990) | Nationwide sample from the 1979 U.S. National Health Interview Survey | 1,318 persons age 17-93 | sinusitis, hay fever, AOD | $O_3$, TSP | Controlling for TSP, $O_3$ significantly related to the initiation (or exacerbation) of sinusitis and hay fever; no effect on AOD. TSP not significantly related to any endpoint, although it is marginally significant for AOD. | |

| Study | Location and Period | Population | Endpoint | Pollutants | Main Findings | Comment |
|---|---|---|---|---|---|---|
| Schwartz (1993) | Nationwide sample from the National Health and Nutrition Examination Survey 1974-1975 | 6,138 individuals ages 30-74 | chronic bronchitis; asthma; shortness of breath (dyspnea); respiratory illness | TSP | TSP significantly related to the prevalence of chronic bronchitis, and marginally significant for respiratory illness. No effect on asthma or dyspnea. | Respiratory illness defined as a significant condition, coded by an examining physician as ICD8 code (460-519) |
| Xu et al. (1993) | Beijing, China Survey conducted August-September 1986 | 1,576 never smokers | chronic bronchitis; asthma | TSP; $SO_2$ | Chronic bronchitis significantly higher in the community with the highest TSP level. TSP not linked to the prevalence of asthma. | |
| Zemp et al. (1999) | Eight sites in Switzerland 1991 | 9,651 individuals ages 18-60 | chronic phlegm, chronic cough, breathlessness , asthma, dyspnea on exertion | TSP, $PM_{10}$, $NO_2$, $O_3$ | Single pollutant models: $PM_{10}$ and $NO_2$ significantly associated with chronic phlegm, chronic cough or phlegm, breathlessness and dyspnea. Similar though less significant associations found for TSP. No significant effect found for $O_3$. | |

## Hospital Admissions

There is a wealth of epidemiological information on the relationship between air pollution and hospital admissions for various respiratory and cardiovascular diseases; in addition, some studies have examined the relationship between air pollution and emergency room (ER) visits. Because most emergency room visits do not result in an admission to the hospital -- the majority of people going to the ER are treated and return home -- we treat hospital admissions and ER visits separately, taking account of the fraction of ER visits that do get admitted to the hospital, as discussed below.

Hospital admissions require the patient to be examined by a physician, and on average may represent more serious incidents than ER visits (Lipfert, 1993, p. 230). The two main groups of hospital admissions estimated in this analysis are respiratory admissions and cardiovascular admissions. There is not much evidence linking air pollution with other types of hospital admissions. The only types of ER visits that have been linked to air pollution in the U.S. or Canada are asthma-related visits.

To estimate the number of hospital admissions for respiratory illness, we pool the incidence estimates from a variety of U.S. and Canadian studies, using a random effects weighting procedure. These studies differ from each other in two important ways: (1) Some studies considered people of all ages while others considered only people ages 65 and older; and (2) The International Classification of Diseases - 9th revision (ICD-9) codes included in studies of respiratory hospital admissions and air pollution vary substantially.

The broadest classification used (for example, in Schwartz, 1996) includes ICD-9 codes 460-519. Other studies, however, considered only subsets of the broader classification. For example, Burnett et al. (1997b) consider ICD-9 codes 466, 480-486, 490-494, and 496. The correct set of ICD codes for this study is difficult to determine. If the broadest category (460-519) is too broad, including respiratory illnesses that are not linked to air pollution, we would expect the estimated pollutant coefficients to be biased downward; however, they would be used in combination with a larger baseline incidence in estimating changes in respiratory hospital admissions associated with changes in pollutant concentrations. If the broadest category is correct (i.e., if all the respiratory illnesses included are actually associated with air pollution), then studies using only subsets of ICD codes within that category would presumably understate the change in respiratory hospital admissions. It is likely, however, that all the studies have included the most important respiratory illnesses, and that the impact of differences in the definition of "all respiratory illnesses" may be less than that of other study design characteristics. We therefore treat each study equally, at least initially, in the pooling process, assuming that each study gives a reasonable estimate of the impact of air pollution on respiratory hospital admissions.

There are several steps in our estimation process:

- Develop study-specific estimates of respiratory admissions incidence change;
- Develop C-R functions for each pollutant in a model from a given study: e.g., Burnett et al. (1997b) included $PM_{2.5-10}$, $O_3$, $NO_2$, and $SO_2$ in their final model for respiratory admissions (ICD-9 codes 464-466, 480-486, 490-494, 496);
- Estimate the change in incidence associated with the change in each air pollutant considered in the model, and aggregate these incidence changes across the pollutants in the model: e.g., for Burnett et al. (1997b) we sum the incidence changes associated with $PM_{2.5-10}$, $O_3$, $NO_2$, and $SO_2$;
- If a study estimated separate models for non-overlapping respiratory illness categories, sum the estimated incidence changes across these non-overlapping categories: e.g., Delfino et al. (1994) estimated two separate models: one for asthma (ICD code 493) and one for all respiratory non-asthma (ICD codes 462-466,

480-487, 490-492, 494, and 496); we estimated and summed incidences for these two categories.

Aggregate estimates across non-overlapping age categories:

- Seven studies estimated C-R functions for respiratory admissions for people ages 65 and older. One study, Sheppard et al. (1999), estimated a C-R function for asthma only for people under 65. Using a Monte Carlo procedure, we aggregate the results from the Sheppard study with those from each of the over-65 respiratory admissions studies.

Pool estimates of respiratory hospital admissions changes:

- Four studies estimated C-R functions for respiratory admissions for people of all ages. With the seven "all ages" estimates developed in step 2, there are eleven separate estimates of the change in respiratory hospital admissions associated with a change in air pollutant concentrations. Using Monte Carlo procedures, the results of these eleven studies are pooled.

Table D-5 summarizes the studies used in estimating respiratory admissions; Table D-6 provides more detailed information on these studies, and other studies that were not chosen for this analysis.

Similar issues of definition arise for cardiovascular hospital admissions. The broadest classification we have seen in the epidemiological literature includes ICD codes 390-429 (see, for example, Schwartz, 1999). Some studies, however, use a much more narrow definition, including only subsets of the larger group of ICD codes. We use a similar procedure for cardiovascular admissions as we used for respiratory hospital admissions. Table D-7 summarizes the studies used in estimating cardiovascular admissions; Table D-8 provides more detailed information on these studies, and other studies that were not chosen this analysis.

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

Table D-9 summarizes the studies used in estimating ER visits for asthma; Tables D-10 and D-11 provide more detailed information on these studies and other ER studies that were not used in the analysis.

**Table D-5**
**Studies Used to Develop Respiratory Admissions Estimates**

| Location | Study | Endpoints Estimated [a] (ICD code) | Pollutants Used in Final Model | Study Population |
|---|---|---|---|---|
| Toronto, Canada | Burnett et al. (1997b) | all respiratory (464-466, 480-486, 490-494, 496) | $PM_{2.5-10}$, $O_3$, $NO_2$, $SO_2$ | all ages |
| Toronto, Canada | Burnett et al. (1999) | asthma (493); respiratory infection (464, 466, 480-487, 494); non-asthma COPD (490-492, 496) | $O_3$, CO, $PM_{2.5-10}$ (asthma); $O_3$, $NO_2$, $PM_{2.5}$ (respiratory infection); $O_3$, CO, $PM_{2.5-10}$ (COPD). | all ages |
| Toronto, Canada | Thurston et al. (1994) | all respiratory (466, 480-482, 485, 490-493) | $O_3$, $PM_{2.5}$ | all ages |
| Minneapolis-St. Paul, MN | Moolgavkar et al. (1997) | pneumonia (480-487); COPD (490-496) | $O_3$, $SO_2$, $NO_2$, $PM_{10}$ (pneumonia); $O_3$, CO, $PM_{10}$ (COPD) | >64 |
| Minneapolis-St. Paul, MN | Schwartz (1994c) | pneumonia (480-486); COPD (490-496) | $O_3$, $PM_{10}$ (pneumonia); $PM_{10}$ (COPD) | >64 |
| Birmingham, AL | Schwartz (1994a) | pneumonia (480-487); COPD (490-496) | $PM_{10}$ | >64 |
| Detroit, MI | Schwartz (1994b) | pneumonia (480-486); non-asthma COPD (491-492, 494-496) | $O_3$, $PM_{10}$ | >64 |
| Spokane, WA | Schwartz (1996) | all respiratory (460-519) | $PM_{10}$ | >64 |
| New Haven, CT | Schwartz (1995) | all respiratory (460-519) | $O_3$, $PM_{10}$ | >64 |
| Tacoma, WA | Schwartz (1995) | all respiratory (460-519) | $O_3$, $PM_{10}$ | >64 |
| Seattle, WA | Sheppard et al. (1999) | asthma (493) | CO, $PM_{2.5}$ | <65 |

[a] Monetized benefits of non-overlapping endpoints within each study are aggregated. Monetized benefits for asthma among people age <65 (Sheppard et al., 1999) are aggregated with the benefits in studies of people age >64.

**Table D-6**
**Summary of Hospital Admissions Studies – Respiratory Illnesses**

| Study | Location and Period | Population | Endpoint | Pollutants [a] | Main Findings | Comment |
|---|---|---|---|---|---|---|
| Anderson et al. (1997) | Barcelona, Paris Amsterdam, Rotterdam, Milano Period varies by city from 5-13 years | all ages; >64 | COPD (490-492, 496) | $NO_2$, BS (black smoke), TSP, $SO_2$, $O_3$ | COPD: Single pollutant models: meta-analysis of city specific results found significant effect for BS, $NO_2$, $O_3$, and $SO_2$ in the all age group; similar results reported for ages >64. Strongest effect found for $O_3$. TSP not significant in meta-analysis. For a given pollutant, results varied considerably by city. | *Study not used to estimate incidence: study outside U.S. and Canada.* |
| Burnett et al. (1995) | southern Ontario, Canada 1/83-12/88 | <65; >64 | all respiratory (466, 480-486, 490-494, 496) | $SO_4$, $O_3$ | All respiratory: $SO_4$ significantly related to respiratory admissions in ages <65 and >64. $O_3$ significant impact from May-September; no effect the rest of the year. | May-September results also discussed in Burnett et al. (1994). *Study not used to estimate incidence: no study specific conversion available between* $SO_4$ *and* $PM_{2.5}$ *or* $PM_{10}$. |
| Burnett et al. (1997b) | Toronto, Canada summers in 1992-1994 | all ages | all respiratory (464-466, 480-486, 490-494, 496) | $O_3$, CO, $NO_2$, $SO_2$, COH (coefficient of haze), $H^+$, $SO_4$, $PM_{2.5}$, $PM_{2.5-10}$, $PM_{10}$, | All respiratory: COH and $O_3$ linked to respiratory admissions; other PM measures less strongly linked. Two pollutant models: CO, $NO_2$, and $SO_2$ not significant, controlling for COH; $O_3$ significant, controlling for COH. Four pollutant models: COH and $O_3$ significant; no effect for $NO_2$ and $SO_2$; other PM measures not significant, controlling for $O_3$, $NO_2$, and $SO_2$. | *Four pollutant model ($PM_{2.5-10}$, $O_3$, $NO_2$, and $SO_2$) used to estimate all respiratory incidence.* |
| Burnett et al. (1997a) | 16 Canadian cities 3/81-12/91 | <65; >64 | all respiratory (466, 480-486, 490-494, 496) | $O_3$, CO, $SO_2$, $NO_2$, COH | All respiratory: Multiple pollutant models: $O_3$ significantly related to admissions, controlling for CO and COH; significant effect also reported for CO and COH; no significant effect found for $NO_2$ and $SO_2$ after controlling for $O_3$ and CO. Montreal and Vancouver decreased the size of the effect of $O_3$ substantially. $O_3$ significant with and without these cites in the model. | *Study not used to estimate incidence: no study specific conversion available between COH and* $PM_{2.5}$ *or* $PM_{10}$. |

| Study | Location and Period | Population | Endpoint | Pollutants [a] | Main Findings | Comment |
|---|---|---|---|---|---|---|
| Burnett et al. (1999) | Toronto, Canada 1980-1994 | all ages | asthma (493); respiratory infection (464, 466, 480-487, 494); COPD (490-492, 496) | $O_3$, CO, $NO_2$, $SO_2$, $PM_{2.5}$, $PM_{2.5-10}$, $PM_{10}$, | Multiple pollutant models estimated, where pollutants for best fitting model chosen using stepwise regression based on AIC criterion. Asthma: $O_3$, CO, $PM_{2.5-10}$ significantly related to asthma admissions; other pollutants not chosen in stepwise regression. Respiratory infection: $O_3$, $NO_2$, and $PM_{2.5}$ chosen in stepwise regression COPD: $O_3$ and $PM_{2.5-10}$ chosen in stepwise regression. | $PM_{2.5}$, $PM_{2.5-10}$, and $PM_{10}$ estimated from TSP, COH, and $SO_4$ data. *Multiple pollutant models used to estimate incidence of: asthma (*$O_3$, CO, $PM_{2.5-10}$*), respiratory infection (*$O_3$, $NO_2$, $PM_{2.5}$*), and COPD (*$O_3$, CO, $PM_{2.5-10}$*).* |
| Delfino et al. (1994) | Montreal, Canada May-October in 1984-1988. July-August subset used to examine all respiratory admissions. | all ages | asthma (493); all respiratory (462-466, 480-487, 490-494, 496); all respiratory non-asthma | $O_3$, $SO_4$, $PM_{10}$ | Asthma: Two pollutant model: marginally significant effect for $PM_{10}$, controlling for $O_3$. No effect for $O_3$ and $SO_4$. All respiratory and all respiratory non-asthma: $PM_{10}$ suggestive but not significant, after controlling for temperature. Significant link between all respiratory non-asthma and $SO_4$. No effect for $O_3$. | $SO_4$ and $PM_{10}$ were both estimated from COH and other variables. *Study not used to estimate incidence* |
| Lipfert and Hammerstrom (1992) | Southern Ontario, Canada January-February and July-August in 4/79-3/85 | all ages | all respiratory (466, 480-482, 485, 490-493) | $O_3$, $SO_4$, $NO_4$, $SO_4$, COH, TSP | All respiratory: $SO_2$, $SO_4$, and $O_3$ found to be significant predictors of respiratory admissions in July-August. | *Study not used to estimate incidence: estimated coefficients not reported.* |

| Study | Location and Period | Population | Endpoint | Pollutants [a] | Main Findings | Comment |
|---|---|---|---|---|---|---|
| Moolgavkar et al. (1997) | Minneapolis-St. Paul, MN; Birmingham, AL 1/86-12/91 | >64 | pneumonia (480-487); COPD (490-496); all respiratory (480-487, 490-496) | $O_3$, CO, $SO_2$, $NO_2$, $PM_{10}$ | Pneumonia: Four pollutant model: $O_3$ significant ($NO_2$, $SO_2$, and $PM_{10}$ not significant) in Minneapolis-St. Paul; no significant effect found for any pollutant in Birmingham. COPD: No significant effect found in Birmingham or Minneapolis-St. Paul for any pollutant. All respiratory: Single pollutant models: $O_3$, $NO_2$, and $PM_{10}$ significant in Minneapolis-St.Paul. Multiple pollutant models (results presented in graph): $O_3$ significant, controlling for other pollutants; $PM_{10}$ significant controlling for $O_3$, but not significant controlling for $O_3$, $SO_2$, and $NO_2$ together. No significant effect found in Birmingham for admissions with $O_3$, CO, or $PM_{10}$; $NO_2$ and $SO_2$ data not available for Birmingham. | *Multiple pollutant models used to estimate pneumonia incidence ($O_3$, $SO_2$, $NO_2$, $PM_{10}$) and COPD incidence ($O_3$, CO, $PM_{10}$) in Minneapolis-St. Paul. No model estimated for Birmingham: coefficients and standard errors not reported.* |
| Morgan et al. (1998) | Sydney, Australia 1/90-12/94 | 1-14; 15-64; >64 | asthma; COPD (490-492,494 496) | $O_3$, $NO_2$, bscat (measure of light scattering) | Asthma: Single pollutant models: $NO_2$ significant for ages 1-14 but not other age groups. $O_3$ and bscat not significant for any age group. Three pollutant model: $NO_2$ remains significantly related to asthma admission in ages 1-14. COPD: No pollutant significantly related to COPD admissions. | *Study not used to estimate incidence: study outside U.S. and Canada.* |
| Pantazopolou et al. (1995) | Athens, Greece 1988 | all ages | all respiratory (not defined by ICD code) | BS, CO, $NO_2$ | All respiratory: Single-pollutant models: BS, CO, $NO_2$ significantly related to respiratory admissions in the winter time. No significant effect found any pollutant in the summer. | *Study not used to estimate incidence: study outside U.S. and Canada.* |
| Ponce de Leon et al. (1996) | London, England 4/87-2/92 | 0-14; 15-64; >64 | all respiratory (460-519) | BS, $SO_2$, $O_3$, $NO_2$ | All respiratory: $O_3$ significantly related to admissions in ages >14. No significant effect found for $SO_2$, $O_3$, and $NO_2$. | *Study not used to estimate incidence: study outside U.S. and Canada.* |
| Ponka and Virtanen (1994) | Helsinki, Finland 1/87-12/89 | <65; >64 | COPD (491-492) | $NO_2$, $SO_2$, $O_3$, TSP | COPD: Single pollutant models: $SO_2$ linked to (491-492) admissions in ages <65; $NO_2$ linked to admissions in ages >64; no significant effect seen for $O_3$ and TSP. | *Study not used to estimate incidence: study outside U.S. and Canada.* |

| Study | Location and Period | Population | Endpoint | Pollutants [a] | Main Findings | Comment |
|---|---|---|---|---|---|---|
| Schwartz (1994a) | Birmingham, AL 1/86-12/89 | >64 | pneumonia (480-487); COPD (490-496) | $PM_{10}$, $O_3$ | Pneumonia: $PM_{10}$ significant and $O_3$ not significant in single pollutant models. COPD: $PM_{10}$ significant and $O_3$ not significant in single pollutant models. | *Single pollutant models ($PM_{10}$) used to estimate pneumonia incidence and COPD incidence.* |
| Schwartz (1994b) | Detroit, MI 1/86-12/89 | >64 | asthma (493); pneumonia (480-486); non-asthma COPD (491-492, 494-496) | $PM_{10}$, $O_3$ | Asthma: admissions not associated with either pollutant; coefficients and standard errors not reported. Pneumonia: Two pollutant model: $PM_{10}$ and $O_3$ both significant for pneumonia. Non-asthma COPD: Two pollutant model: $PM_{10}$ and $O_3$ both significant. | *Two pollutant models ($PM_{10}$ and $O_3$) used to estimate pneumonia incidence and non-asthma COPD incidence.* |
| Schwartz (1996) | Spokane, WA 1/88-12/90 | >64 | pneumonia (480-487); COPD (490-496); all respiratory (460-519) | $PM_{10}$, $O_3$ | Pneumonia: $PM_{10}$ marginally significant and $O_3$ not significant for pneumonia in single pollutant models. COPD: $PM_{10}$ significant and $O_3$ not significant in single pollutant models. All respiratory: Single pollutant models: $PM_{10}$ and $O_3$ both significant. Two pollutant model not estimated because of limited overlap between $PM_{10}$ and $O_3$ data. | *Single pollutant model ($PM_{10}$) used to estimate all-respiratory incidence.* |
| Schwartz (1994c) | Minneapolis-St. Paul, MN 1/86-12/89 | >64 | pneumonia (480-486); COPD (490-496) | $PM_{10}$, $O_3$ | Pneumonia: Two pollutant model: $PM_{10}$ significantly related to pneumonia; $O_3$ weakly linked to pneumonia. COPD: Single pollutant models: $PM_{10}$ significant and $O_3$ not significant. | *Two pollutant model ($PM_{10}$, $O_3$) used to estimate pneumonia incidence; single pollutant model ($PM_{10}$) used to estimate COPD incidence.* |
| Schwartz (1995) | New Haven, CT; Tacoma, WA 1/88-12/90 | >64 | all respiratory (460-519) | $PM_{10}$, $O_3$, $SO_2$ | All respiratory: Single pollutant models: $PM_{10}$, $O_3$, $SO_2$ significant, except $O_3$ in New Haven. Two pollutant model results varied by city: $O_3$ significant (3 of 4 models) and stable coefficient estimates $PM_{10}$ significant (3 of 4 models), but less stable estimates. $SO_2$ significant (1of 4 models). | *Two pollutant model ($PM_{10}$, $O_3$) used to estimate all respiratory incidence.* |

| Study | Location and Period | Population | Endpoint | Pollutants [a] | Main Findings | Comment |
|---|---|---|---|---|---|---|
| Sheppard et al. (1999) | Seattle, WA 1/87-12/94 | <65 | asthma (493) | CO, $SO_2$, $O_3$, $PM_{2.5}$, $PM_{2.5-10}$, $PM_{10}$ | Asthma: Single pollutant models: each pollutant significantly related to asthma, except $SO_2$. Multiple pollutant models: $PM_{2.5}$ and CO reported to have best fit in models without $O_3$. Both $PM_{2.5}$ and CO are significant when included together in a model. | In most years, $O_3$ data was available only from April through October. $O_3$ reported to have the best fit, but authors did not consider $O_3$ further because of limited data. *Two pollutant model (CO, $PM_{2.5}$) used to estimate asthma incidence.* |
| Spix et al. (1998) | London, Amsterdam, Rotterdam, Paris, Milano Period varies by city from 5-13 years | 15-64; >64 | all respiratory (460-519) | $NO_2$, $SO_2$, $O_3$, TSP, BS | All respiratory: Single pollutant models: $O_3$ significantly related to admissions in ages 15-64 and >64. BS significantly related to admissions in ages 15-64. $SO_2$ significantly related to admissions in ages >64. | *Study not used to estimate incidence: study outside U.S. and Canada.* |
| Sunyer et al. (1997) | Barcelona, Helsinki, London, Paris Period varies by city from 3-6 years | 0-14; 15-64 | asthma | $NO_2$, $SO_2$, $O_3$, BS | Asthma: Two pollutant models: $NO_2$ significant in ages 15-64, controlling for BS; $NO_2$ had no effect on ages 0-14. $SO_2$ significant in ages 0-14, controlling for either BS or $NO_2$; $SO_2$ had no effect on ages 15-64. | *Study not used to estimate incidence: study outside U.S. and Canada.* |
| Tenias et al. (1998) | Valencia, Spain 1/93-12/95 | >14 | asthma | $NO_2$, $SO_2$, $O_3$, BS | Asthma: Two pollutant models: $O_3$ and $NO_2$ both significant. No significant effect found for $SO_2$ and BS. | *Study not used to estimate incidence: study outside U.S. and Canada.* |
| Thurston et al. (1994) | Toronto, Canada six weeks in July and August 1986-1988 | all ages | asthma (493); all respiratory (466, 480-482, 485, 490-493) | $H^+$, $SO_4$, $O_3$, $PM_{2.5}$, $PM_{2.5-10}$, $PM_{10}$, TSP | Asthma: Single pollutant models: $O_3$, $H^+$, $SO_4$, $O_3$, and TSP linked to all respiratory admissions; $PM_{2.5}$, $PM_{2.5-10}$, $PM_{10}$ not significant. Two pollutant models: $O_3$ significant, but PM measures no longer significant. Best fitting PM measure is $H^+$. All respiratory: Single pollutant models: $O_3$ and various measures of PM linked to all respiratory admissions. Two pollutant models: with $O_3$ and PM together, $O_3$ still significant, but PM often not significant (only $H^+$ significant). | *Two pollutant model ($O_3$, $PM_{2.5}$) used to estimate all respiratory incidence.* |

| Study | Location and Period | Population | Endpoint | Pollutants [a] | Main Findings | Comment |
|---|---|---|---|---|---|---|
| Thurston et al. (1992) | Buffalo, NY; New York City June-August in 1988-1989 | all ages | all respiratory (466, 480-486, 490-493) | $H^+$, $SO_4$, $O_3$ | All respiratory: Three pollutant model: $H^+$, $SO_4$, and $O_3$ are all significant. This result is found in both Buffalo and New York City. | *Study not used to estimate incidence: no study specific conversion available between study pollutants ($H^+$ and $SO_4$) and $PM_{2.5}$ or $PM_{10}$.* |
| Vigotti et al. (1996) | Milan, Italy 1/89-12/89 | 15-64; >64 | all respiratory (460-519) | TSP, $SO_2$ | All respiratory: Single pollutant models: TSP and $SO_2$ linked to admissions. | *Study not used to estimate incidence: study outside U.S. and Canada.* |

[a] Not all pollutants considered in a study are necessarily included in the model used to develop C-R functions.

**Table D-7**
**Studies Used to Develop Cardiovascular Admissions Estimates**

| Location | Study | Endpoints Estimated (ICD code) | Pollutants Used in Final Model | Study Population |
|---|---|---|---|---|
| Toronto, Canada | Burnett et al. (1997b) | cardiac (410-414, 427-428) | $O_3$, $PM_{2.5-10}$ | all ages |
| Toronto, Canada | Burnett et al. (1999) | ischemic heart disease (410-414); dysrhythmias (427); congestive heart failure (428) | $NO_2$, $SO_2$ (ischemic heart disease); $PM_{2.5}$, CO, $O_3$ (dysrhythmias); CO, $NO_2$ (heart failure incidence) | all ages |
| Detroit, MI | Schwartz and Morris (1995) | ischemic heart disease (410-414); congestive heart failure (428) | CO, $PM_{10}$ | >64 |
| Eight U.S. counties 1/88-12/90 | Schwartz (1999) | cardiovascular disease (390-429) | CO, $PM_{10}$ | >64 |
| Tucson, AZ 1/88-12/90 | Schwartz (1999) | cardiovascular disease (390-429) | CO, $PM_{10}$ | >64 |

**Table D-8**
**Summary of Hospital Admissions Studies – Cardiovascular Illnesses**

| Study | Location and Period | Population | Endpoint (ICD code) | Pollutants | Main Findings | Comment |
|---|---|---|---|---|---|---|
| Burnett et al. (1995) | southern and central Ontario, Canada 1/83--12/88 | all ages | cardiac (410, 413, 427-428) | $SO_4$, $O_3$ | Cardiac: Two pollutant model: $SO_4$ significantly related to cardiac admissions; $O_3$ not significant, in any season or over the whole year. | *Study not used to estimate incidence: no study specific conversion available between* $SO_4$ *and* $PM_{2.5}$ *or* $PM_{10}$. |
| Burnett et al. (1997b) | Toronto, Canada summers 1992-1994 | all ages | cardiac (410-414, 427-428) | $O_3$, CO, $NO_2$, $SO_2$, COH (coefficient of haze), $H^+$, $SO_4$, $PM_{2.5}$, $PM_{2.5-10}$, $PM_{10}$, | Cardiac: COH and $O_3$ linked to cardiac admissions; other PM measures less strongly linked. Two pollutant models: CO, $NO_2$, and $SO_2$ not significant, controlling for COH. $O_3$ significant, controlling for COH. Four pollutant models: COH and $O_3$ significant; no effect for $NO_2$ and $SO_2$. Other PM measures not significant, controlling for $O_3$, $NO_2$, and $SO_2$. | *Two pollutant model (*$O_3$, $PM_{2.5-10}$*) used to estimate cardiac incidence.* |
| Burnett et al. (1997c) | 10 Canadian cities 1/81-12/91 | >64 | congestive heart failure (428) | $O_3$, CO, $NO_2$, $SO_2$, COH | Congestive heart failure: Single pollutant models: CO, $NO_2$, $SO_2$, COH are significant; no effect for $O_3$. CO and $NO_2$ have the best fit. Two pollutant models: Controlling for $NO_2$, CO significant, with only small reduction in coefficient size; $NO_2$ insignificant in this model. | *Study not used to estimate incidence: limited endpoint.* |
| Burnett et al. (1999) | Toronto, Canada 1980-1994 | all ages | ischemic heart disease (410-414); dysrhythmias (427); congestive heart failure (428) | $O_3$, CO, $NO_2$, $SO_2$, $PM_{2.5}$, $PM_{2.5-10}$, $PM_{10}$, | Multiple pollutant model, where pollutants for best fitting model chosen using stepwise regression based on AIC criterion. Ischemic heart disease: $NO_2$ and $SO_2$, chosen by stepwise regression. Other pollutants not chosen. Dysrhythmias: pollu$O_3$, CO, and $PM_{2.5}$ chosen by stepwise regression. Other pollutants not chosen. Congestive heart failure: $NO_2$ and CO chosen by stepwise regression procedure. other pollutants not chosen in stepwise regression. | $PM_{2.5}$, $PM_{2.5-10}$, and $PM_{10}$ estimated from TSP, COH, and sulfate ($SO_4$) data. *Multiple pollutant models used to estimate ischemic heart disease (*$NO_2$, $SO_2$*), dysrhythmias (*$PM_{2.5}$, CO, $O_3$*), and congestive heart failure incidence (CO,* $NO_2$*).* |

| Study | Location and Period | Population | Endpoint (ICD code) | Pollutants | Main Findings | Comment |
|---|---|---|---|---|---|---|
| Morgan et al. (1998) | Sydney, Australia 1/90-12/94 | 0-64; >64 | heart disease (410,413, 427-428) | $O_3$, $NO_2$, bscat (measure of light scattering) | Single pollutant models: bscat significant for ages >64; $NO_2$ significant in ages 0-64 and >64. Three pollutant model: $NO_2$ significant in ages >64; $O_3$ and bscat not significant. | Results from three pollutant model for ages 0-64 not presented. *Study not used to estimate incidence: study outside U.S. and Canada.* |
| Morris et al. (1995) | seven U.S. cities 1/86-12/89 | >64 | congestive heart failure (428) | $O_3$, CO, $NO_2$, $SO_2$ | Single pollutant models: CO, $NO_2$, and $SO_2$ significant in single pollutant models. Four pollutant model: CO is significant in five of the seven cities; $NO_2$ is significant in one city; $SO_2$ and $O_3$ are not significant in any cities. | *Study not used to estimate incidence: no PM measure used in the study, plus limited endpoint.* |
| Morris and Naumova (1998) | Chicago, IL 1/86-12/89 | >64 | congestive heart failure (428) | $O_3$, CO, $NO_2$, $SO_2$, $PM_{10}$ | Single pollutant models: CO, $NO_2$, $SO_2$, and $PM_{10}$ significant. Five pollutant model: CO significant (CI for RR=1.03-1.12); $PM_{10}$ borderline significant (CI for RR=0.99-1.06); other pollutants not significant. | *Study not used to estimate incidence: limited endpoint.* |
| Pantazopolou et al. (1995) | Athens, Greece 1988 | all ages | cardiac (not defined by ICD code) | BS (black smoke), CO, $NO_2$ | Single pollutant models: BS, CO, $NO_2$ significantly related to cardiac admissions in the winter. No significant effect found for any pollutant in the summer. | *Study not used to estimate incidence: study outside U.S. and Canada.* |
| Schwartz and Morris (1995) | Detroit, MI 1/86-12/89 | >64 | ischemic heart disease (410-414); dysrhythmias (427); congestive heart failure (428) | $O_3$, CO, $SO_2$, $PM_{10}$ | Ischemic heart disease: Two pollutant models: $PM_{10}$ and CO both significant; no effect seen for $SO_2$ and $O_3$. Dysrhythmias: Air pollutants did not have a significant effect. Congestive heart failure: Single pollutant models: $PM_{10}$ and CO significant; $SO_2$ and $O_3$ not significant. Two pollutant models: $PM_{10}$ significant, controlling for CO and $SO_2$. Controlling for $PM_{10}$, CO significant. | *Two pollutant models ($PM_{10}$, CO) used to estimate ischemic heart disease and congestive heart failure incidence.* |

| Study | Location and Period | Population | Endpoint (ICD code) | Pollutants | Main Findings | Comment |
|---|---|---|---|---|---|---|
| Schwartz (1999) | Eight U.S. counties 1/88-12/90 | >64 | cardiovascular disease (390-429) | CO, $PM_{10}$ | Two pollutant model: CO and $PM_{10}$ both significant. | *Two pollutant model ($PM_{10}$, CO) used to estimate incidence of cardiovascular admissions.* |
| Schwartz (1997) | Tucson, AZ 1/88-12/90 | >64 | cardiovascular disease (390-429) | $O_3$, CO, $SO_2$, $NO_2$, $PM_{10}$ | In a model with the two pollutants, CO and $PM_{10}$ were both significant. No effect seen for $O_3$, $SO_2$, and $NO_2$. | *Two pollutant model ($PM_{10}$, CO) used to estimate incidence of cardiovascular admissions.* |
| Yang et al. (1998) | Reno/Sparks, NV 1/89-12/94 | all ages | cardiovascular illness (390-459) | CO | Reported significant relationship between CO and admissions. | *Study not used to estimate incidence: no PM measure used in the study.* |

**Table D-9**
**Studies Used to Develop Asthma Emergency Room Visits**

| Location | Study | Endpoints Estimated | Pollutants Used in Final Model | Study Population |
|---|---|---|---|---|
| central and northern NJ | Cody et al. (1992) | asthma | $O_3$ | all ages |
| central and northern NJ | Weisel et al. (1995) | asthma | $O_3$ | all ages |
| Seattle, WA | Schwartz et al. (1993) | asthma | $PM_{10}$ | <65 |
| St. John, New Brunswick, Canada | Stieb et al. (1996) | asthma | $O_3$ | all ages |

**Table D-10**
**Summary of Selected Studies for Emergency Room Visits -- Asthma and Acute Wheezing**

| Study | Location and Period | Population | Endpoint | Pollutants | Main Findings | Comment |
|---|---|---|---|---|---|---|
| Atkinson et al. (1999) | London, England 1/92-12/94 | 0-14; 15-64; >64; all ages | asthma | $NO_2$, BS (black smoke), $PM_{10}$, $SO_2$, CO, $O_3$ | Single pollutant models: $PM_{10}$ and $NO_2$ significantly related to asthma visits in all age groups. $SO_2$ significant in ages 0-14. BS is significant for ages 15-64. No effect seen for $O_3$. Two pollutant results only for ages 0-14: $NO_2$ and $SO_2$ significant; other pollutants not significant. | *Study not used to estimate incidence: study outside U.S. and Canada.* |
| Bates et al. (1990) | Vancouver, Canada 7/84-10/86 | 1-14; 15-60; >60 | asthma | $NO_2$, $SO_2$, $SO_4$, $O_3$ | $SO_4$ correlated with asthma in all age groups with some variation by season. $SO_2$ correlated with asthma visits in ages 15 and up. No effect found for $NO_2$ and $O_3$. | *Study not used to estimate incidence: correlations only presented.* |
| Buchdahl et al. (1996) | London, England 3/92-2/93 | <17 | acute wheezing | $NO_2$, $SO_2$, $O_3$ | $SO_2$ significantly related to acute wheezing. $O_3$ has a significant, U-shaped result, suggesting that the optimal level of ozone is not zero. $NO_2$ not significant. | *Study not used to estimate incidence: study outside U.S. and Canada.* |
| Castellsague et al. (1995) | Barcelona, Spain January-March and July-September in 1985-1989 | >14 | asthma | $NO_2$, BS, $SO_2$, $O_3$ | Single pollutant models: $NO_2$ significant in both July-September and January-March. BS linked to asthma ER visits in July-September. No significant effect found for $SO_2$ and $O_3$. | *Study not used to estimate incidence: study outside U.S. and Canada.* |
| Cody et al. (1992) | central and northern NJ May-August in 1988-1989 | all ages | asthma | $PM_{10}$, $SO_2$, $O_3$ | Two pollutant model: $O_3$ linked to asthma visits; $SO_2$ not significant. No significant effect seen for $PM_{10}$; $PM_{10}$ considered in separate analysis, because of limited (every sixth day) sampling. | *Single pollutant model ($O_3$) used to estimate incidence of asthma visits.* |
| Goldstein and Weinstein (1986) | New York City 1/69-2/72 | all ages | asthma | $SO_2$ | No significant correlation found between $SO_2$ and asthma ER visits. | *Study not used to estimate incidence: only $SO_2$ in the analysis.* |

| Study | Location and Period | Population | Endpoint | Pollutants | Main Findings | Comment |
|---|---|---|---|---|---|---|
| Lipsett et al. (1997) | Santa Clara County, CA November-January in 1988-1992 | all ages | asthma | $PM_{10}$, COH (coefficient of haze), $O_3$, $NO_2$ | Single pollutant models: $NO_2$, $PM_{10}$, and COH significant; $O_3$ not significant. Two pollutant models: $PM_{10}$ and COH linked to ER visits controlling for $NO_2$; $NO_2$ not significant. $PM_{10}$ reported to provide a slightly better fit than COH | $PM_{10}$ estimated from COH observations. *Study not used to estimate incidence: results depend on temperature interaction that we cannot model.* |
| Richards et al. (1981) | Los Angeles, CA 8/79-1/80 | children (median age =6) | asthma and bronchiolitis (92% asthma only) | COH, HC (hydrocar-bons), NO, $NO_2$, $O_3$, $SO_2$, $SO_4$, TSP | COH, HC, NO, and $NO_2$ have positive and significant correlation with ER visits; $O_3$ and $SO_2$ have negative significant correlation; $SO_4$ and TSP have insignificant correlation. | 13% of reported visits subsequently admitted to the hospital. *Study not used to estimate incidence: correlations only presented.* |
| Romieu et al. (1995) | Mexico City, Mexico 1/90-6/90 | <16 | asthma | $SO_2$, $O_3$ | Two pollutant model: $O_3$ significant and $SO_2$ marginally significant. | *Study not used to estimate incidence: study outside U.S. and Canada.* |
| Rosas et al. (1998) | Mexico City, Mexico 1991 | <15; 16-59; >59 | asthma | $O_3$, $SO_2$, $NO_2$, $PM_{10}$, TSP | Little effect found for air pollutants. Strong effect found for aeroallergens, such as grass pollen. | *Study not used to estimate incidence: study outside U.S. and Canada.* |
| Schwartz et al. (1993) | Seattle, WA 9/89-9/90 | <65; >64 | asthma | $SO_2$, $PM_{10}$, $O_3$ | Single pollutant models: $PM_{10}$ linked to ER visits in ages <65, with no effect in ages >64. No effect for $SO_2$ and $O_3$ on ER visits in either age group. | $O_3$ only available May-September. *Single pollutant model ($PM_{10}$) used to estimate incidence of asthma visits.* |
| Stieb et al. (1996) | St. John, New Brunswick, Canada May-September in 1984-1992 | 0-15; >15; all ages | asthma | $NO_2$, TSP, $SO_2$, $SO_4$, $O_3$ | $O_3$ linked to ER visits in ages >15, especially when $O_3$ levels exceed 75 ppb; $O_3$ not significant in ages 0-15. No significant effect seen for the other pollutants. | TSP and $SO_4$ gathered every sixth day. *Single pollutant model ($O_3$) used to estimate incidence of asthma visits.* |
| Weisel et al. (1995) | central and northern NJ May-August in 1986-1990 | all ages | asthma | $O_3$ | $O_3$ linked to ER visits. | *Single pollutant model used.* |

| Study | Location and Period | Population | Endpoint | Pollutants | Main Findings | Comment |
|---|---|---|---|---|---|---|
| White et al. (1994) | Atlanta, GA 6/90-8/90 | 1-16 | asthma and restrictive airway disease | $O_3$, $SO_2$, $PM_{10}$ | $O_3$ linked to ER visits when $O_3$ levels exceeded 110 ppb. No significant effect reported for $SO_2$ or $PM_{10}$. | $PM_{10}$ estimated from visibility levels. *Study not used to estimate incidence: limited study data.* |

**Table D-11**
**Summary of Selected Studies for Emergency Room Visits -- All-Cause, All-Respiratory, Chronic Obstructive Pulmonary Disease (COPD), and Bronchitis**

| Study | Location and Period | Population | Endpoint (ICD code) | Pollutants | Main Findings | Comment |
|---|---|---|---|---|---|---|
| Atkinson et al. (1999) | London, England 1/92-12/94 | 0-14; 15-64; >64; all ages | all respiratory (not defined by ICD code) | $NO_2$, BS (black smoke), $PM_{10}$, $SO_2$, CO, $O_3$ | Single pollutant models: $PM_{10}$ significant in ages 0-14 and 15-64. BS and $SO_2$ significant in ages 0-14. CO and $NO_2$ significant in ages >64. $O_3$ not significant. | *Study not used to estimate incidence: study outside U.S. and Canada.* |
| Bates et al. (1990) | Vancouver, Canada 7/84-10/86 | 1-14; 15-60; >60 | all respiratory (466, 480-486, 491-493, 496) | $NO_2$, $SO_2$, $SO_4$, $O_3$ | $SO_2$ correlated with respiratory visits in all age groups. $SO_4$ correlated in ages >14. $NO_2$ correlated in ages 15-60. $O_3$ not significant. | Results varied somewhat by season. *Study not used to estimate incidence: correlations only presented.* |
| Cody et al. (1992) | central and northern New Jersey May-August 1988-1989 | all ages | bronchitis (466, 490, 491, 496) | $PM_{10}$, $SO_2$, $O_3$ | No significant effect seen for $PM_{10}$, $O_3$, or $SO_2$ on bronchitis admissions. | $PM_{10}$ sampled every sixth day, so limited dataset. $PM_{10}$ considered in separate analysis. *Study not used to estimate incidence.* |
| Delfino et al. (1997) | Montreal, Canada June-September 1992-1993 | <2; 2-64; >64 | all respiratory (not defined by ICD code) | $O_3$, $PM_{10}$, $PM_{2.5}$, $SO_4$, $H^+$ | Single pollutant models: $H^+$ and $SO_4$ significant in ages <2; no effect in ages 2-64 for any pollutants; $O_3$, $PM_{10}$, $PM_{2.5}$, and $SO_4$ significant in ages >64. Two pollutant model: $O_3$ significant and $PM_{2.5}$ not significant in ages >64. | Limited number of results presented for two pollutant models. *Study not used to estimate incidence: all respiratory not defined by ICD code.* |
| Delfino et al. (1998) | Montreal, Canada June-August 1989-1990 | >64 | all respiratory (not defined by ICD code) | $PM_{2.5}$, $O_3$ | Two pollutant model: $O_3$ significant; $PM_{2.5}$ has consistent link but not significant. | $PM_{2.5}$ measured every sixth day, with rest of daily observations estimated from visibility and other data. *Study not used to estimate incidence: all respiratory not defined by ICD code.* |
| Samet et al. (1981) | Steubenville, Ohio March-April and October-November 1974-1977 | all ages | all respiratory (not defined by ICD code) | $NO_2$, TSP, $SO_2$, CO, $O_3$ | Single pollutant models: TSP and $SO_2$ significant; $NO_2$,, CO, or $O_3$ were not significant. | *Study not used to estimate incidence: all respiratory not defined by ICD code.* |

| Study | Location and Period | Population | Endpoint (ICD code) | Pollutants | Main Findings | Comment |
|---|---|---|---|---|---|---|
| Pantazopolou et al. (1995) | Athens, Greece 1988 | all ages | all outpatient visits | BS, CO, $NO_2$ | Single-pollutant models: $NO_2$ significant in the winter. No effects found for any pollutant in the summer. | *Study not used to estimate incidence: study outside U.S. and Canada.* |
| Sunyer et al. (1993) | Barcelona, Spain 1985-1989 | >14 | COPD (not defined by ICD code) | $SO_2$, BS | $SO_2$ correlated with ER visits in the summer and winter. BS significant in the winter only | *Study not used to estimate incidence: study outside U.S. and Canada.* |
| Xu et al. (1995b) | Beijing, China 1990 | all ages | all causes | $SO_2$, TSP | $SO_2$ and TSP both linked to ER visits. | *Study not used to estimate incidence: study outside U.S. and Canada.* |

## Minor Illness

In addition to chronic illnesses and hospital admissions, there is a considerable body of scientific research that has estimated significant relationships between elevated air pollution levels and other morbidity health effects. Chamber study research has established relationships between specific air pollution chemicals and symptoms such as coughing, pain on deep inspiration, wheezing, eye irritation and headaches. In addition, epidemiological research has found air pollution relationships with acute infectious diseases (e.g., bronchitis, sinusitis) and a variety of "symptom-day" categories. Some "symptom-days" studies examine excess incidences of days with identified symptoms such as wheezing, coughing, or other specific upper or lower respiratory symptoms. Other studies estimate relationships for days with a more general descriptions of days with adverse health impacts, such as "respiratory restricted activity days" or work loss days.

A major challenge in preparing an analysis of the minor morbidity effects is identifying a set of effect estimates that reflects the full range of identified adverse health effects but avoids double counting. From the definitions of the specific health effects examined in each research project, it is possible to identify a set of effects that are non-overlapping, and can be ultimately treated as additive in the monetary benefits analysis. This section primarily focuses on the set of effect relationships that have been identified that make up a non-overlapping set. Table D-12 summarizes the studies used in estimating minor illnesses; Tables D-13 and D-14 provide more detailed information on these studies and other studies that were not used in the analysis.

### Acute Bronchitis

Dockery et al. (1996) examined the relationship between PM and other pollutants on the reported rates of asthma, persistent wheeze, chronic cough, and bronchitis, in a study of 13,369 children ages 8-12 living in 24 communities in U.S. and Canada. Health data were collected in 1988-1991. Single-pollutant models were used in the analysis. Annual levels of sulfates and particle acidity were significantly related to bronchitis, and $PM_{2.5}$ and $PM_{10}$ were marginally significant. Earlier work, based on six U.S. cities, by Dockery et al. (1989) found acute bronchitis and chronic cough significantly related to $PM_{15}$. Because it is based on a larger sample, the Dockery et al. (1996) study is used to develop a C-R function linking $PM_{2.5}$ with acute bronchitis.

### Upper Respiratory Symptoms (URS)

Using logistic regression, Pope et al. (1991) estimated the impact of $PM_{10}$ on the incidence of a variety of minor symptoms in 55 subjects (34 "school-based" and 21 "patient-based") living in the Utah Valley from December 1989 through March 1990. The children in the Pope et al. study were asked to record respiratory symptoms in a daily diary, and the daily occurrences of upper respiratory symptoms (URS) and lower respiratory symptoms (LRS), as defined above, were related to daily $PM_{10}$ concentrations. Pope et al. describe URS as consisting of one or more of the following symptoms: runny or stuffy nose; wet cough; and burning, aching, or red eyes. Levels of ozone, $NO_2$, and $SO_2$ were reported low during this period, and were not included in the analysis. The sample in this study is relatively small and is most representative of the asthmatic population, rather than the general population. The school-based subjects (ranging in age from 9 to 11) were chosen based on "a positive response to one or more of three questions: ever wheezed without a cold, wheezed for 3 days or more out of the week for a month or longer, and/or had a doctor say the 'child has asthma' (Pope et al., 1991, p. 669)." The patient-based subjects (ranging in age from 8 to 72) were receiving treatment for asthma and were referred by local physicians. Regression results for the school-based sample (Pope et al., 1991, Table 5) show $PM_{10}$ significantly associated with both upper and lower respiratory symptoms. The patient-based sample did not find a significant $PM_{10}$ effect. The results from the school-based sample are used here.

## Lower Respiratory Symptoms (LRS)

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

## Respiratory Illness

Several epidemiological studies report that $NO_2$ exposure increases risk of respiratory illness in children. The results of many of the studies are not statistically significant. In addition, many of the studies do not provide ambient $NO_2$ measurements, having focused on the presence or absence of gas stoves as surrogates for exposure. However, there are data available from a well-designed study with adequate ambient exposure measurements. Based on work by Melia et al. (1980; 1982), Hasselblad et al. (1992) examined data from 103 children in homes where gas stoves were present and where bedroom $NO_2$ measurements were taken. A significant increase in respiratory illness was found to be a function of bedroom $NO_2$ levels, independent of social class, age, gender, or the presence of a smoker in the house. Hasselblad et al. used a multiple logistic model fitted to the Melia data with a linear slope for $NO_2$ and separate intercepts for boys and girls. This analysis uses the average slope of these two estimates.

## Work Loss Days (WLD)

Ostro (1987) estimated the impact of PM on the incidence of work-loss days (WLD) in a national sample of the adult working population, ages 18 to 65, living in metropolitan areas. Separate coefficients were developed for each year in the analysis (1976-1981); we then combined these coefficients for use in this analysis using a weighted average based on the inverse of the variances.

## Minor Restricted Activity Days (MRAD) / Any of 19 Respiratory Symptoms

Two studies by Ostro and Rothschild (1989b) and Krupnick et al. (1990) cover a variety of minor respiratory symptoms. To avoid double counting, we treat these two studies as alternative measures of the same health effect, and pool the incidence estimates.

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

Krupnick et al. (1990) estimated the impact of coefficient of haze (COH, a measure of particulate matter concentrations), ozone and $SO_2$ on the incidence of any of 19 respiratory symptoms or conditions.[14] They used a logistic regression model that takes into account whether a respondent was well or not the previous day. A key difference between this and the usual logistic model is that the model they used includes a lagged value of the dependent variable.

## Moderate or Worse Asthma

This health endpoint comes from Ostro et al. (1991), a study in which asthmatics, ages 18 to 70, were asked to record daily a subjective rating of their overall asthma status each day (0=none, 1=mild, 2=moderate, 3=severe, 4=incapacitating). Ostro et al.

[14]Krupnick et al. (1990) list 13 specific "symptoms or conditions": head cold, chest cold, sinus trouble, croup, cough with phlegm, sore throat, asthma, hay fever, doctor-diagnosed ear infection, flu, pneumonia, bronchitis, and bronchiolitis. The other six symptoms or conditions are not specified.

then examined the relationship between moderate (or worse) asthma and $H^+$, sulfate, $SO_2$, $PM_{2.5}$, estimated $PM_{2.5}$, $PM_{10}$, nitrate, and nitric acid. The published results used in the prospective analysis are from a single-pollutant linear regression model where the log of the pollutant is used.

Asthma "attacks" associated with ozone are estimated using the study by Whittemore and Korn (1980). Symptoms in asthmatic children associated with $SO_2$ are from Linn et al. (1987; 1988; 1990) and Roger et al. (1985).

## Shortness of Breath

Using a logistic regression estimation, Ostro et al. (1995) estimated the impact of $PM_{10}$, ozone, $NO_2$, and $SO_2$ on the incidence of coughing, shortness of breath, and wheezing in 83 African-American asthmatic children ages 7-12 living in Los Angeles from August through September 1992. Regression results show both $PM_{10}$ and ozone significantly linked to shortness of breath; the beginning of an asthma episode was also significantly linked to ozone. Results for single-pollutant models only were presented in the published paper.

## Restricted Activity Days (RADs)

Ostro (1987) used a log-linear regression to estimate the impact of $PM_{2.5}$ on the incidence of restricted activity days (RAD) in a national sample of the adult population, ages 18 to 65, living in metropolitan areas. Separate coefficients were developed for each year in the analysis (1976-1981); these coefficients were pooled. The coefficient used in the concentration-response function used here is a weighted average of the coefficients in Ostro (1987, Table III) using the inverse of the variance as the weight.

**Table D-12**
**Studies Used to Develop Minor Illness Estimates**

| Endpoints Estimated | Study Population Age | Study | Pollutants Used in Final Model |
|---|---|---|---|
| acute bronchitis | 8-12 | Dockery et al. (1996) | $PM_{2.5}$ |
| upper respiratory symptoms | 9-11 | Pope et al. (1991) | $PM_{10}$ |
| lower respiratory symptoms | 7-14 | Schwartz et al. (1994) | $PM_{2.5}$ |
| respiratory illness | 6-7 | Hasselblad et al. (1992) | $NO_2$ |
| any of 19 respiratory symptoms | 18-65 | Krupnick et al. (1990) | $O_3$, $PM_{10}$ |
| moderate or worse asthma | all ages (asthmatics) | Ostro et al. (1991) | $PM_{2.5}$ |
| asthma attacks | all ages (asthmatics) | Whittemore and Korn (1980) | $O_3$, $PM_{10}$ |
| chest tightness, shortness of breath, or wheeze | all ages (asthmatics) | Linn et al. (1987; 1988; 1990) and Roger et al. (1985) | $SO_2$ |
| shortness of breath | 7-12 (African-American asthmatics) | Ostro et al. (1995) | $PM_{10}$ |
| work loss days | 18-65 | Ostro (1987) | $PM_{2.5}$ |
| minor restricted activity days | 18-65 | Ostro and Rothschild (1989b) | $PM_{2.5}$, $O_3$ |
| restricted activity days | 18-65 | Ostro (1987) | $PM_{2.5}$ |

**Table D-13**
**Summary of Selected Studies for Minor Illness**

| Study | Location and Period | Population | Endpoint | Pollutants | Main Findings | Comment |
|---|---|---|---|---|---|---|
| Dockery et al. (1996) | 24 communities in U.S. and Canada 1988-1991 | 13,369 children ages 8-12 | asthma, persistent wheeze, chronic cough, bronchitis | particle acidity, $SO_4$, $PM_{2.1}$, $PM_{10}$, $HNO_2$, $HNO_3$, $O_3$ | Annual level of sulfates and particle acidity related to bronchitis. $HNO_2$ and $HNO_3$ linked to asthma. $SO_2$ linked to chronic phlegm. | Study examined annual pollution exposures, and the authors did not rule out that acute (daily) exposures could be related to asthma attacks and other acute episodes. |
| Dockery et al. (1989) | Six U.S. cities 1980-1981 | 5,422 children ages 10-12 | bronchitis, chest illness, cough, wheeze, asthma | $SO_2$, $NO_2$, $O_3$, $PM_{2.5}$, $PM_{15}$, TSP, $SO_4$ | Annual level of $PM_{15}$ significantly related to bronchitis and chronic cough. Annual $O_3$ significantly related to asthma. | |
| Hasselblad et al. (1992) | Meta-analysis of 11 studies from the U.S. and Europe | children ages 5-12 | lower respiratory tract illness | $NO_2$ | Annual $NO_2$ change of 30 µg/m$^3$ associated with lower respiratory tract illness. | |
| Hoek and Brunekreef (1995) | Two rural towns in the Netherlands. Spring-Summer 1989 | 300 children ages 7-11 | symptoms including: cough, phlegm, wheeze, runny nose, throat pain, headache, eye irritation, physician visit | $SO_2$, $NO_2$, $O_3$, $PM_{2.5}$, $PM_{10}$, $PM_{2.5-10}$, $SO_4$, $NO_3$ | Daily pollutant levels not associated with any of the symptoms studied. | |
| Krupnick et al. (1990) | Three communities in Los Angeles County, California 9/78-3/79 | 570 adults and 756 children | any of 19 respiratory symptoms including cough with phlegm | $O_3$, COH (coefficient of haze), $SO_2$, $NO_2$ | In single pollutant models, daily $O_3$, COH, and $SO_2$ related to respiratory symptoms in adults. $O_3$ significant controlling for other pollutants. Results more variable for COH and $SO_2$, perhaps due to collinearity. $NO_2$ had no significant effect. No effect seen in children for any pollutant. | |
| Ostro (1987) | Nationwide sample from U.S. Health Interview Survey 1976-1981 | Adults ages 18-65 | work-loss days restricted activity days (RADs), respiratory-related RADs | $PM_{2.5}$ | Two-week average $PM_{2.5}$ levels significantly linked to work-loss days, RADs, and respiratory-related RADs. Some year-to-year variability in results. | $PM_{2.5}$ estimated from visibility data. |

| Study | Location and Period | Population | Endpoint | Pollutants | Main Findings | Comment |
|---|---|---|---|---|---|---|
| Ostro et al. (1993) | Three communities in Los Angeles County, California 9/78-3/79 | 321 non-smoking adults | lower respiratory symptoms, upper respiratory symptoms, eye irritation | $O_3$, COH, $SO_4$, $SO_2$, $NO_2$ | In single pollutant model, daily $O_3$ linked to lower and upper respiratory symptoms. $SO_4$ linked to lower respiratory symptoms. No significant effects seen for COH, $SO_2$, and $NO_2$. | |
| Ostro and Rothschild (1989b) | Nationwide sample from U.S. Health Interview Survey 1976-1981 | Adults ages 18-65 | respiratory-related RADs, minor RADs. | $O_3$, $PM_{2.5}$ | Controlling for $PM_{2.5}$, two-week average $O_3$ has highly variable association with respiratory-related and minor RADs. Controlling for $O_3$, two-week average $PM_{2.5}$ significantly linked to both health endpoints in most years. | $PM_{2.5}$ estimated from visibility data. |
| Peters et al. (1999) | Twelve communities in southern California 1994 | 3,676 fourth, seventh, tenth grade students | asthma, wheeze, bronchitis, cough | $SO_2$, $NO_2$, $O_3$, $PM_{2.5}$, $PM_{10}$, $PM_{2.5-10}$, $SO_4$, $NO_3$, $NH_4$, gaseous acids | Wheeze in males, linked to annual average $NO_2$ and acid in 1994 (similar link for exposure averaged over 1986-1990). Peak ozone reported associated with decreased asthma prevalence in females. No other reported effects. | |
| Pope and Dockery (1992) | Utah Valley 12/90-3/91 | 79 children ages 10-12 | upper respiratory symptoms, lower respiratory symptoms, cough | $PM_{10}$ | $PM_{10}$ linked to daily reported incidences of upper and lower respiratory symptoms and cough. Effect seen in symptomatic sample. Only cough in symptomatic sample linked to $PM_{10}$. | Of the 79 children in the sample, 39 were symptomatic, and the other 40 were asymptomatic. |
| Pope et al. (1991) | Utah Valley 12/89-3/90 | 34 children ages 9-11, and 21 asthmatics ages 8-72 | upper respiratory symptoms, lower respiratory symptoms, took asthma medication | $PM_{10}$ | $PM_{10}$ significantly linked to upper and lower respiratory symptoms in sample of 34 children. $PM_{10}$ linked only to increased asthma medication use in the asthmatic sample. | |

| Study | Location and Period | Population | Endpoint | Pollutants | Main Findings | Comment |
|---|---|---|---|---|---|---|
| Schwartz et al. (1994) | Six U.S. cities April-August in one year between 1984 and 1988 (year varies by city) | 1,844 children | upper respiratory symptoms, lower respiratory symptoms, cough | $SO_2$, $NO_2$, $O_3$, $PM_{2.5}$, $PM_{10}$, $SO_4$, $H^+$ | In single pollutant models $SO_2$, $NO_2$, $PM_{2.5}$, and $PM_{10}$ significantly linked to cough. In two-pollutant models, $PM_{10}$ has most consistent effect; other pollutants not significant, controlling for $PM_{10}$. In single pollutant models, $SO_2$, $O_3$, $PM_{2.5}$, $PM_{10}$, $SO_4$, and $H^+$ linked to lower respiratory symptoms. No effect seen for upper respiratory symptoms. | |
| Schwartz and Zeger (1990) | Los Angeles, CA 1961-1964 | 110 student nurses | cough, phlegm, sore throat, headache, chest discomfort, eye irritation | CO, $SO_2$, $NO_2$, $O_x$ | $NO_2$ linked to sore throat, phlegm, and eye irritation. Oxidants ($O_x$) linked to chest discomfort and eye irritation. CO linked to headache. | Results presented as a mix of single pollutant and dual pollutant models. Stepwise selection used to pick significant covariates. |
| von Mutius et al. (1995) | Leipzig, Germany 10/91-7/92 | 1,500 children ages 9-11 | upper respiratory symptoms | $SO_2$, $NO_x$, PM | In single pollutant models, $SO_2$, $NO_x$, and PM linked to upper respiratory symptoms in winter (high pollution season). In the summer, only $NO_x$ linked to respiratory symptoms. | PM measured by beta-absorption. The limited modeling results presented for models with more than one pollutant were similar to single pollutant results. |

**Table D-14**
**Summary of Selected Studies for Asthmatics**

| Study | Location and Period | Population | Endpoint | Pollutants | Main Findings | Comment |
|---|---|---|---|---|---|---|
| Forsberg et al. (1993) | Pitea, Sweden about sixty days | 31persons ages 9-71 | shortness of breath, wheeze, cough, phlegm | BS (black smoke), $SO_2$, $NO_2$ | Controlling for other pollutants, daily levels of BS linked to shortness of breath. No link between pollutants and wheeze, cough, and phlegm. | Black smoke is an indirect measure of PM. |
| Gielen et al. (1997) | Amsterdam, Netherlands summer 1995 | 61 children ages 7-13 | upper respiratory symptoms, lower respiratory symptoms, medication use | $O_3$, $PM_{10}$, BS | In single pollutant model, daily levels of BS significantly linked to lower and upper respiratory symptoms and medication use. $PM_{10}$ linked to lower respiratory symptoms and medication use. $O_3$ linked to upper respiratory symptoms. | Results in the model highly dependent on the lag length used. The five-day mean black smoke and $PM_{10}$ yielded significant results, but current, one and two day lags did not. Current day $O_3$ significant. |
| Hiltermann et al. (1998) | Leiden University, Netherlands 7/3/95-10/6/95 | 60 adults ages 18-55 | symptoms include: shortness of breath, cough, phlegm, wheeze, runny nose, throat pain, headache, eye irritation, physician visit | $O_3$, $PM_{10}$, BS, $NO_2$, $SO_2$ | In single pollutant models, daily levels of $O_3$, $PM_{10}$, BS, and $NO_2$ linked to shortness of breath. Some significant negative associations reported for nasal symptoms and levels of $PM_{10}$, BS, and $NO_2$. No significant effect reported for $SO_2$. | |
| Linn et al. (1987; 1988; 1990) and Roger et al. (1985) | Chamber studies. | Exercising, young asthmatics | chest tightness, shortness of breath, or wheeze | $SO_2$ | $SO_2$ exposure linked to moderate symptoms in these studies of moderately exercising young asthmatics. | |

| Study | Location and Period | Population | Endpoint | Pollutants | Main Findings | Comment |
|---|---|---|---|---|---|---|
| Neukirch et al. (1998) | Paris, France 11/92-5/93 | 40 persons (mean age of sample was 46) | asthma, wheeze, shortness of breath, cough, respiratory infection | $PM_{13}$, BS, $NO_2$, $SO_2$ | In single pollutant models, daily levels of $PM_{13}$, BS, $NO_2$, and $SO_2$ were each significantly associated with asthma attacks, wheeze, cough, respiratory infections, and shortness of breath. | $PM_{13}$ used rather than the more common $PM_{10}$. |
| Ostro et al. (1991) | Denver, CO 12/87-2/88 | 207 persons ages 18-70 | severity of asthma symptoms, cough, wheeze, shortness of breath, chest tightness | $SO_2$, $PM_{2.5}$, $SO_4$, $NO_3$, $H^+$, nitric acid | Daily levels of $H^+$ linked to cough, asthma, and shortness of breath. $PM_{2.5}$ linked to asthma. $SO_4$ linked to shortness of breath. No effects seen for other pollutants. | Some $PM_{2.5}$ estimated. Exclusion of estimated data removes significant link to asthma. Only single pollutant models reported. |
| Ostro et al. (1995) | Los Angeles, CA 8/92-11/92 | 83 children ages 7-12 | cough, shortness of breath, wheeze | $O_3$, $NO_2$, $SO_2$, $PM_{10}$ | In single pollutant models, daily levels of $O_3$ and $PM_{10}$ linked only to shortness of breath. No effect seen for $NO_2$ and $SO_2$. | |
| Peters et al. (1996) | Three cities in East Germany and the Czech Republic 9/90-6/92 | 155 children ages 7-15 and 102 adults ages 32-80 | symptom score based on a variety of respiratory symptoms | TSP, $SO_2$, $SO_4$, particle acidity | Daily $SO_2$ linked to the respiratory symptom score. No link between the other pollutants and the symptom score. | |
| Roemer et al. (1998) | 28 locations in Europe winter 1993-1994 | 2,010 children ages 6-12 | symptoms include: shortness of breath, cough, phlegm, wheeze, runny nose, sore throat, headache, eye irritation | $PM_{10}$, BS, $NO_2$, $SO_2$ | Daily pollutant levels not related to adverse health symptoms. | |
| Romieu et al. (1996) | Mexico City | 71 children ages 5-7 | cough, phlegm, difficulty breathing, wheezing, lower respiratory illness | $PM_{10}$, $PM_{2.5}$, $O_3$, $NO_2$, $SO_2$ | Controlling for $PM_{2.5}$, daily levels of $O_3$ linked to cough and lower respiratory illness. Controlling for $O_3$, $PM_{2.5}$ linked to cough, phlegm, and lower respiratory symptoms. | $PM_{10}$ also linked adverse symptoms. Published results focused on $O_3$ and $PM_{2.5}$. Results for $NO_2$ and $SO_2$ not reported. |

| Study | Location and Period | Population | Endpoint | Pollutants | Main Findings | Comment |
|---|---|---|---|---|---|---|
| Whittemore and Korn (1980) | Six communities in southern CA Three 34-week periods 1972-1975 | 443 children and adults | asthma | $O_x$, TSP | In a two pollutant model, daily levels of both TSP and $O_x$ were significantly related to reported asthma attacks. | Respirable PM, $NO_2$, $SO_2$ were highly correlated with TSP and excluded from the analysis. |

## C-R Functions Linking Air Pollution and Adverse Health Effects

After selecting studies appropriate for the present analysis, the published information was used to derive a C-R 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, information needed to apply these functions, and references for information.

### *Carbon Monoxide*

Four C-R relationships are available for estimating hospital admissions related to ambient CO levels. These are summarized in Table D-15.

**Table D-15**
**Summary of C-R Functions for Carbon Monoxide**

| Health Endpoint | C-R Function | Source of C-R Function |
|---|---|---|
| hospital admissions – asthma | $\Delta asthma\,admissions = -\left[y_0 \cdot (e^{-\beta \cdot \Delta CO} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admission rate for asthma per person = 4.75 E-6<br>$\beta$ = CO coefficient = 0.0332<br>$\Delta CO$ = change in daily average CO concentration (ppm)<br>pop = population of all ages<br>$\sigma_\beta$ = standard error of $\beta$ = 0.00861 | Study: Burnett et al. (1999)<br>Location: Toronto, Canada<br>Other pollutants in model: $PM_{2.5\text{-}10}$, $O_3$ |
| hospital admissions – obstructive lung disease | $\Delta obs.lung\,disease\,admissions = -\left[y_0 \cdot (e^{-\beta \cdot \Delta CO} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admission rate for obstructive lung disease per person = 5.76 E-6<br>$\beta$ = CO coefficient = 0.0250<br>$\Delta CO$ = change in daily average CO concentration (ppm)<br>pop = population of all ages<br>$\sigma_\beta$ = standard error of $\beta$ = 0.0165 | Study: Burnett et al. (1999)<br>Location: Toronto, Canada<br>Other pollutants in model: $PM_{2.5\text{-}10}$, $O_3$ |
| hospital admissions – COPD | $\Delta COPD\,admissions = -\left[y_0 \cdot (e^{-\beta \cdot \Delta CO} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admission rate for COPD per person = 3.75 E-5<br>$\beta$ = CO coefficient = 0.0573<br>$\Delta$ CO = change in daily average CO concentration (ppm)<br>pop = population age 65 and older<br>$\sigma_\beta$ = standard error of $\beta$ = 0.0329 | Study: Moolgavkar (1997)<br>Location: Minneapolis-St. Paul<br>Other pollutants in model: $O_3$, $PM_{10}$ |
| hospital admissions – asthma | $\Delta asthma\,admissions = -\left[y_0 \cdot (e^{-\beta \cdot \Delta CO} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admission rate for asthma per person = 4.52 E-6<br>$\beta$ = CO coefficient = 0.0528<br>$\Delta$ CO = change in daily average CO concentration (ppm)<br>pop = population of ages < 65<br>$\sigma_\beta$ = standard error of $\beta$ = 0.0185 | Study: Sheppard (1999)<br>Location: Seattle, WA<br>Other pollutants in model: $PM_{2.5}$ |
| hospital admissions – dysrhythmias | $\Delta dysrhythmias\,admissions = -\left[y_0 \cdot (e^{-\beta \cdot \Delta CO} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admission rate for dysrhythmias per person = 6.46 E-6<br>$\beta$ = CO coefficient = 0.0573<br>$\Delta CO$ = change in daily average CO concentration (ppm)<br>pop = population of all ages<br>$\sigma_\beta$ = standard error of $\beta$ = 0.0229 | Study: Burnett et al. (1999)<br>Location: Toronto, Canada<br>Other pollutants in model: $PM_{2.5}$, $O_3$ |

| Health Endpoint | C-R Function | Source of C-R Function |
|---|---|---|
| hospital admissions – congestive heart failure | $\Delta congestive\ heart\ failure\ admissions = -\left[y_0 \cdot (e^{-\beta \cdot \Delta CO} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admission rate for congestive heart failure per person = 9.33 E-6<br>$\beta$ = CO coefficient = 0.0340<br>$\Delta CO$ = change in daily average CO concentration (ppm)<br>pop = population of all ages<br>$\sigma_\beta$ = standard error of $\beta$ = 0.0163 | Study: Burnett et al. (1999)<br>Location: Toronto, Canada<br>Other pollutants in model: $NO_2$ |
| hospital admissions – ischemic heart disease | $\Delta ischemic\ heart\ disease\ admissions = -\left[y_0 \cdot (e^{-\beta \cdot \Delta CO} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admission rate for ischemic heart disease per person 65 and older = 9.96 E-5<br>$\beta$ = CO coefficient= 0.000467<br>$\Delta CO$ = change in daily one-hour maximum CO concentration (ppm)<br>pop = population age 65 and older<br>$\sigma_\beta$ = standard error of $\beta$ = 0.000435 | Study: Schwartz and Morris (1995)<br>Location: Detroit, MI<br>Other pollutants in model: $PM_{10}$ |
| hospital admissions – congestive heart failure | $\Delta congestive heart\ failure\ admissions = -\left[y_0 \cdot (e^{-\beta \cdot \Delta CO} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admission rate for congestive heart failure per person 65 and older = 5.82 E-5<br>$\beta$ = CO coefficient = 0.0170<br>$\Delta CO$ = change in daily one-hour maximum CO concentration (ppm)<br>pop = population age 65 and older<br>$\sigma_\beta$ = standard error of $\beta$ = 0.00468 | Study: Schwartz and Morris (1995)<br>Location: Detroit, MI<br>Other pollutants in model: $PM_{10}$ |
| hospital admissions – cardiovascular | $\Delta cardiovascular\ admissions = -\left[y_0 \cdot (e^{-\beta \cdot \Delta CO} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admission rate for cardiovascular disease per person 65 and older = 2.23 E-4<br>$\beta$ = CO coefficient = 0.0127<br>$\Delta CO$ = change in daily one-hour maximum CO concentration<br>pop = population age 65 and older<br>$\sigma_\beta$ = standard error of $\beta$ = 0.00255 | Study: Schwartz (1999)<br>Location: eight U.S. counties<br>Other pollutants in model: $PM_{10}$ |
| hospital admissions – cardiovascular | $\Delta cardiovascular\ admissions = -\left[y_0 \cdot (e^{-\beta \cdot \Delta CO} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admission rate for cardiovascular disease per person 65 and older = 2.23 E-4<br>$\beta$ = CO coefficient = 0.0139<br>$\Delta CO$ = change in daily one-hour maximum CO concentration<br>pop = population age 65 and older<br>$\sigma_\beta$ = standard error of $\beta$ = 0.00715 | Study: Schwartz (1997)<br>Location: Tucson, AZ<br>Other pollutants in model: $PM_{10}$ |

### Nitrogen Dioxide

Nitrogen dioxide ($NO_2$) is the primary focus of health studies on the nitrogen oxides and serves as the basis for this analysis. Table D-16 summarizes the C-R functions that are used to quantify the relationship between $NO_2$ and adverse health effects.

**Table D-16**
**Summary of C-R Functions for Nitrogen Dioxide**

| Health Endpoint | C-R Function | Source of C-R Function |
|---|---|---|
| hospital admissions – all respiratory | $\Delta All\ respiratory = -\left[y_0 \cdot (e^{-\beta \cdot \Delta NO_2} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admission rate for all respiratory per person = 2.58 E-5<br>$\beta$ = $NO_2$ coefficient = 0.00378<br>$\Delta NO_2$ = change in daily 12-hour average $NO_2$ concentration (ppb)<br>pop = population of all ages<br>$\sigma_\beta$ = standard error of $\beta$ = 0.00221 | Study: Burnett et al. (1997b)<br>Location: Toronto, Canada<br>Other pollutants in model: $PM_{2.5\text{-}10}$, $O_3$, $SO_2$ |
| hospital admissions – respiratory infection | $\Delta Respiratory\ Infection\ Admissions = -\left[y_0 \cdot (e^{-\beta \cdot \Delta NO_2} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admission rate for respiratory infection per person = 1.56 E-5<br>$\beta$ = $NO_2$ coefficient = 0.00172<br>$\Delta NO_2$ = change in daily average $NO_2$ concentration (ppb)<br>pop = population of all ages<br>$\sigma_\beta$ = standard error of $\beta$ = 0.000521 | Study: Burnett et al. (1999)<br>Location: Toronto, Canada<br>Other pollutants in model: $PM_{2.5}$, $O_3$ |
| hospital admissions – pneumonia | $\Delta pneumonia\ admissions = -\left[y_0 \cdot (e^{-\beta \cdot \Delta NO_2} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admission rate for pneumonia per person = 5.30 E-5<br>$\beta$ = $NO_2$ coefficient = 0.00169<br>$\Delta$ $NO_2$ = change in daily average $NO_2$ concentration (ppb)<br>pop = population age 65 and older<br>$\sigma_\beta$ = standard error of $\beta$ = 0.00125 | Study: Moolgavkar et al. (1997)<br>Location: Minneapolis, MN<br>Other pollutants in model: $O_3$, $SO_2$, $PM_{10}$ |
| hospital admissions – congestive heart failure | $\Delta Congestive\ Heart\ Failure\ Admissions = -\left[y_0 \cdot (e^{-\beta \cdot \Delta NO_2} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admission rate for congestive heart failure per person = 9.33 E-6<br>$\beta$ = $NO_2$ coefficient = 0.00264<br>$\Delta NO_2$ = change in daily average $NO_2$ concentration (ppb)<br>pop = population of all ages<br>$\sigma_\beta$ = standard error of $\beta$ = 0.000769 | Study: Burnett et al. (1999)<br>Location: Toronto, Canada<br>Other pollutants in model: CO |

| Health Endpoint | C-R Function | Source of C-R Function |
|---|---|---|
| hospital admissions – ischemic heart disease | $$\Delta Ischemic\ Heart\ Disease\ Admissions = -\left[y_0 \cdot (e^{-\beta \cdot \Delta NO_2} - 1)\right] \cdot pop,$$ where:<br>$y_0$ = daily hospital admission rate for ischemic heart disease per person = 2.23 E-5<br>$\beta$ = $NO_2$ coefficient = 0.00318<br>$\Delta NO_2$ = change in daily average $NO_2$ concentration (ppb)<br>pop = population of all ages<br>$\sigma_\beta$ = standard error of $\beta$ = 0.000521 | Study: Burnett et al. (1999)<br>Location: Toronto, Canada<br>Other pollutants in model: $SO_2$ |
| respiratory symptoms | $$\Delta resp.symptoms = \left[\frac{1}{1+e^{-\alpha - NO_{2,pre-CAAA} \cdot \beta - gender \cdot \gamma}} - \frac{1}{1+e^{-\alpha - NO_{2,post-CAAA} \cdot \beta - gender \cdot \gamma}}\right] \cdot pop,$$ where:<br>$\alpha$ = constant = -0.536<br>$\beta$ = $NO_2$ coefficient = 0.0275<br>$\gamma$ = gender coefficient (used for males only) = -0.0295<br>$\Delta NO_2$ = change in annual $NO_2$ concentration (ppb)<br>pop = children ages 6-7<br>$\sigma_\beta$ = standard error of $\beta$ = 0.0132 | Study: Hasselblad et al. (1992)<br>Location: Middlesborough, England<br>Other pollutants in model: none<br>Comments: The $NO_2$ coefficient was reported by Hasselblad et al. The constant and the gender coefficient were obtained via personal communication with V. Hasselblad 2/28/95 by Abt Associates. The equation is based on study results by Melia et al. (1980). |

### *Ozone*

The health effects literature includes studies of the relationships between ozone and a variety of health effects. Table D-17 summarizes the ozone C-R functions used in this analysis.

**Table D-17**
**Summary of C-R Functions for Ozone**

| Health Endpoint | C-R Function | Source of C-R Function |
|---|---|---|
| mortality | $\Delta Mortality = -\left[y_0 \cdot (e^{-\beta \cdot \Delta O_3} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = non-accidental deaths per person of any age<br>$\beta$ = ozone coefficient = 0.000634<br>$\Delta O_3$ = change in daily one-hour maximum ozone concentration (ppb)<br>pop = population all ages<br>$\sigma_\beta$ = standard error of $\beta$ = 0.000251 | Study: Ito and Thurston (1996)<br>Location: Chicago, IL<br>Other pollutants in model: $PM_{10}$ |
| mortality | $\Delta Mortality = -\left[y_0 \cdot (e^{-\beta \cdot \Delta O_3} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = non-accidental deaths per person of any age<br>$\beta$ = ozone coefficient = 0<br>$\Delta O_3$ = change in daily 1-hour maximum ozone concentration (ppb)<br>pop = population all ages<br>$\sigma_\beta$ = standard error of $\beta$ = 0.000214 | Study: Kinney et al. (1995)<br>Location: Los Angeles, CA<br>Other pollutants in model: $PM_{10}$ |
| mortality | $\Delta Mortality = -\left[y_0 \cdot (e^{-\beta \cdot \Delta O_3} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = non-accidental deaths per person of any age<br>$\beta$ = ozone coefficient = 0.000611<br>$\Delta O_3$ = change in daily average ozone concentration (ppb)<br>pop = population all ages<br>$\sigma_\beta$ = standard error of $\beta$ = 0.000216 | Study: Moolgavkar et al. (1995)<br>Location: Philadelphia, PA<br>Other pollutants in model: $SO_2$, TSP |
| mortality | $\Delta Mortality = -\left[y_0 \cdot (e^{-\beta \cdot \Delta O_3} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = non-accidental deaths per person of any age<br>$\beta$ = ozone coefficient = 0.000936<br>$\Delta O_3$ = change in daily average ozone concentration (ppb)<br>pop = population all ages<br>$\sigma_\beta$ = standard error of $\beta$ = 0.000312 | Study: Samet et al. (1997)<br>Location: Philadelphia, PA<br>Other pollutants in model: CO, $NO_2$, $SO_2$, TSP |
| adult onset asthma | $\Delta Chronic\ Asthma = -\left[\frac{y_0}{(1-y_0) \cdot e^{\Delta O_3 \cdot \beta} + y_0} - y_0\right] \cdot pop,$<br>where:<br>$y_0$ = annual asthma incidence rate per person = 0.00219<br>$\beta$ = estimated $O_3$ coefficient = 0.0277<br>$\Delta O_3$ = change in annual average 8-hour $O_3$ concentration<br>pop = population of non-asthmatic males ages 27 and older<br>$\sigma_\beta$ = standard error of $\beta$ = 0.0135 | Study: McDonnell et al. (1999)<br>Location: California<br>Other pollutants in model: none |
| hospital admissions – all respiratory | $\Delta All\ respiratory = -\left[y_0 \cdot (e^{-\beta \cdot \Delta O_3} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admission rate for all respiratory per person = 2.58 E-5<br>$\beta$ = $O_3$ coefficient = 0.00498<br>$\Delta O_3$ = change in daily 12-hour average $O_3$ concentration (ppb)<br>pop = population of all ages<br>$\sigma_\beta$ = standard error of $\beta$ = 0.00106 | Study: Burnett et al. (1997b)<br>Location: Toronto, Canada<br>Other pollutants in model: $PM_{2.5\text{-}10}$, $NO_2$, $SO_2$ |

| Health Endpoint | C-R Function | Source of C-R Function |
|---|---|---|
| hospital admissions – asthma | $\Delta Asthma\ Admissions = -\left[y_0 \cdot (e^{-\beta \cdot \Delta O_3} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admission rate for asthma per person = 4.75 E-6<br>$\beta$ = ozone coefficient = 0.00250<br>$\Delta O_3$ = change in daily average ozone concentration (ppb)<br>pop = population of all ages<br>$\sigma_\beta$ = standard error of $\beta$ = 0.000718 | Study: Burnett et al. (1999)<br>Location: Toronto, Canada<br>Other pollutants in model: CO, $PM_{2.5\text{-}10}$ |
| hospital admissions – obstructive lung disease | $\Delta Obstructive\ Lung\ Disease\ Admissions = -\left[y_0 \cdot (e^{-\beta \cdot \Delta O_3} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admission rate for obstructive lung disease per person = 5.76 E-6<br>$\beta$ = ozone coefficient = 0.00303<br>$\Delta O_3$ = change in daily average ozone concentration (ppb)<br>pop = population of all ages<br>$\sigma_\beta$ = standard error of $\beta$ = 0.00110 | Study: Burnett et al. (1999)<br>Location: Toronto, Canada<br>Other pollutants in model: CO, $PM_{2.5\text{-}10}$ |
| hospital admissions – respiratory infection | $\Delta Respiratory\ Infection\ Admissions = -\left[y_0 \cdot (e^{-\beta \cdot \Delta O_3} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admission rate for respiratory infection per person = 1.56 E-5<br>$\beta$ = ozone coefficient = 0.00198<br>$\Delta O_3$ = change in daily average ozone concentration (ppb)<br>pop = population of all ages<br>$\sigma_\beta$ = standard error of $\beta$ = 0.000520 | Study: Burnett et al. (1999)<br>Location: Toronto, Canada<br>Other pollutants in model: $PM_{2.5}$, $NO_2$ |
| hospital admissions – all respiratory | $\Delta all\ respiratory\ admissions = \beta \cdot \Delta O_3 \cdot pop,$<br>where:<br>$\beta$ = ozone coefficient = 1.68 E-8<br>$\Delta O_3$ = change in daily one-hour maximum ozone concentration (ppb)<br>pop = population all ages<br>$\sigma_\beta$ = standard error of $\beta$ = 9.71 E-9 . | Study: Thurston et al. (1994)<br>Location: Toronto, Canada<br>Other pollutants in model: $PM_{2.5}$ |
| hospital admissions – pneumonia | $\Delta pneumonia\ admissions = -\left[y_0 \cdot (e^{-\beta \cdot \Delta O_3} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admission rate for pneumonia per person = 5.30 E-5<br>$\beta$ = $O_3$ coefficient = 0.00370<br>$\Delta O_3$ = change in daily average $O_3$ concentration (ppb)<br>pop = population age 65 and older<br>$\sigma_\beta$ = standard error of $\beta$ = 0.00103 | Study: Moolgavkar et al. (1997)<br>Location: Minneapolis, MN<br>Other pollutants in model: $SO_2$, $NO_2$, $PM_{10}$ |
| hospital admissions – COPD | $\Delta COPD\ admissions = -\left[y_0 \cdot (e^{-\beta \cdot \Delta O_3} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admission rate for COPD per person = 3.75 E-5<br>$\beta$ = $O_3$ coefficient = 0.00274<br>$\Delta O_3$ = change in daily average $O_3$ concentration (ppb)<br>pop = population age 65 and older<br>$\sigma_\beta$ = standard error of $\beta$ = 0.00170 | Study: Moolgavkar et al. (1997)<br>Location: Minneapolis, MN<br>Other pollutants in model: CO, $PM_{10}$ |

| Health Endpoint | C-R Function | Source of C-R Function |
|---|---|---|
| hospital admissions – pneumonia | $\Delta pneumonia\,admissions = -\left[y_0 \cdot (e^{-\beta \cdot \Delta O_3} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admission rate for pneumonia per person = 5.30 E-5<br>$\beta$ = $O_3$ coefficient = 0.00280<br>$\Delta O_3$ = change in daily average $O_3$ concentration (ppb)<br>pop = population age 65 and older<br>$\sigma_\beta$ = standard error of $\beta$ = 0.00172 | Study: Schwartz (1994c)<br>Location: Minneapolis, MN<br>Other pollutants in model: $PM_{10}$ |
| hospital admissions – pneumonia | $\Delta pneumonia\,admissions = -\left[y_0 \cdot (e^{-\beta \cdot \Delta O_3} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admission rate for pneumonia per person = 5.18 E-5<br>$\beta$ = $O_3$ coefficient = 0.00521<br>$\Delta O_3$ = change in daily average $O_3$ concentration (ppb)<br>pop = population age 65 and older<br>$\sigma_\beta$ = standard error of $\beta$ = 0.0013 | Study: Schwartz (1994b)<br>Location: Detroit, MI<br>Other pollutants in model: $PM_{10}$ |
| hospital admissions – COPD | $\Delta COPD\,admissions = -\left[y_0 \cdot (e^{-\beta \cdot \Delta O_3} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admission rate for COPD per person = 3.05 E-5<br>$\beta$ = $O_3$ coefficient = 0.00549<br>$\Delta O_3$ = change in daily average $O_3$ concentration<br>pop = population age 65 and older<br>$\sigma_\beta$ = standard error of $\beta$ = 0.00205 | Study: Schwartz (1994b)<br>Location: Detroit, MI<br>Other pollutants in model: $PM_{10}$ |
| hospital admissions – all respiratory | $\Delta all\,respiratory\,admissions = -\left[y_0 \cdot (e^{-\beta \cdot \Delta O_3} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admissions for all respiratory per person 65 and older = 1.187 E-4<br>$\beta$ = ozone coefficient = 0.00265<br>$\Delta O_3$ = change in daily average ozone concentration (ppb)<br>pop = population age 65 and older<br>$\sigma_\beta$ = standard error of $\beta$ = 0.00140 | Study: Schwartz (1995)<br>Location: New Haven, CT<br>Other pollutants in model: $PM_{10}$ |
| hospital admissions – all respiratory | $\Delta all\,respiratory\,related\,admissions = -\left[y_0 \cdot (e^{-\beta \cdot \Delta O_3} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admissions for all respiratory conditions per person 65 and older = 1.187 E-4<br>$\beta$ = ozone coefficient = 0.00715<br>$\Delta O_3$ = change in daily average ozone concentration (ppb)<br>pop = population age 65 and older<br>$\sigma_\beta$ = standard error of $\beta$ = 0.00257 | Study: Schwartz (1995)<br>Location: Tacoma, WA<br>Other pollutants in model: $PM_{10}$ |
| hospital admissions – cardiac | $\Delta cardiac = -\left[y_0 \cdot (e^{-\beta \cdot \Delta O_3} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admission rate for cardiac per person = 3.81 E-5<br>$\beta$ = $O_3$ coefficient = 0.00531<br>$\Delta O_3$ = change in daily 12-hour average $O_3$ concentration (ppb)<br>pop = population of all ages<br>$\sigma_\beta$ = standard error of $\beta$ = 0.00142 | Study: Burnett et al. (1997b)<br>Location: Toronto, Canada<br>Other pollutants in model: $PM_{2.5\text{-}10}$ |

| Health Endpoint | C-R Function | Source of C-R Function |
|---|---|---|
| hospital admissions – dysrhythmias | $\Delta Dysrhythmias\,Admissions = -\left[y_0 \cdot (e^{-\beta \cdot \Delta O_3} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admission rate for dysrhythmias per person = 6.46 E-6<br>$\beta$ = ozone coefficient = 0.00168<br>$\Delta O_3$ = change in daily average ozone concentration (ppb)<br>pop = population of all ages<br>$\sigma_\beta$ = standard error of $\beta$ = 0.00103 | Study: Burnett et al. (1999)<br>Location: Toronto, Canada<br>Other pollutants in model: $PM_{2.5}$, CO |
| emergency room visits - asthma | $\Delta asthma\,related\,ER\,visits = \frac{\beta}{BasePop} \cdot \Delta O_3 \cdot pop,$<br>where:<br>$\beta$ = ozone coefficient = 0.0203<br>BasePop = baseline population in northern New Jersey = 4,436,976<br>$\Delta O_3$ = change in daily five-hour average ozone concentration (ppb)<br>pop = population all ages<br>$\sigma_\beta$ = standard error of $\beta$ = 0.00717 | Study: Cody et al. (1992)<br>Location: Northern NJ<br>Other pollutants in model: none<br>Comment: 63 % of estimate used to avoid double-counting hospital admissions for asthma. |
| emergency room visits - asthma | $\Delta asthma\,related\,ER\,visits = \frac{\beta}{BasePop} \cdot \Delta O_3 \cdot pop,$<br>where:<br>$\beta$ = ozone coefficient = 0.0443<br>BasePop = baseline population in northern New Jersey = 4,436,976<br>$\Delta O_3$ = change in daily five-hour average ozone concentration (ppb)<br>pop = population all ages<br>$\sigma_\beta$ = standard error of $\beta$ = 0.00723 | Study: Weisel et al. (1995)<br>Location: Northern, NJ<br>Other pollutants in model: none<br>Comment: 63 % of estimate used to avoid double-counting hospital admissions for asthma. |
| emergency room visits - asthma | $\Delta asthma\,related\,ER\,visits = \frac{\beta}{BasePop} \cdot \Delta O_3 \cdot pop,$<br>where:<br>$\beta$ = ozone coefficient = 0.0035<br>BasePop = baseline population in Saint John, New Brunswick = 125,000<br>$\Delta O_3$ = change in the daily one-hour maximum ozone concentration (ppb)<br>pop = population all ages<br>$\sigma_\beta$ = standard error of $\beta$ = 0.0018 | Study: Stieb et al. (1996)<br>Location: New Brunswick, Canada<br>Other pollutants in model: none<br>Comment: 63 % of estimate used to avoid double-counting hospital admissions for asthma. |
| presence of any of 19 acute respiratory symptoms | $\Delta ARD2 \cong \beta^*_{PM_{10}} \cdot \Delta O_3 \cdot pop,$<br>where:<br>$\beta^*$ = first derivative of the stationary probability = 0.000137<br>$\Delta O_3$ = change in daily one-hour maximum ozone concentration (ppb)<br>pop = population aged 18-65 years old<br>$\sigma_\beta$ = standard error of $\beta^*$ = 0.0000697 | Study: Krupnick et al. (1990)<br>Location: Glendora-Covina-Azusa, CA<br>Other pollutants in model: $SO_2$, COH |
| self-reported asthma attacks | $\Delta asthma\,attacks = -\left[\frac{y_0}{(1-y_0) \cdot e^{\Delta O_3 \cdot \beta} + y_0} - y_0\right] \cdot pop,$<br>where:<br>$y_0$ = daily incidence of asthma attacks = 0.027<br>$\beta$ = ozone coefficient = 0.00184<br>$\Delta O_3$ = change in daily one-hour maximum ozone concentration (ppb)<br>pop = population of asthmatics of all ages = 5.61% of the population of all ages<br>$\sigma_\beta$ = standard error of $\beta$ = 0.000714 | Study: Whittemore and Korn (1980)<br>Location: Los Angeles, CA<br>Other pollutants in model: TSP |

| Health Endpoint | C-R Function | Source of C-R Function |
|---|---|---|
| respiratory and nonrespiratory conditions resulting in a minor restricted activity day (MRAD) | $\Delta MRAD = -\left[y_0 \cdot (e^{-\beta \cdot \Delta O_3} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily MRAD incidence per person = 0.02137<br>$\beta$ = inverse-variance weighted $PM_{2.5}$ coefficient = 0.00220<br>$\Delta O_3$ = change in two-week average of the daily one-hour maximum ozone concentrations (ppb)<br>pop = adult population aged 18 to 65<br>$\sigma_\beta$ = standard error of $\beta$ = 0.000658 | Study: Ostro and Rothschild (1989b)<br>Location: U.S.<br>Other pollutants in model: $PM_{2.5}$<br>Comments: An inverse-variance weighting used to estimate the coefficient, based on Ostro and Rothschild. |

## Particulate Matter

The C-R functions used to quantify expected changes in health effects associated with reduced exposure to particulate matter are summarized in Table D-18. The measures of particulate matter used in this analysis are $PM_{2.5}$ and $PM_{10}$, with a preference for $PM_{2.5}$. Other measures of PM, however, have been used, including total suspended particulates (TSP) and coefficient of haze.

**Table D-18**
**Summary of C-R Functions for Particulate Matter**

| Health Endpoint | C-R Function | Source of C-R Function |
|---|---|---|
| mortality | $$\Delta Nonaccidental\,Mortality = -\left[y_0 \cdot (e^{-\beta \cdot \Delta PM_{2.5}} - 1)\right] \cdot pop,$$ where:<br>$y_0$ = county-level annual non-accidental deaths of persons ages 30+ per person<br>$\beta$ = $PM_{2.5}$ coefficient = 0.006408<br>$\Delta PM_{2.5}$ = change in annual <u>median</u> $PM_{2.5}$ concentration<br>pop = population ages 30 and older<br>$\sigma_\beta$ = standard error of $\beta$ = 0.001509 | Study: Pope et al. (1995)<br>Location: 50 U.S. cities<br>Other pollutants in model: none |
| mortality | $$\Delta Nonaccidental\,Mortality = -\left[y_0 \cdot (e^{-\beta \cdot \Delta PM_{2.5}} - 1)\right] \cdot pop,$$ where:<br>$y_0$ = county-level annual non-accidental deaths of persons ages 25+ per person<br>$\beta$ = $PM_{2.5}$ coefficient = 0.0124<br>$\Delta PM_{2.5}$ = change in annual mean $PM_{2.5}$ concentration<br>pop = population ages 25 and older<br>$\sigma_\beta$ = standard error of $\beta$ = 0.00423 | Study: Dockery et al. (1993)<br>Location: six U.S. cities<br>Other pollutants in model: none |
| neonatal mortality | $$\Delta Infant\,Mortality = -\left[\frac{y_0}{(1-y_0) \cdot e^{\Delta PM_{10} \cdot \beta} + y_0} - y_0\right] \cdot pop,$$ where:<br>$y_0$ = county annual postneonatal infant deaths per infant 0-1 years old<br>$\beta$ = $PM_{10}$ coefficient = 0.00392<br>$\Delta PM_{10}$ = change in annual average $PM_{10}$ concentration<br>pop = population infants ages 0-1<br>$\sigma_\beta$ = standard error of $\beta$ = 0.00122 | Study: Woodruff et al. (1997)<br>Location: 86 U.S. metropolitan areas<br>Other pollutants in model: none |
| chronic bronchitis | $$\Delta Chronic\ Bronchitis = -\left[y_0 \cdot (e^{-\beta \cdot \Delta PM_{10}} - 1)\right] \cdot pop,$$ where:<br>$y_0$ = annual bronchitis incidence rate per person = 0.00378<br>$\beta$ = estimated $PM_{10}$ logistic regression coefficient = 0.00932<br>$\Delta PM_{10}$ = change in annual average $PM_{10}$ concentration<br>pop = population ages 27 and older "without chronic bronchitis"<br>$\sigma_\beta$ = standard error of $\beta$ = 0.00475 | Study: Abbey et al. (1993)<br>Location: California<br>Other pollutants in model: none<br>Comments: Abbey et al. used TSP to measure PM. The TSP coefficient is applied to changes in $PM_{10}$. |

| Health Endpoint | C-R Function | Source of C-R Function |
|---|---|---|
| chronic bronchitis | $\Delta Chronic\ Bronchitis = -\left[y_0 \cdot (e^{-\beta \cdot \Delta PM_{2.5}} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = annual bronchitis incidence rate per person = 0.00378<br>$\beta$ = estimated $PM_{2.5}$ logistic regression coefficient = 0.09132<br>$\Delta PM_{2.5}$ = change in annual average $PM_{2.5}$ concentration<br>pop = population ages 27 and older "without chronic bronchitis"<br>$\sigma_\beta$ = standard error of $\beta$ = 0.00680 | Study: Abbey et al. (1995)<br>Location: California<br>Other pollutants in model: none |
| chronic bronchitis | $\Delta Chronic\ Bronchitis = -\left[\frac{y_0}{(1-y_0) \cdot e^{\Delta PM_{10} \cdot \beta} + y_0} - y_0\right] \cdot \left[\frac{z_0}{y_0}\right] \cdot pop,$<br>where:<br>$y_0$ = national chronic bronchitis prevalence rate for individuals 18 and older= 0.0535<br>$z_0$ = annual bronchitis incidence rate per person = 0.00378<br>$\beta$ = estimated $PM_{10}$ logistic regression coefficient = 0.0123<br>$\Delta PM_{10}$ = change in annual average $PM_{10}$ concentration<br>pop = population ages 30 and older "without chronic bronchitis"<br>$\sigma_\beta$ = standard error of $\beta$ = 0.00434 | Study: Schwartz (1993)<br>Location: 53 U.S. urban areas<br>Other pollutants in model: none |
| hospital admissions – all respiratory | $\Delta All\ respiratory = -\left[y_0 \cdot (e^{-\beta \cdot \Delta PM_{2.5-10}} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admission rate for all respiratory per person = 2.58 E-5<br>$\beta$ = $PM_{2.5\text{-}10}$ coefficient = 0.00147<br>$\Delta PM_{2.5\text{-}10}$ = change in daily average $PM_{2.5\text{-}10}$ concentration<br>pop = population of all ages<br>$\sigma_\beta$ = standard error of $\beta$ = 0.00179 | Study: Burnett et al. (1997b)<br>Location: Toronto, Canada<br>Other pollutants in model: $O_3$, $NO_2$, $SO_2$ |
| hospital admissions – asthma | $\Delta Asthma\ Admissions = -\left[y_0 \cdot (e^{-\beta \cdot \Delta PM_{2.5-10}} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admission rate for asthma per person = 4.75 E-6<br>$\beta$ = $PM_{2.5\text{-}10}$ coefficient = 0.00321<br>$\Delta PM_{2.5\text{-}10}$ = change in daily average $PM_{2.5\text{-}10}$ concentration<br>pop = population of all ages<br>$\sigma_\beta$ = standard error of $\beta$ = 0.00106 | Study: Burnett et al. (1999)<br>Location: Toronto, Canada<br>Other pollutants in model: CO, $O_3$ |

| Health Endpoint | C-R Function | Source of C-R Function |
|---|---|---|
| hospital admissions – obstructive lung disease | $\Delta OLD\ Admissions = -\left[y_0 \cdot (e^{-\beta \cdot \Delta PM_{2.5-10}} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admission rate for obstructive lung disease per person = 5.76 E-6<br>$\beta$ = $PM_{2.5\text{-}10}$ coefficient = 0.00310<br>$\Delta$ $PM_{2.5\text{-}10}$ = change in daily average $PM_{2.5\text{-}10}$ concentration<br>pop = population of all ages<br>$\sigma_\beta$ = standard error of $\beta$ = 0.00163 | Study: Burnett et al. (1999)<br>Location: Toronto, Canada<br>Other pollutants in model: CO, $O_3$ |
| hospital admissions – respiratory infection | $\Delta Respiratory\ Infection\ Admissions = -\left[y_0 \cdot (e^{-\beta \cdot \Delta PM_{2.5}} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admission rate for respiratory infection per person = 1.56 E-5<br>$\beta$ = $PM_{2.5}$ coefficient = 0.00328<br>$\Delta PM_{2.5}$ = change in daily average $PM_{2.5}$ concentration<br>pop = population of all ages<br>$\sigma_\beta$ = standard error of $\beta$ = 0.000735 | Study: Burnett et al. (1999)<br>Location: Toronto, Canada<br>Other pollutants in model: $NO_2$, $O_3$ |
| hospital admissions – all respiratory | $\Delta all\ respiratory\ admissions = \beta \cdot \Delta PM_{2.5} \cdot pop,$<br>where:<br>$\beta$ = ozone coefficient = 1.81 E-8<br>$\Delta PM_{2.5}$ = change in daily average $PM_{2.5}$<br>pop = population all ages<br>$\sigma_\beta$ = standard error of $\beta$ = 1.79 E-8 . | Study: Thurston et al. (1994)<br>Location: Toronto, Canada<br>Other pollutants in model: $O_3$ |
| hospital admissions – pneumonia | $\Delta pneumonia\ admissions = -\left[y_0 \cdot (e^{-\beta \cdot \Delta PM_{10}} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admission rate for pneumonia per person = 5.30 E-5<br>$\beta$ = $PM_{10}$ coefficient = 0.000498<br>$\Delta$ $PM_{10}$ = change in daily average $PM_{10}$ concentration<br>pop = population age 65 and older<br>$\sigma_\beta$ = standard error of $\beta$ = 0.000505 | Study: Moolgavkar et al. (1997)<br>Location: Minneapolis, MN<br>Other pollutants in model: $O_3$, $SO_2$, $NO_2$ |
| hospital admissions – COPD | $\Delta COPD\ admissions = -\left[y_0 \cdot (e^{-\beta \cdot \Delta PM_{10}} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admission rate for COPD per person = 3.75 E-5<br>$\beta$ = $PM_{10}$ coefficient = 0.000877<br>$\Delta$ $PM_{10}$ = change in daily average $PM_{10}$ concentration<br>pop = population age 65 and older<br>$\sigma_\beta$ = standard error of $\beta$ = 0.000777 | Study: Moolgavkar et al. (1997)<br>Location: Minneapolis, MN<br>Other pollutants in model: CO, $O_3$ |

| Health Endpoint | C-R Function | Source of C-R Function |
|---|---|---|
| hospital admissions – pneumonia | $\Delta pneumonia\,admissions = -\left[y_0 \cdot (e^{-\beta \cdot \Delta PM_{10}} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admission rate for pneumonia per person = 5.18 E-5<br>$\beta$ = $PM_{10}$ coefficient = 0.00157<br>$\Delta$ $PM_{10}$ = change in daily average $PM_{10}$ concentration<br>pop = population age 65 and older<br>$\sigma_\beta$ = standard error of $\beta$ = 0.000677 | Study: Schwartz (1994c)<br>Location: Minneapolis, MN<br>Other pollutants in model: $O_3$ |
| hospital admissions – COPD | $\Delta COPD\,admissions = -\left[y_0 \cdot (e^{-\beta \cdot \Delta PM_{10}} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admission rate for COPD per person = 3.75 E-5<br>$\beta$ = $PM_{10}$ coefficient = 0.00451<br>$\Delta$ $PM_{10}$ = change in daily average $PM_{10}$ concentration<br>pop = population age 65 and older<br>$\sigma_\beta$ = standard error of $\beta$ = 0.00138 | Study: Schwartz (1994c)<br>Location: Minneapolis, MN<br>Other pollutants in model: none |
| hospital admissions – pneumonia | $\Delta pneumonia\,admissions = -\left[y_0 \cdot (e^{-\beta \cdot \Delta PM_{10}} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admission rate for pneumonia per person = 5.30 E-5<br>$\beta$ = $PM_{10}$ coefficient = 0.00174<br>$\Delta$ $PM_{10}$ = change in daily average $PM_{10}$ concentration<br>pop = population age 65 and older<br>$\sigma_\beta$ = standard error of $\beta$ = 0.000536 | Study: Schwartz (1994a)<br>Location: Birmingham, AL<br>Other pollutants in model: none |
| hospital admissions – COPD | $\Delta COPD\,admissions = -\left[y_0 \cdot (e^{-\beta \cdot \Delta PM_{10}} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admission rate for COPD per person = 3.75 E-5<br>$\beta$ = $PM_{10}$ coefficient = 0.00239<br>$\Delta$ $PM_{10}$ = change in daily average $PM_{10}$ concentration<br>pop = population age 65 and older<br>$\sigma_\beta$ = standard error of $\beta$ = 0.000838 | Study: Schwartz (1994a)<br>Location: Birmingham, AL<br>Other pollutants in model: none |
| hospital admissions – pneumonia | $\Delta pneumonia\,admissions = -\left[y_0 \cdot (e^{-\beta \cdot \Delta PM_{10}} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admission rate for pneumonia per person = 5.18 E-5<br>$\beta$ = $PM_{10}$ coefficient = 0.00115<br>$\Delta$ $PM_{10}$ = change in daily average $PM_{10}$ concentration<br>pop = population age 65 and older<br>$\sigma_\beta$ = standard error of $\beta$ = 0.00039 | Study: Schwartz (1994b)<br>Location: Detroit, MI<br>Other pollutants in model: $O_3$ |

| Health Endpoint | C-R Function | Source of C-R Function |
|---|---|---|
| hospital admissions – COPD | $\Delta COPD\ admissions = -\left[y_0 \cdot (e^{-\beta \cdot \Delta PM_{10}} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admission rate for COPD per person = 3.05 E-5<br>$\beta$ = $PM_{10}$ coefficient = 0.00202<br>$\Delta PM_{10}$ = change in daily average $PM_{10}$ concentration<br>pop = population age 65 and older<br>$\sigma_\beta$ = standard error of $\beta$ = 0.00059 | Study: Schwartz (1994b)<br>Location: Detroit, MI<br>Other pollutants in model: $O_3$ |
| hospital admissions – all respiratory | $\Delta all\ respiratory\ admissions = -\left[y_0 \cdot (e^{-\beta \cdot \Delta PM_{10}} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admission rate for all respiratory per person 65 or older = 1.187 E-4<br>$\beta$ = $PM_{10}$ coefficient = 0.00163<br>$\Delta PM_{10}$ = change in daily average $PM_{10}$ concentration<br>pop = population age 65 and older<br>$\sigma_\beta$ = standard error of $\beta$ = 0.000470 | Study: Schwartz (1996)<br>Location: Spokane, WA<br>Other pollutants in model: none |
| hospital admissions – all respiratory | $\Delta all\ respiratory\ admissions = -\left[y_0 \cdot (e^{-\beta \cdot \Delta PM_{10}} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admissions for all respiratory per person 65 and older = 1.187 E-4<br>$\beta$ = $PM_{10}$ coefficient = 0.00172<br>$\Delta PM_{10}$ = change in daily average $PM_{10}$ concentration<br>pop = population age 65 and older<br>$\sigma_\beta$ = standard error of $\beta$ = 0.000930 | Study: Schwartz (1995)<br>Location: New Haven, CT<br>Other pollutants in model: $O_3$ |
| hospital admissions – all respiratory | $\Delta all\ respiratory\ related\ admissions = -\left[y_0 \cdot (e^{-\beta \cdot \Delta PM_{10}} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admissions for all respiratory conditions per person 65 and older = 1.187 E-4<br>$\beta$ = $PM_{10}$ coefficient = 0.00227<br>$\Delta PM_{10}$ = change in daily average $PM_{10}$ concentration<br>pop = population age 65 and older<br>$\sigma_\beta$ = standard error of $\beta$ = 0.00145 | Study: Schwartz (1995)<br>Location: Tacoma, WA<br>Other pollutants in model: $O_3$ |
| hospital admissions – asthma | $\Delta Asthma\ Admissions = -\left[y_0 \cdot (e^{-\beta \cdot \Delta PM_{2.5}} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admission rate for asthma per person = 4.52 E-6<br>$\beta$ = $PM_{2.5}$ coefficient = 0.0027<br>$\Delta PM_{2.5}$ = change in daily average $PM_{2.5}$ concentration<br>pop = population of ages < 65<br>$\sigma_\beta$ = standard error of $\beta$ = 0.000948 | Study: Sheppard et al. (1999)<br>Location: Seattle, WA<br>Other pollutants in model: CO |

| Health Endpoint | C-R Function | Source of C-R Function |
|---|---|---|
| hospital admissions – cardiac | $\Delta cardiac = -\left[y_0 \cdot (e^{-\beta \cdot \Delta PM_{2.5-10}} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admission rate for cardiac per person = 3.81 E-5<br>$\beta$ = $PM_{2.5\text{-}10}$ coefficient = 0.00704<br>$\Delta PM_{2.5\text{-}10}$ = change in daily average $PM_{2.5\text{-}10}$ concentration<br>pop = population of all ages<br>$\sigma_\beta$ = standard error of $\beta$ = 0.00215 | Study: Burnett et al. (1997b)<br>Location: Toronto, Canada<br>Other pollutants in model: $O_3$ |
| hospital admissions – dysrhythmias | $\Delta Dysrhythmias\ Admissions = -\left[y_0 \cdot (e^{-\beta \cdot \Delta PM_{2.5}} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admission rate for dysrhythmias per person = 6.46 E-6<br>$\beta$ = $PM_{2.5}$ coefficient = 0.00136<br>$\Delta PM_{2.5}$ = change in daily average $PM_{2.5}$ concentration<br>pop = population of all ages<br>$\sigma_\beta$ = standard error of $\beta$ = 0.000910 | Study: Burnett et al. (1999)<br>Location: Toronto, Canada<br>Other pollutants in model: CO, $O_3$ |
| hospital admissions – ischemic heart disease | $\Delta Ischemic\ Heart\ Disease\ Admissions = -\left[y_0 \cdot (e^{-\beta \cdot \Delta PM_{10}} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admission rate for ischemic heart disease per person 65 and older = 9.96 E-5<br>$\beta$ = $PM_{10}$ coefficient= 0.000496<br>$\Delta PM_{10}$ = change in daily average $PM_{10}$ concentration<br>pop = population age 65 and older<br>$\sigma_\beta$ = standard error of $\beta$ = 0.000220 | Study: Schwartz and Morris (1995)<br>Location: Detroit, MI<br>Other pollutants in model: CO |
| hospital admissions – congestive heart failure | $\Delta Congestive\ Heart\ Failure\ Admissions = -\left[y_0 \cdot (e^{-\beta \cdot \Delta PM_{10}} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admission rate for congestive heart failure per person 65 and older = 5.82 E-5<br>$\beta$ = $PM_{10}$ coefficient = 0.000741<br>$\Delta PM_{10}$ = change in daily average $PM_{10}$ concentration<br>pop = population age 65 and older<br>$\sigma_\beta$ = standard error of $\beta$ = 0.000311 | Study: Schwartz and Morris (1995)<br>Location: Detroit, MI<br>Other pollutants in model: CO |
| hospital admissions – cardiovascular | $\Delta cardiovascular\ admissions = -\left[y_0 \cdot (e^{-\beta \cdot \Delta PM_{10}} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admission rate for cardiovascular disease per person 65 and older = 2.23 E-4<br>$\beta$ = $PM_{10}$ coefficient = 0.000737<br>$\Delta PM_{10}$ = change in daily average $PM_{10}$ concentration<br>pop = population age 65 and older<br>$\sigma_\beta$ = standard error of $\beta$ = 0.000170 | Study: Schwartz (1999)<br>Location: eight U.S. counties<br>Other pollutants in model: CO |

| Health Endpoint | C-R Function | Source of C-R Function |
|---|---|---|
| hospital admissions – cardiovascular | $\Delta cardiovascular\ admissions = -\left[y_0 \cdot (e^{-\beta \cdot \Delta PM_{10}} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admission rate for cardiovascular disease per person 65 and older = 2.23 E-4<br>$\beta$ = $PM_{10}$ coefficient = 0.00102<br>$\Delta PM_{10}$ = change in daily average $PM_{10}$ concentration<br>pop = population age 65 and older<br>$\sigma_\beta$ = standard error of $\beta$ = 0.000423 | Study: Schwartz (1997)<br>Location: Tucson, AZ<br>Other pollutants in model: CO |
| emergency room visits | $\Delta\ asthma\ visits = -\left[y_0 \cdot (e^{-\beta \cdot \Delta PM_{10}} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily ER visits for asthma per person under 65 years old = 7.69 E-6<br>$\beta$ = $PM_{10}$ coefficient (Schwartz et al., 1993, p. 829) = 0.00367<br>$\Delta PM_{10}$ = change in daily average $PM_{10}$ concentration<br>pop = population ages 0-64<br>$\sigma_\beta$ = standard error of $\beta$ (Schwartz et al., 1993, p. 829) = 0.00126 | Study: Schwartz (1993)<br>Location: Seattle, WA<br>Other pollutants in model: none |
| acute bronchitis | $\Delta Acute\ Bronchitis = -\left[\frac{y_0}{(1-y_0) \cdot e^{\Delta PM_{2.5} \cdot \beta} + y_0} - y_0\right] \cdot pop,$<br>where:<br>$y_0$ = annual bronchitis incidence rate per person = 0.044<br>$\beta$ = estimated $PM_{2.5}$ logistic regression coefficient = 0.0272<br>$\Delta PM_{2.5}$ = change in annual average $PM_{2.5}$ concentration<br>pop = population ages 8-12<br>$\sigma_\beta$ = standard error of $\beta$ = 0.0171 | Study: Dockery et al. (1996)<br>Location: 24 U.S. and Canadian cities<br>Other pollutants in model: none |
| lower respiratory symptoms (LRS) defined as cough, chest pain, phlegm, and wheeze | $\Delta Lower\ Respiratory\ Symptoms = -\left[\frac{y_0}{(1-y_0) \cdot e^{\Delta PM_{2.5} \cdot \beta} + y_0} - y_0\right] \cdot pop.$<br>where:<br>$y_0$ = daily lower respiratory symptom incidence rate per person = 0.0012<br>$\beta$ = estimated $PM_{2.5}$ logistic regression coefficient = 0.01823<br>$\Delta PM_{2.5}$ = change in daily average $PM_{2.5}$ concentration<br>pop = population ages 7-14<br>$\sigma_\beta$ = standard error of $\beta$ = 0.00586 | Study: Schwartz, et al. (1994)<br>Location: six U.S. cities<br>Other pollutants in model: none |

| Health Endpoint | C-R Function | Source of C-R Function |
|---|---|---|
| Shortness of breath, days | $$\Delta Shortness\,of\,Breath = -\left[\frac{y_0}{(1-y_0)\cdot e^{\Delta PM_{10}\cdot\beta}+y_0} - y_0\right]\cdot pop,$$ where:<br>$y_0$ = daily shortness of breath incidence rate per person = 0.056<br>$\beta$ = estimated $PM_{10}$ logistic regression coefficient = 0.00841<br>$\Delta PM_{10}$ = change in daily average $PM_{10}$ concentration<br>pop = asthmatic African-American population ages 7 to 12<br>$\sigma_\beta$ = standard error of $\beta$ = 0.00363 | Study: Ostro et al. (1995)<br>Location: Los Angeles, CA<br>Other pollutants in model: none |
| URS, defined as runny or stuffy nose, wet cough, burning, aching, or red eyes | $$\Delta Upper\,\mathrm{Re}spiratory\,Symptoms = -\left[\frac{y_0}{(1-y_0)\cdot e^{\Delta PM_{10}\cdot\beta}+y_0} - y_0\right]\cdot pop,$$ where:<br>$y_0$ = daily upper respiratory symptom incidence rate per person = 0.3419<br>$\beta$ = estimated $PM_{10}$ logistic regression coefficient = 0.0036<br>$\Delta PM_{10}$ = change in daily average $PM_{10}$ concentration<br>pop = asthmatic population ages 9 to 11 = 6.91% of population ages 9 to 11<br>$\sigma_\beta$ = standard error of $\beta$ = 0.0015 | Study: Pope et al. (1991)<br>Location: Utah Valley<br>Other pollutants in model: none |
| presence of any of 19 acute respiratory symptoms | $$\Delta ARD2 \cong \beta^*_{PM_{10}}\cdot\Delta PM_{10}\cdot pop,$$ where:<br>$\beta^*$ = first derivative of the stationary probability = 0.000461<br>$\Delta PM_{10}$ = change in daily average $PM_{10}$ concentration<br>pop = population ages 18-65<br>$\sigma_\beta$ = standard error of $\beta^*$ = 0.000239 | Study: Krupnick et al. (1990)<br>Location: Glendora-Covina-Azusa, CA<br>Other pollutants in model: $SO_2$, $O_3$<br>Comments: COH used in estimation of model. The estimated COH coefficient is used with $PM_{10}$ data. |
| moderate or worse asthma status | $$\Delta Days\,Moderate/Worse\,Asthma = -\beta\cdot\ln\left(\frac{PM_{2.5,\,after}}{PM_{2.5,\,before}}\right)\cdot pop,$$ where:<br>$\beta$ = estimated $PM_{2.5}$ coefficient for year i = 0.0006<br>$PM_{2.5}$ = change in daily average $PM_{2.5}$ concentration<br>pop = asthmatic population of all ages = 5.61% of the population of all ages<br>$\sigma_\beta$ = standard error of $\beta$ = 0.0003 | Study: Ostro et al. (1991)<br>Location: Denver<br>Other pollutants in model: none<br>Comments: The estimated coefficient is applied to populations of all ages. |

| Health Endpoint | C-R Function | Source of C-R Function |
|---|---|---|
| asthma attacks | $$\Delta asthma\,attacks = -\left[\frac{y_0}{(1-y_0)\cdot e^{\Delta PM_{10}\cdot\beta} + y_0} - y_0\right]\cdot pop,$$ where:<br>$y_0$ = daily incidence of asthma attacks = 0.027<br>$\beta$ = $PM_{10}$ coefficient = 0.00144<br>$\Delta PM_{10}$ = change in daily $PM_{10}$ concentration<br>pop = population of asthmatics of all ages = 5.61% of the population of all ages<br>$\sigma_\beta$ = standard error of $\beta$ = 0.000556 | Study: Whittemore and Korn (1980)<br>Location: Los Angeles, CA<br>Other pollutants in model: $O_3$ |
| Restricted Activity Days (RADs) | $$\Delta RAD = \Delta y \cdot pop = -\left[y_0 \cdot (e^{-\beta\cdot\Delta PM_{2.5}} - 1)\right]\cdot pop,$$ where:<br>$y_0$ = daily RAD incidence rate per person = 0.0177<br>$\beta$ = inverse-variance weighted $PM_{2.5}$ coefficient = 0.00475<br>$\Delta PM_{2.5}$ = change in daily average $PM_{2.5}$ concentration<br>pop = adult population ages 18 to 65<br>$\sigma_\beta$ = standard error of $\beta$ = 0.00029 | Study: Ostro (1987)<br>Location: U.S. metropolitan areas<br>Other pollutants in model: none<br>Comments: An inverse-variance weighting used to estimate the coefficient, based on Ostro (1987, Table III)]. |
| respiratory and nonrespiratory conditions resulting in a minor restricted activity day (MRAD) | $$\Delta MRAD = \Delta y \cdot pop = -\left[y_0 \cdot (e^{-\beta\cdot\Delta PM_{2.5}} - 1)\right]\cdot pop,$$ where:<br>$y_0$ = daily MRAD daily incidence rate per person = 0.02137<br>$\beta$ = inverse-variance weighted $PM_{2.5}$ coefficient = 0.00741<br>$\Delta PM_{2.5}$ = change in daily average $PM_{2.5}$ concentration<br>pop = adult population ages 18 to 65<br>$\sigma_\beta$ = standard error of $\beta$ = 0.0007 | Study: Ostro and Rothschild (1989b)<br>Location: U.S.<br>Other pollutants in model: $O_3$<br>Comments: An inverse-variance weighting used to estimate the coefficient, based on Ostro and Rothschild (1989b, Table 4) |
| work loss days (WLDs) | $$\Delta WLD = \Delta y \cdot pop = -\left[y_0 \cdot (e^{-\beta\cdot\Delta PM_{2.5}} - 1)\right]\cdot pop,$$ where:<br>$y_0$ = daily work-loss-day incidence rate per person = 0.00648<br>$\beta$ = inverse-variance weighted $PM_{2.5}$ coefficient = 0.0046<br>$\Delta PM_{2.5}$ = change in daily average $PM_{2.5}$ concentration<br>pop = population ages 18 to 65<br>$\sigma_\beta$ = standard error of $\beta$ = 0.00036 | Study: Ostro (1987)<br>Location: U.S. metropolitan areas<br>Other pollutants in model: none<br>Comments: An inverse-variance weighting used to estimate the coefficient, based on Ostro (1987, Table III). |

### Sulfur Dioxide

The C-R functions used to estimate the impact of sulfur dioxide are summarized in Table D-19.

**Table D-19**
**Summary of C-R Functions for Sulfur Dioxide**

| Health Endpoint | Concentration-Response Function | Source of C-R Function |
|---|---|---|
| hospital admissions – all respiratory | $\Delta All\,respiratory = -\left[y_0 \cdot (e^{-\beta \cdot \Delta SO_2} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admission rate for all respiratory per person = 2.58 E-5<br>$\beta$ = $SO_2$ coefficient = 0.00446<br>$\Delta SO_2$ = change in daily one-hour maximum $SO_2$ concentration (ppb)<br>pop = population of all ages<br>$\sigma_\beta$ = standard error of $\beta$ = 0.00293 | Study: Burnett et al. (1997b)<br>Location: Toronto, Canada<br>Other pollutants in model: $PM_{2.5\text{-}10}$, $NO_2$, $O_3$ |
| hospital admissions – pneumonia | $\Delta pneumonia\,admissions = -\left[y_0 \cdot (e^{-\beta \cdot \Delta SO_2} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admission rate for pneumonia per person = 5.30 E-5<br>$\beta$ = $SO_2$ coefficient = 0.00143<br>$\Delta$ $SO_2$ = change in daily average $SO_2$ concentration (ppb)<br>pop = population age 65 and older<br>$\sigma_\beta$ = standard error of $\beta$ = 0.00290 | Study: Moolgavkar et al. (1997)<br>Location: Minneapolis, MN<br>Other pollutants in model: $O_3$, $NO_2$ $PM_{10}$ |
| hospital admissions – ischemic heart disease | $\Delta Ischemic\,Heart\,Disease\,Admissions = -\left[y_0 \cdot (e^{-\beta \cdot \Delta SO_2} - 1)\right] \cdot pop,$<br>where:<br>$y_0$ = daily hospital admission rate for ischemic heart disease per person = 2.23 E-5<br>$\beta$ = $SO_2$ coefficient = 0.00177<br>$\Delta SO_2$ = change in daily average $SO_2$ concentration<br>pop = population of all ages<br>$\sigma_\beta$ = standard error of $\beta$ = 0.000854 | Study: Burnett et al. (1999)<br>Location: Toronto, Canada<br>Other pollutants in model: $NO_2$ |
| chest tightness, shortness of breath, or wheeze | $\Delta symptoms = \left[\frac{1}{1+e^{-\alpha - SO_{2,pre-CAAA} \cdot \beta - \gamma}} - \frac{1}{1+e^{-\alpha - SO_{2,post-CAAA} \cdot \beta - \gamma}}\right] \cdot pop,$<br>.where:<br>$\alpha$ = constant = -5.65<br>$\beta$ = $SO_2$ coefficient = 0.00589<br>$\gamma$ = status coefficient (used for moderate asthmatics only) = 1.10<br>$SO_2$ = peak five minute $SO_2$ concentration (ppb) in an hour = hourly $SO_2$ concentration (ppb) multiplied by 2.5 peak to mean ratio of 2.5<br>pop = exercising asthmatics = population of asthmatics of all ages (5.61% of the population of all ages (Adams et al., 1995 Table 57)) of whom 1.7% are exercising. Moderate asthmatics compose one third of exercising asthmatics; mild asthmatics compose the other two thirds (U.S. EPA, 1997, p. D-39).<br>$\sigma_\beta$ = standard error of $\beta$ = 0.00247 | Study: Linn et al. (1987; 1988; 1990) and Roger et al. (1985)<br>Location: Chamber study<br>Other pollutants in model: none<br>Comments: The results of four chamber studies were combined to develop this C-R function. Moderate asthmatics compose one third of exercising asthmatics; mild asthmatics compose the other two thirds. |

## Modeling Results

This section presents the results of the health effects modeling resulting from improvements in air quality between the Pre-CAAA and Post-CAAA scenarios for the years 2000 and 2010. Tables D-20 and D-21 summarize the health effects for each study included in the analysis, presenting the mean, as well as the estimated credible interval ($5^{th}$ and $95^{th}$ percentiles) of the number of avoided cases of each endpoint. Table D-20 presents these results for the subpopulation living within 50 kilometers of an air quality monitor. Table D-21 presents results for the entire population of the 48 contiguous states. Table D-22 summarizes the life-years lost by age group; Tables D-23 and D-24 present illustrative calculations of the impact of air pollution on mortality; and Figure D-2 presents the results of using alternative effect thresholds in the calculation of mortality.

The ranges of estimates presented in Tables D-20 and D-21 reflect the measured uncertainty inherent in the estimated C-R coefficients used in calculating the avoided incidence for each endpoint. These ranges are only a partial measure of the total uncertainty associated with the estimation of the avoided incidence of each health effect. There are other potentially important sources of uncertainty in this benefits analysis that would likely lead to a wider uncertainty range. For example, some of the analytical components are point estimates that do not incorporate information about the uncertainty inherent in the estimates, such as the emissions and air quality estimates. A complete depiction of the uncertainty of the estimates would include the uncertainty in these important analytical components. Incorporating quantitative uncertainty estimates into each of these components is not feasible for this current analysis. Therefore, the range of estimates presented herein is only a partial reflection of the total uncertainty range.

### *Uncertainty*

The stated goal of this study is to provide a comprehensive estimate of the benefits of the Clean Air Act Amendments of 1990. To achieve this goal, information with very different levels of confidence must be used. The analysis presents information on the plausible range of estimates through the use of two approaches. The first approach is to reflect the measured uncertainty in estimating the avoided incidence of health effects by using an estimated probability distribution for each C-R coefficient used in the analysis. The second approach is to present analysis using different key assumptions. The threshold choice, the time between PM exposure and mortality, the choice of studies, and whether to estimate mortality using statistical life years or statistical lives lost are important assumptions that are examined in this analysis.

To capture the variation in the C-R function coefficient estimates used to estimate the avoided incidence of health effects, this analysis uses a Monte Carlo procedure to generate distributions of estimated effects by randomly sampling the distribution of coefficients (given by the mean coefficient and standard deviation reported in the literature) and then evaluating the C-R function with the randomly selected coefficient. This yielded an estimate of avoided incidence for the given effect and was repeated many times to generate distributions of avoided incidence. Both the mean estimates and the $5^{th}$ and $95^{th}$ percentile estimates of the resulting distributions of avoided incidence estimates are presented here for each health effect.

The second type of uncertainty considered here addresses the fact that different published results reported in the scientific literature typically do not report identical findings; in some instances the differences are substantial. For this analysis, some health endpoints used more than one concentration-response function, each representing a different study. The alternative concentration-response functions provide differing measures of the effect air quality reductions have on changes in particular health endpoints. This between-study variability is captured by considering the range of estimates for a given endpoint, and can be used to derive a range of possible results. For example, concentration-response functions for developing chronic bronchitis from

three different studies are used to estimate the range of avoided cases of chronic bronchitis.

Another important source of uncertainty that is considered as an alternative analysis is the estimation of statistical life years lost. Table D-22 presents the percentage of lives lost for each age group considered and the average number of life years lost. The majority of the estimated deaths occur in people over the age of 65 (due to their higher baseline mortality rates), and this group has a short life expectancy relative to other age groups.

## Sensitivity Analyses

One particularly important uncertainty is the impact that alternative threshold assumptions have on both the estimates of specific health effects and ultimately on monetary benefits. The available evidence has failed to identify thresholds – or safe levels of air pollution – for any of the effects associated with criteria pollutants, so this analysis assumes that there are no effective thresholds and that air pollution has effects down to zero ambient levels. Nevertheless, thresholds may exist and their potential impact on the overall benefits analysis could be substantial. Any of the health effects estimated in this analysis could have a threshold; however a threshold for PM-related mortality would have the greatest impact on the overall benefits analysis. Figure D-2 shows the effect of incorporating a range of possible thresholds, using 2010 PM levels and the Pope et al. (1995) study.

Pope et al. (1995) did not explicitly include a threshold in their analysis. However, if the true mortality C-R relationship has a threshold, then Pope et al.'s slope coefficient would likely have been underestimated for that portion of the C-R relationship above the threshold. This would likely lead to an underestimate of the incidences of avoided cases above any assumed threshold level. It is difficult to determine the size of the underestimate without data on a likely threshold and without re-analyzing the Pope et al. data.

The quantitative results of several other sensitivity analyses are also presented. As discussed above, there is information suggesting a possible relationship between ozone and premature mortality, and between PM and infant mortality. However, there is considerable uncertainty about these relationships at this time, so quantitative estimates of these effects are not included in the aggregated results. The possible magnitude of these health effects are explored as sensitivity analyses, reported in Tables D-23 (for the population within 50 kilometers of a monitor) and D-24 (for the entire population of the 48 contiguous states). In addition, the results of an alternative estimate of the premature adult mortality associated with long-term PM exposure based on Dockery et al. (1993) are also presented in Tables D-23 and D-24. The Dockery et al. study used a smaller sample of individuals from fewer cities than the study by Pope et al., although it features improved exposure estimates, a slightly broader study population, and a follow-up period nearly twice as long as that of Pope et al. The results based on Dockery are presented only as sensitivity calculation for this important health effect; the Pope et al. (1995) estimate is used in the primary analysis.

Finally, this study includes a sensitivity analysis illustrating the effect of alternative assumptions about a potential lag between PM exposure and premature mortality on monetized benefit estimates. As discussed earlier, a change in the assumed lag period will have no effect on the total estimate of avoided mortality presented in Table D-21; it will only affect the distribution of those avoided deaths through time. Changes to this distribution will, however, affect monetized benefit estimates if the values of the avoided future deaths are discounted. Therefore, although we discuss the various lag scenarios here, the results of this sensitivity analysis are presented in the valuation appendix, Appendix H.

Before describing the lag scenarios, we emphasize that no scientific evidence currently exists to support the assumption of a significant lag (i.e., several years or more) between PM exposure and premature mortality. The prospective cohort study design of long-term epidemiological studies of PM

exposure (including Pope et al., 1995) provides no information about whether a lag exists or whether a particular length of exposure is required to elicit an effect. Further, we have identified no studies specifically designed to test for such a PM/mortality lag. However, we have incorporated a lag into our primary analysis and conducted this sensitivity analysis for two reasons. First, other similar types of exposures, such as cigarette smoking, do show evidence of a lag. Studies of reductions in cigarette smoking suggest that the benefits of smoking cessation occur over a several year period. Second, differences between the relative risk estimates of short-term studies of PM exposure and those of long-term (i.e., cohort) studies may suggest the presence of a lag for some portion of the overall mortality effect of PM exposure.

Short-term epidemiological studies linking daily measures of PM exposures with daily mortality rates show statistically significant increases in mortality within days of increased PM exposure. However, the appropriate lag period for the portion of deaths that do not occur immediately is unclear. Some interpret the analysis by Brunekreef (1997) as indicative of a much longer mortality lag of 15 years; however, it appears that Brunekreef simply employed assumptions consistent with the cohort design of the Dockery et al., 1993 study, which examined the relative risk for a cohort aged 25 to 74 over a 15-year period. The selection of such a follow-up period by Dockery et al. was not based on biological or epidemiological evidence of a 15-year lag, and Brunekreef cites no evidence supporting a lag of this length. Therefore, we do not find the Brunekreef (1997) study to be convincing evidence of a fifteen-year lag.

Table D-25 compares the distribution of avoided mortality benefits assumed in the primary analysis with each of the sensitivity analysis scenarios. In the primary analysis, we apply the same lag structure used as a sensitivity analysis in the draft RIA for the proposed Tier 2 motor vehicle emission standards (U.S. EPA, 1999). Under this scenario, the avoided mortality occurs over a five year period, with fifty percent of the avoided mortality occurring within the first two years (i.e. 25 percent per year), and the remainder of avoided deaths distributed evenly across the last three years (approximately 16.7 percent per year). As mentioned above, the appropriate length of the lag period is highly uncertain, and so is the distribution of deaths over that period, although evidence from short-term studies suggests weighting the distribution toward the first couple of years following exposure. The assumptions of the Tier 2 lag structure reflect the best judgment of the Agency on this issue; however, they do not represent any known lag structure for PM mortality.

We evaluate three lag scenarios for the sensitivity analysis. The first scenario assumes no lag; that is, all avoided mortality occurs in the same year as exposure (Year 1 in Table D-25). The second distributes avoided mortality evenly across eight years; this scenario is based on the eight-year cohort follow-up period of the Pope et al., 1995 study. The third scenario is based on the Dockery et al., 1993 follow-up period of 15 years, with avoided mortality distributed evenly across that period. As discussed earlier, we find the 15-year lag to be an extremely conservative assumption.

The effect of these different lag assumptions on our estimate of monetary benefits depends on the discount rate used. Given the discount rate used in the primary analysis, five percent, the no lag scenario would increase the primary mortality reduction benefits estimate by nine percent; the Pope-based lag estimate would decrease the estimate by eight percent; and the Dockery-based lag estimate would decrease the estimate by 21 percent. The actual monetary benefit estimates generated by this sensitivity analysis are presented in Appendix H.

**Table D-20**
**Change in Incidence of Adverse Health Effects Associated with Criteria Pollutants (Pre-CAAA minus Post-CAAA) – 48 State U.S. Population within 50 km of a Monitor (avoided cases per year)**

| | | 2000 | | | 2010 | | | % of Baseline Incidences for the mean estimates [a] | |
|---|---|---|---|---|---|---|---|---|---|
| **Endpoint** | **Pollutant** | **5th %** | **mean** | **95th %** | **5th %** | **mean** | **95th %** | **2000** | **2010** |
| **Mortality** | | | | | | | | | |
| ages 30 and older | PM | 7,900 | 13,000 | 18,000 | 13,000 | 20,000 | 28,000 | 0.67%[b] | 0.95%[b] |
| **Chronic Illness** | | | | | | | | | |
| chronic bronchitis | PM | 2,300 | 11,000 | 20,000 | 4,300 | 18,000 | 30,000 | 2.05% | 3.09% |
| chronic asthma | $O_3$ | 910 | 4,900 | 8,300 | 1,100 | 5,500 | 8,700 | 3.46% | 3.59% |
| **Hospitalization** | | | | | | | | | |
| respiratory admissions | PM, CO, $NO_2$, $SO_2$, $O_3$ | 7,300 | 12,000 | 17,000 | 12,000 | 19,000 | 29,000 | 0.52% | 0.76% |
| cardiovascular admissions | PM, CO, $NO_2$, $SO_2$, $O_3$ | 5,400 | 19,000 | 41,000 | 9,400 | 38,000 | 90,000 | 0.59% | 1.10% |
| emergency room visits for asthma | PM, $O_3$ | 200 | 1,300 | 6,600 | 310 | 2,000 | 10,000 | 0.19% | 0.27% |
| **Minor Illness** | | | | | | | | | |
| acute bronchitis | PM | 0[c] | 25,000 | 49,000 | 0[c] | 40,000 | 79,000 | 3.19% | 4.71% |
| upper respiratory symptoms | PM | 170,000 | 570,000 | 970,000 | 260,000 | 870,000 | 1,500,000 | 0.61% | 0.86% |
| lower respiratory symptoms | PM | 130,000 | 270,000 | 420,000 | 210,000 | 440,000 | 670,000 | 2.19% | 3.30% |
| respiratory illness | $NO_2$ | 24,000 | 110,000 | 180,000 | 63,000 | 270,000 | 450,000 | 7.62% | 17.29% |
| moderate or worse asthma[d] | PM | 32,000 | 210,000 | 370,000 | 48,000 | 310,000 | 570,000 | 0.15% | 0.21% |
| asthma attacks[d] | $O_3$, PM | 520,000 | 960,000 | 1,400,000 | 800,000 | 1,500,000 | 2,100,000 | 0.74% | 1.06% |

| | | 2000 | | | 2010 | | | % of Baseline Incidences for the mean estimates [a] | |
|---|---|---|---|---|---|---|---|---|---|
| **Endpoint** | **Pollutant** | **5th %** | **mean** | **95th %** | **5th %** | **mean** | **95th %** | **2000** | **2010** |
| chest tightness, shortness of breath, or wheeze | $SO_2$ | 200 | 80,000 | 370,000 | 270 | 100,000 | 470,000 | 0.003% | 0.004% |
| shortness of breath | PM | 16,000 | 57,000 | 95,000 | 25,000 | 88,000 | 150,000 | 1.25% | 1.79% |
| work loss days | PM | 1,900,000 | 2,200,000 | 2,500,000 | 3,100,000 | 3,500,000 | 4,000,000 | 0.60% | 0.87% |
| minor restricted activity days / any of 19 respiratory symptoms[e] | $O_3$, PM | 14,000,000 | 17,000,000 | 20,000,000 | 22,000,000 | 27,000,000 | 32,000,000 | 1.47% | 2.16% |
| restricted activity days[d] | PM | 5,500,000 | 6,200,000 | 6,800,000 | 9,000,000 | 10,000,000 | 11,000,000 | 0.61% | 0.91% |

[a] The baseline incidence generally is the same as that used in the C-R function for a particular health effect. However, there are a few exceptions. To calculate the baseline incidence rate for respiratory-related hospital admissions, we used admissions for persons of all ages for ICD codes 460-519; for cardiovascular admissions, we used admissions for persons of all ages for ICD codes 390-429; for emergency room visits for asthma, we used the estimated ER visit rate for persons of all ages; for chronic bronchitis we used the incidence rate for individuals 27 and older; for the pooled estimate of minor restricted activity days and any-of-19 respiratory symptoms, we used the incidence rate for minor restricted activity days.

[b] Calculated as the ratio of avoided mortality to the projected baseline annual non-accidental mortality for adults aged 30 and over. Non-accidental mortality was approximately 95% of total mortality for this subpopulation in 2010.

[c] Monte Carlo modeling returned a negative value for the fifth percentile estimate of this endpoint. However, we believe the negative result represents an artifact of the statistical methods employed in the uncertainty analysis, since none of the studies used in the health benefits analysis suggest a negative correlation between criteria air pollutant exposure and this health endpoint. We therefore truncate this value at zero for presentation. The full distribution of estimates, including negative values, is used in all aggregations of benefits estimates presented in this document.

[d] These health endpoints overlap with the "any-of-19 respiratory symptoms" category. As a result, although we present estimates for each endpoint individually, these results are not aggregated into the total benefits estimates.

[e] Minor restricted activity days and any-of-19 respiratory symptoms have overlapping definitions and are pooled.

**Table D-21**
**Change in Incidence of Adverse Health Effects Associated with Criteria Pollutants (Pre-CAAA minus Post-CAAA) – 48 State U.S. Population (avoided cases per year)**

| | | 2000 | | | 2010 | | | % of Baseline Incidences for the mean estimates [a] | |
|---|---|---|---|---|---|---|---|---|---|
| **Endpoint** | **Pollutant** | **5th %** | **mean** | **95th %** | **5th %** | **mean** | **95th %** | **2000** | **2010** |
| **Mortality** | | | | | | | | | |
| ages 30 and older | PM | 8,800 | 14,000 | 19,000 | 14,000 | 23,000 | 32,000 | 0.66%[b] | 1.00%[b] |
| **Chronic Illness** | | | | | | | | | |
| chronic bronchitis | PM | 3,100 | 13,000 | 22,000 | 5,000 | 20,000 | 34,000 | 2.21% | 3.14% |
| chronic asthma | $O_3$ | 1,300 | 5,600 | 9,600 | 1,800 | 7,200 | 12,000 | 3.22% | 3.83% |
| **Hospitalization** | | | | | | | | | |
| respiratory admissions | PM, CO, $NO_2$, $SO_2$, $O_3$ | 8,100 | 13,000 | 20,000 | 13,000 | 22,000 | 34,000 | 0.40% | 0.62% |
| cardiovascular admissions | PM, CO, $NO_2$, $SO_2$, $O_3$ | 5,800 | 22,000 | 48,000 | 10,000 | 42,000 | 100,000 | 0.49% | 0.86% |
| emergency room visits for asthma | PM, $O_3$ | 260 | 3,100 | 8,900 | 430 | 4,800 | 14,000 | 0.39% | 0.55% |
| **Minor Illness** | | | | | | | | | |
| acute bronchitis | PM | 0[c] | 29,000 | 59,000 | 0[c] | 47,000 | 94,000 | 3.39% | 5.06% |
| upper respiratory symptoms | PM | 180,000 | 620,000 | 1,000,000 | 280,000 | 950,000 | 1,600,000 | 0.61% | 0.86% |
| lower respiratory symptoms | PM | 150,000 | 320,000 | 480,000 | 240,000 | 520,000 | 770,000 | 2.38% | 3.57% |
| respiratory illness | $NO_2$ | 31,000 | 130,000 | 220,000 | 76,000 | 330,000 | 550,000 | 4.46% | 10.44% |
| moderate or worse asthma[d] | PM | 52,000 | 260,000 | 460,000 | 80,000 | 400,000 | 720,000 | 0.17% | 0.24% |
| asthma attacks[d] | $O_3$, PM | 590,000 | 1,100,000 | 1,600,000 | 920,000 | 1,700,000 | 2,500,000 | 0.73% | 1.04% |

| | | 2000 | | | 2010 | | | % of Baseline Incidences for the mean estimates [a] | |
|---|---|---|---|---|---|---|---|---|---|
| **Endpoint** | **Pollutant** | **5th %** | **mean** | **95th %** | **5th %** | **mean** | **95th %** | **2000** | **2010** |
| chest tightness, shortness of breath, or wheeze | $SO_2$ | 220 | 88,000 | 410,000 | 290 | 110,000 | 520,000 | 0.002% | 0.003% |
| shortness of breath | PM | 16,000 | 59,000 | 98,000 | 26,000 | 91,000 | 150,000 | 1.19% | 1.69% |
| work loss days | PM | 2,200,000 | 2,500,000 | 2,900,000 | 3,600,000 | 4,100,000 | 4,600,000 | 0.62% | 0.94% |
| minor restricted activity days / any of 19 respiratory symptoms[e] | $O_3$, PM | 16,000,000 | 19,000,000 | 23,000,000 | 25,000,000 | 31,000,000 | 37,000,000 | 1.43% | 2.15% |
| restricted activity days[d] | PM | 6,400,000 | 7,200,000 | 7,900,000 | 10,000,000 | 12,000,000 | 13,000,000 | 0.65% | 1.00% |

[a] The baseline incidence generally is the same as that used in the C-R function for a particular health effect. However, there are a few exceptions. To calculate the baseline incidence rate for respiratory-related hospital admissions, we used admissions for persons of all ages for ICD codes 460-519; for cardiovascular admissions, we used admissions for persons of all ages for ICD codes 390-429; for emergency room visits for asthma, we used the estimated ER visit rate for persons of all ages; for chronic bronchitis we used the incidence rate for individuals 27 and older; for the pooled estimate of minor restricted activity days and any-of-19 respiratory symptoms, we used the incidence rate for minor restricted activity days.

[b] Calculated as the ratio of avoided mortality to the projected baseline annual non-accidental mortality for adults aged 30 and over. Non-accidental mortality was approximately 95% of total mortality for this subpopulation in 2010.

[c] Monte Carlo modeling returned a negative value for the fifth percentile estimate of this endpoint. However, we believe the negative result represents an artifact of the statistical methods employed in the uncertainty analysis, since none of the studies used in the health benefits analysis suggest a negative correlation between criteria air pollutant exposure and this health endpoint. We therefore truncate this value at zero for presentation. The full distribution of estimates, including negative values, is used in all aggregations of benefits estimates presented in this document.

[d] These health endpoints overlap with the "any-of-19 respiratory symptoms" category. As a result, although we present estimates for each endpoint individually, these results are not aggregated into the total benefits estimates.

[e] Minor restricted activity days and any-of-19 respiratory symptoms have overlapping definitions and are pooled.

**Table D-22**
**Mortality Distribution by Age in Primary Analysis, Based on Pope et al. (1995)**

| Age Group | Proportion of Premature Mortality by Age [a] | Life Expectancy (years) |
|---|---|---|
| Infants | not estimated | -- |
| 1-29 | not estimated | -- |
| 30-34 | 1% | 48 |
| 35-44 | 4% | 38 |
| 45-54 | 6% | 29 |
| 55-64 | 12% | 21 |
| 65-74 | 24% | 14 |
| 75-84 | 30% | 9 |
| 85+ | 24% | 6 |

[a] Percentages sum to 101 percent due to rounding.

**Table D-23**
**Illustrative Estimates of the Impact of Criteria Pollutants on Mortality – 48 State U.S. Population within 50 km of a Monitor (cases per year)**

| | | 2000 | | | 2010 | | | % of Baseline Incidences for the mean estimates | |
|---|---|---|---|---|---|---|---|---|---|
| **Endpoint** | **Pollutant** | **5th %** | **mean** | **95th %** | **5th %** | **mean** | **95th %** | **2000** | **2010** |
| ages 30 and older (Pope et al., 1995) | PM | 7,900 | 13,000 | 18,000 | 13,000 | 20,000 | 28,000 | 0.67% | 0.95% |
| ages 25 and older (Dockery et al., 1993)* | PM | 12,000 | 29,000 | 46,000 | 20,000 | 47,000 | 73,000 | 1.35% | 2.19% |
| all ages * | $O_3$ | 81 | 1,100 | 2,200 | 130 | 1,600 | 3,400 | 0.06% | 0.08% |
| post-neonatal * | PM | 39 | 81 | 120 | 59 | 120 | 180 | 0.93% | 1.39% |

*The Dockery et al. (1993), ozone mortality, and post-neonatal mortality estimates are not aggregated into total benefits estimates.

**Table D-24**
**Illustrative Estimates of the Impact of Criteria Pollutants on Mortality – 48 State U.S. Population (cases per year)**

| | | 2000 | | | 2010 | | | % of Baseline Incidences for the mean estimates | |
|---|---|---|---|---|---|---|---|---|---|
| **Endpoint** | **Pollutant** | **5th %** | **mean** | **95th %** | **5th %** | **mean** | **95th %** | **2000** | **2010** |
| ages 30 and older (Pope et al., 1995) | PM | 8,800 | 14,000 | 19,000 | 14,000 | 23,000 | 32,000 | 0.66% | 1.00% |
| ages 25 and older (Dockery et al., 1993)* | PM | 15,987 | 34,860 | 54,677 | 26,000 | 56,000 | 88,000 | 1.60% | 2.39% |
| all ages * | $O_3$ | 0† | 1,400 | 2,800 | 0† | 2,200 | 4,600 | 0.07% | 0.09% |
| post-neonatal * | PM | 45 | 88 | 130 | 69 | 130 | 200 | 1.02% | 1.38% |

*The Dockery et al. (1993), ozone mortality, and post-neonatal mortality estimates are not aggregated into total benefits estimates.

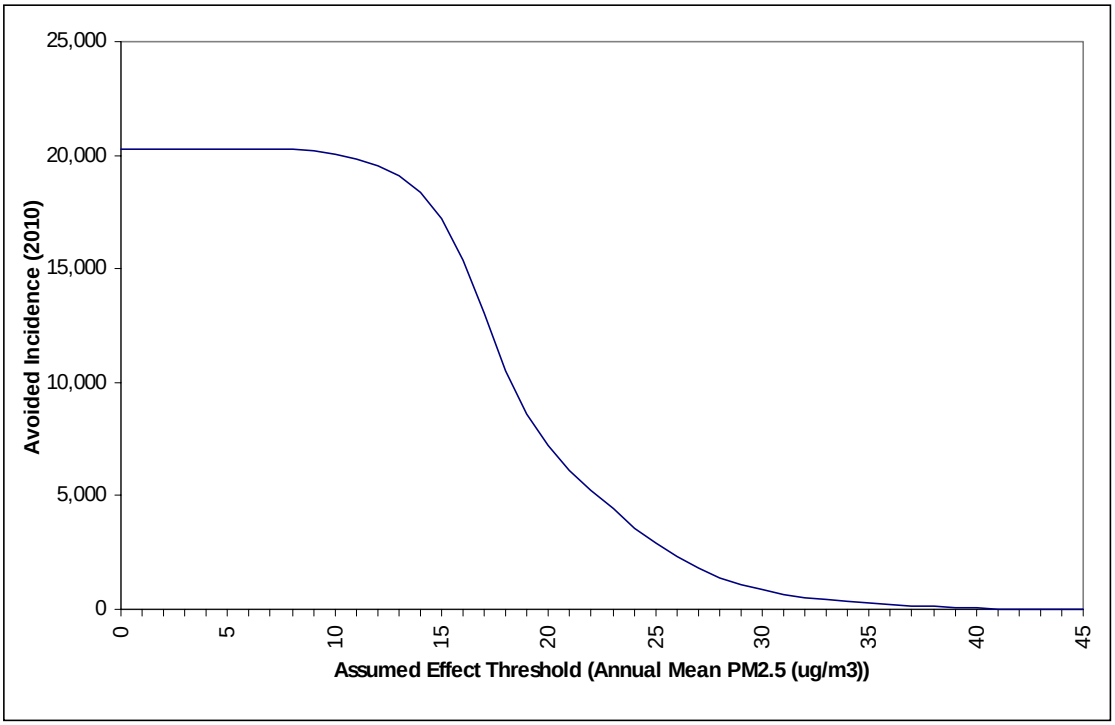
†Monte Carlo modeling returned a negative value for the fifth percentile estimate of this endpoint. However, we believe the negative result represents an artifact of the statistical methods employed in the uncertainty analysis, since none of the studies used in the health benefits analysis suggest a negative correlation between criteria air pollutant exposure and this health endpoint. We therefore truncate this value at zero for presentation. The full distribution of estimates, including negative values, is used in all aggregations of benefits estimates presented in this document.

**Table D-25**
**Comparison of Alternative Lag Assumptions for Premature Mortality Associated with PM Exposure**

| Year | Percent of Avoided Mortality By Year | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 |
| Tier II SA Lag (Primary Estimate) | 25 | 25 | 16.67 | 16.67 | 16.67 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| No Lag | 100 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Lag Distributed Evenly Over the Period Covered by Pope et al., 1995 | 12.5 | 12.5 | 12.5 | 12.5 | 12.5 | 12.5 | 12.5 | 12.5 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Lag Distributed Evenly Over the Period Covered by Dockery et al., 1993 | 6.67 | 6.67 | 6.67 | 6.67 | 6.67 | 6.67 | 6.67 | 6.67 | 6.67 | 6.67 | 6.67 | 6.67 | 6.67 | 6.67 | 6.67 |

Totals may not sum to 100 percent due to rounding.

**Figure D-2**
**Long-Term Mortality Based on Pope (1995): National Avoided Incidence Estimates (2010) at Different Assumed Effect Thresholds, Based on a 50 Km Maximum Distance**



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# Ecological Effects

Appendix E

## Introduction

This appendix characterizes the ecological benefits of the Clean Air Act Amendments. Although EPA's focus on a clean environment has long included protection of both ecosystem health and human health, many past analyses, particularly economic analyses, have focused on human health benefits of pollution control. Ecological benefits, by comparison, have not always been as well-represented, for a variety of reasons:

- Ecological impacts may be complex and non-linear, involving relationships at various levels of biological organization. Important ecological effects such as population decline of a keystone species can ripple through a food web and alter community structure and ecosystem function.

- Ecological systems, like human bodies, possess a wide range of adaptive capacities that can mitigate or mask effects and make them difficult to detect. What differentiates, and further complicates the measurement of ecological effects is the lack of sufficient baseline data on natural ecosystem structure and function though successional stages.

- Prevention of ecological effects may be viewed by the public and decision-makers as a lower priority than the protection of human health.

Nonetheless, within the last few decades air pollution started to receive attention for not only affecting human health but also its dramatic injuries to ecosystems. Increased public awareness and research results have led to the development of air pollution research as an important branch of applied biological sciences. Numerous scientific studies have revealed adverse effects of air pollution on natural systems that have, in turn, led to increasingly heightened levels of public concern and subsequent environmental statutory developments. Public policy concerning the regulation of air pollution to mitigate these impacts requires accurate appraisals of the effectiveness of regulatory options, but not until quite recently has it become possible to reliably quantify at least some of the ecological and economic benefits of ecosystem impacts linked to air pollution.

This analysis attempts to incrementally expand the base of quantitative and qualitative information that can be used to assess affects to ecosystems associated with air pollution. There are two major goals of the analysis: to provide a broad overall characterization of the range of effects of air pollutants on ecosystem structure, function, and health; and to extend existing methods and data to characterize the potential magnitude of economic benefits derived from the 1990 Clean Air Act Amendments (CAAA). The economic analysis is focused on a relatively small subset of effects for which ecologists' and economists' understanding of and ability to model an effect is sufficient to develop a quantitative characterization. In most cases, we rely on published, peer-reviewed literature to establish the validity of the methods and data applied.

The remainder of this appendix is comprised of seven major sections. We first provide a broad overview of the ecological impacts of the air pollutants regulated by the CAAA, and then outline the rationale for choosing a subset of these effects for quantitative and economic analysis. Following this largely qualitative characterization of effects, we describe the methods, data, and results used to quantitatively assess benefits of the Clean Air Act Amendments for the following categories of effects:

- Eutrophication of estuaries associated with airborne nitrogen deposition;
- Acidification of freshwater bodies associated with airborne nitrogen and sulfur deposition;
- Reduced tree growth associated with ozone exposure;
- Accumulation of toxics in freshwater fisheries associated with airborne toxics deposition;
- Aesthetic degradation of forests associated with ozone and airborne toxics exposure;
- Other less well-understood effects of air pollution on ecosystem health.

The concluding section includes a summary of those economic estimates that are used in the larger 812 analysis, a summary of major limitations, and recommendations for future research.[1]

Because the breadth and complexity of air pollutant-ecosystem interactions does not allow for comprehensive quantitative analysis of all the ecological benefits of the CAAA we stress the importance of continued consideration of those impacts not valued in this report in policy decision-making and in further technical research. Judging from the geographic breadth and magnitude of the relatively modest subset of impacts that we find sufficiently well-understood to quantify and monetize, it is evident that the economic benefits of the CAAA's reduction of air pollution impacts on ecosystems are of a large magnitude.

[1] More detailed documentation of the ecological benefits of the CAAA can be found in a series of memoranda and other work products prepared as a part of EPA's benefits analysis and research effort. These memoranda provide comprehensive descriptions of the ecological impacts avoided by the CAAA, methods used to characterize those damages, data sources, and ecological and economic benefits assessments. The more detailed documentation can be obtained through the EPA contacts identified in the Acknowledgements section of the overall report.

## Ecological Overview of the Impacts of Air Pollutants Regulated by the CAAA

The purpose of this section is to provide an overview of potential interactions between air pollutants and the natural environment. We identify major single pollutant-environment interactions, as well as the synergistic impacts of ecosystem exposure to multiple air pollutants. Although a wide variety of complex air pollution-environment interactions are described or hypothesized in the literature, for the purposes of this analysis we focus on major aspects of ecosystem-pollutant interactions. We do this by limiting our review according to the following criteria:

- Pollutants regulated by the CAAA;
- Known interactions between pollutants and natural systems as documented in peer-reviewed literature; and,
- Pollutants present in the atmosphere in sufficient amounts after 1970 to cause significant damages to natural systems.

Our understanding of air pollution effects on ecosystems has progressed considerably during the past decades. Previously, air pollution was regarded primarily as a local phenomenon and concern was associated with the vicinity of industrial facilities, power plants or urban areas. The pollutants of concern were gaseous (e.g., sulfur dioxide and ozone) or heavy metals (e.g., lead) and the observed effects were visible stress- specific symptoms of injury (e.g., foliar chlorosis). The most typical approach to document the effects of specific pollutants was a dose-response experiment, where the objective was to develop a regression equation describing the relationship between exposure and some easily measured effect (e.g., growth, yield or mortality). As analytic methods improved and ecology progressed, a broader range of effects of air pollutants were identified and understanding of the mechanisms of effect improved. Observations made on various temporal scales (e.g., long-term studies) and spatial scales (e.g., watershed studies) lead to the recognition

that air pollution can affect all organizational levels of biological systems.

In this analysis, we attempt to broadly describe the impacts of air pollutants at all levels of organization, though we are constrained to a review of the most significant impacts on a national scale. For a comprehensive review of the ecological impacts of air pollutants regulated under the CAAA, see *Overview of Ecological Impacts of Air Pollutants Regulated by the 1990 Clean Air Act Amendments* (EPA, 1998a).

## Effects of Atmospheric Pollutants on Natural Systems

Ecosystem impacts can be organized by the pollutants of concern and by the level of biological organization at which impacts are directly measured. We attempt to address both dimensions of categorization in this overview. In Table E-1 we summarize the major pollutants of concern, and the documented acute and long-term ecological impacts associated with them. We follow with a description of each of the major pollutant classes and conclude with a summary of pollutant impacts at each level of biological organization.

**Table E-1**
**Classes of Pollutants And Ecological Effects**

| Pollutant Class | Major Pollutants and Precursors | Acute Effects | Long-term Effects |
|---|---|---|---|
| Acidic deposition | Sulfuric acid, nitric acid<br><br>Precursors: Sulfur dioxide, nitrogen oxides | Direct toxic effects to plant leaves and aquatic organisms. | Progressive deterioration of soil quality. Chronic acidification of surface waters. |
| Nitrogen Deposition | Nitrogen compounds (e.g., nitrogen oxides) | | Saturation of terrestrial ecosystems with nitrogen. Progressive nitrogen enrichment of coastal estuaries. |
| Hazardous Air Pollutants (HAPs) | Mercury, dioxins | Direct toxic effects to animals. | Conservation of mercury and dioxins in biogeochemical cycles and accumulation in the food chain. |
| Ozone | Tropospheric ozone<br><br>Precursors: Nitrogen oxides and Volatile Organic Compounds (VOCs) | Direct toxic effects to plant leaves. | Alterations of ecosystem wide patterns of energy flow and nutrient cycling. |

## Acidic Deposition

Acidification is perhaps the best-studied effect of atmospheric pollutant deposition to natural environments. Acidification of ecosystems has been shown to cause direct toxic effects to sensitive organisms as well as long-term changes in ecosystem functions. Acidification can affect all levels of biological organization in both terrestrial and aquatic ecosystems. Adverse effects seen in terrestrial ecosystems can include acute toxic interactions of acids with terrestrial plants or, more importantly, chronic acidification of terrestrial ecosystems leading to nutrient deficiencies in soils, aluminum mobilization, and concomitant decreases in health and biological productivity of forests (Smith, 1990; SOS/T 18, 1989). Similar to terrestrial ecosystems, adverse acidification-induced effects on surface waters may include elevated mortality rates of sensitive species, changes in the composition of communities, and changes in ecosystem-level interactions like nutrient cycling and energy flows. In the United States, acidification-related injuries to aquatic ecosystems may be more significant than injuries of terrestrial sites (EPA, 1995a; NAPAP 1991).

The predominant causes of acidic precipitation are sulfuric and nitric acid ($H_2SO_4$ and $HNO_3$). These strong mineral acids are formed from sulfur dioxide ($SO_2$) and nitrogen oxides ($NO_x$) in the atmosphere (i.e., they are secondary pollutants). Sulfur compounds are emitted from anthropogenic sources in the form of sulfur dioxide and, to a much lesser extent, primary sulfates, principally from coal and residual-oil combustion and a few industrial processes. Since the late 1960s electric utilities have been the major contributor to $SO_2$ emissions (NAPAP, 1991 p. 178). The combustion of fuels is the principal anthropogenic source of emissions of $NO_x$. Such combustion occurs in internal combustion engines, residential and commercial furnaces, industrial boilers, electric utility boilers, engines, and other miscellaneous sources. Because a large portion of anthropogenic $NO_x$ emissions come from transportation sources (i.e., non-point source pollution), $NO_x$ sources are on average more dispersed compared with anthropogenic sources of $SO_2$ (NAPAP, 1991, p. 189).

In the atmosphere, $SO_2$ and $NO_x$ are converted to sulfates and nitrates, transported over long distances, and deposited over large areas downwind of point sources or in the vicinity of urban areas. Deposition occurs via three main pathways: 1) precipitation or wet deposition, where material is dissolved in rain or snow; 2) dry deposition, involving direct deposition of gases and particles (aerosols) to any surface; and 3) cloud-water deposition, involving material dissolved in cloud droplets that is deposited when cloud or fog droplets are intercepted by vegetation (NAPAP, 1991, p. 181).

Initially, it was thought that $SO_2$ emissions were the only significant contributor to acidic deposition. Subsequently, emissions of $SO_2$ declined substantially between 1970 and 1988 due to a variety of factors, including emissions controls mandated by the Clean Air Act and changes in industrial processes such as the switch of electric utility plants to coal with lower sulfur content (NAPAP, 1991, p. 198). During this period, the role of nitrogen deposition as a contributor to aquatic acidification became apparent. While initial evidence suggested that most deposited nitrogen would be taken up by biota, more recent research has indicated that nitrogen may be leaching from terrestrial systems and causing aquatic acidification.

Comprehensive research on the ecological impacts of acidification is found in the publications of the National Acid Precipitation Assessment Program and EPA's *Acid Deposition Standard Feasibility Study Report to Congress* (1995a). In this analysis we rely heavily upon the extensive research conducted under these two programs.

### Nitrogen Deposition

Atmospheric nitrogen deposition to terrestrial and aquatic ecosystems can cause deleterious ecological effects ranging from eutrophication to acidification (as discussed above). Deposition of nitrogen can stimulate nitrogen-uptake by plants and microorganisms and increase biological productivity and growth. Chronic deposition of nitrogen may adversely affect biogeochemical cycles of watersheds

by progressively saturating terrestrial portions with nitrogen. Nitrogen saturation is a gradually occurring process, during which watersheds undergo progressive changes in their nitrogen cycle. This process can lead to increases in the amount of nitrogen leached into lower-elevation terrestrial ecosystems, wetlands and, most notably, surface waters (Stoddard, 1994; Aber et al., 1989).

Among the most pernicious effects of nitrogen leaching from terrestrial ecosystems can be acidification of fresh water bodies (as previously discussed) and eutrophication of estuaries (Richardson, 1996; Vollweider et al., 1990). Similar to terrestrial ecosystems, nitrogen enrichment of coastal estuaries can have a fertilizing effect, stimulating productivity of algae, marine plants (Vitusek and Howarth, 1991) and aquatic animals, including fish and shell fish. If eutrophication is excessive, however, it is likely to result in serious damages to estuarine ecosystems. Specifically, massive algae blooms can develop, leading to declining oxygen levels, habitat loss, and declines in fish and shellfish populations.

Nitrogen loading to estuaries is a major and growing problem. A 1996 inventory of estuarine water quality performed by coastal states and encompassing 72 percent of estuaries in the U.S. shows that nutrient enrichment pollutes 6,254 square miles (22 percent) of the surveyed waters, and contributes to 57 percent of all the reported water quality problems. At a recent meeting of National Estuary Program directors, eleven out of twenty-eight directors ranked nutrient overloading as a high priority issue for their programs, and seven additional directors ranked it as a mid-level priority (EPA, 1997b). Eighty-six percent of East Coast estuaries are considered susceptible to nitrogen enrichment (EPA, 1997c), and many coastal communities are finding that the nutrient loading problem is already so severe that they must add advanced wastewater treatment to existing plants, add infrastructure to promote water reuse, and impose stricter controls on all development and agricultural practices (EPA, 1997a).

Atmospherically derived nitrogen makes up a sizable fraction of total nitrogen inputs to estuaries in the Northeast (Hinga et al. 1991; Jaworski et al. 1997; Paerl 1997; Paerl et al. 1990; McMahon and Woodside, 1996; Rendell et al. 1993; Valiela et al. 1997). Atmospheric nitrogen is deposited to waters and watersheds in wet (rain, snow, and fog) and dry (aerosols and gases) forms. Approximately 10 to 50 percent of total nitrogen load to coastal waters is derived from direct and indirect atmospheric deposition. Estuaries on the eastern seaboard, and those downwind of urban areas tend to have a larger percentage of total nitrogen coming from atmospheric deposition.

### *Hazardous Air Pollutant Deposition*

Hazardous air pollutants (HAPs), are a general category of toxic substances covered under a single title of the Clean Air Act. Title III lists 189 HAPs, though only five are responsible for the majority of currently documented ecosystem impacts. These HAPs are mercury, polychlorinated biphenyls (PCBs), chlordane, dioxins, and dichlorodiphenyl-trichloroethane (DDT). The use of three of these compounds (PCBs, chlordane, and DDT) was effectively illegal in the United States prior to 1990 (EPA 1992), and there are currently no plans for additional CAAA regulations of these compounds (Federal Register Unified Agenda 1998). Emissions of the remaining two toxins, mercury and dioxins, continue to cause ecosystem impacts.

Mercury (Hg) is a toxic element found ubiquitously throughout the environment. Unlike many HAPs, much of the mercury released to the environment comes from natural sources. Anthropogenic sources can also release mercury to the environment. Estimates of the percentage of mercury emissions attributable to anthropogenic sources range from 10 to 80 percent (Mason et al. 1994, Hudson et al. 1995, Stein et al. 1996), although most estimates cluster between 40 and 75 percent (EPA 1997d).

About 80 percent of all anthropogenic mercury loadings to the environment are from air emissions. Global atmospheric concentrations of mercury have approximately tripled since pre-industrial times (Mason et al. 1994). Atmospheric deposition of

mercury has increased by a factor of about 3.7 (Swain et al. 1992), and the concentration of mercury in sediments in remote lakes has increased by a factor of 2.3 (Lucotte et al. 1995). These findings suggest that approximately 57 to 73 percent of atmospherically deposited mercury is anthropogenic in origin.

Atmospheric deposition of mercury and its subsequent movement in ecosystems may result in the concentration of mercury within organisms ("bioaccumulation") and its subsequent transfer throughout the food chain. As a consequence, mercury tends to accumulate along the hierarchical organization of food webs, with increasing concentrations found in animals at higher levels of the food chain ("biomagnification"). Fish, birds and mammals are among the group of organisms most threatened by mercury contamination of the environment. Symptoms may range from behavioral abnormalities to reduced reproductive success and death (EPA, 1997d). In 1996, mercury levels in fish were high enough that 11 states had mercury-based statewide fish consumption advisories. Twenty-eight more had at least one water body under advisory because of mercury concerns. These observations suggest that atmospheric mercury deposition may contribute significantly to mercury levels in freshwater ecosystems nationally.

Mercury is a neurotoxin that, at sufficient levels, can cause neurologic damage and death in both animals and humans. Adverse effects on wildlife include neurotoxicity, reproductive, and developmental effects (EPA 1997d). While fish are unlikely to experience toxic effects from mercury poisoning in the absence of point discharges, piscivorous predators and predators who eat piscivores accumulate more mercury and may suffer from mercury poisoning. However, the only species for which there is currently strong evidence of poisoning from atmospheric mercury are the common loon and possibly the Florida panther. It is unclear whether other piscivorous species, such as kingfishers, mink, and river otters, have suffered adverse health effects as a consequence of atmospheric mercury deposition (EPA 1997d).

Mercury is likely to persist at levels of concern in ecosystems for some time. The majority of atmospherically-released mercury is deposited to terrestrial environments, where it is largely sequestered. However, as mercury accumulates in soils, some amount (less than 30 percent of that which is deposited within a watershed) will be slowly released to freshwater bodies and oceans. Modeling efforts by Swain et al. (1992, reviewed in Mason et al. 1994) suggest that the retention of mercury by some lakes is essentially complete. Studies by Mason et al. (1994) predicted that elimination of anthropogenic mercury presently in the oceans and in the atmosphere would take 15 to 20 years after the complete termination of all anthropogenic emissions. Because of mercury's persistence in terrestrial and aquatic environments, it will probably take some time for reductions in mercury emissions to be notable in ecosystems (Swain et al. 1992, reviewed in Mason et al. 1994).

The other HAPs of concern are polychlorinated dibenzo-p-dioxins (PCDDs), a group of 75 organochlorine compounds that are sometimes referred to as dioxins. The most toxic member of this group is 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD). Because TCDD is the most toxic dioxin, the toxicity of a dioxin mixture is often expressed as the *toxic equivalency* (TEQ) of some amount of TCDD. Polychlorinated dibenzofurans (PCDFs) are close chemical relatives of PCDDs. Both classes of compounds are produced by the same processes, and both are ubiquitous in the environment (WHO 1989). TEQ estimates are often given jointly for dioxins and furans.

Dioxins and furans, unlike mercury, are not natural to the environment. They are formed during the combustion of wastes and fossil fuels, and as a consequence of fires or spills that involve particular chemicals like benzenes or PCBs. They are also formed as by-products in both the manufacturing of other chemicals and in pulp and paper mill bleaching processes (WHO 1989, EPA 1992a). EPA estimates that combustion sources emit over ten times as many TEQs as did all other categories combined.

Dioxins accumulate in the fat of animals and bioaccumulate through food chains. Fish are among the most sensitive vertebrates to the effects of TCDD, especially during early life stages. Fish are exposed to dioxins primarily through their food (EPA 1993), but some studies have reported that they can also absorb the trace amounts of dioxins present in water. EPA (1992) reported bioconcentration factors (BCFs)[2] for dioxins in fish of 5,000 to 9,000, and EPA (1993) estimated that bioaccumulation factors (BAFs) for lake trout can be on the order of 500,000 to 1,200,000.[3] Toxic effects on young fish include decreased feeding, weight loss, and fin necrosis; however, with a few exceptions, TCDD levels in the environment are generally too low to result in toxicity to juvenile or adult fish (EPA 1993, Walker and Peterson 1994).

The risk that dioxins pose to other wildlife is difficult to assess because both laboratory and field studies in this area are limited (EPA 1993, Giesey et al. 1994). One study (White et al. 1994) found that wood duck eggs from a contaminated area had levels of PCDDs and PCDFs 50 times higher than levels in control eggs. The contaminated nests were significantly less successful than control nests, and contaminated ducklings also suffered from teratogenic effects.

TCDD is an extremely stable chemical and is unlikely to be significantly degraded by chemical or biological hydrolysis under normal environmental conditions. Its half-life in soils may be on the order of a decade or more, and it may be even more persistent in aquatic sediments (Webster and Commoner 1990). For example, Johnson et al. (1996) found that, though TCDD levels in fish and sediments from an Arkansas river declined significantly during the 12 years following the initial pollution of the river, fish from some locations continued to have levels of TCDD that exceeded Food and Drug Administration (FDA) guidelines. TCDD is subject to photochemical degradation, but since the penetration of light into soils and many natural water bodies is limited, this degradation is not likely to be environmentally significant (WHO 1989, Zook and Rappe 1990). Because of dioxins' toxicity and persistence, their presence in freshwater ecosystems is likely to be an issue of concern for decades.

### Tropospheric Ozone

Ozone pollution is widespread in the eastern United States, in southern California, and in the vicinity of most major cities. Many of the observed effects of ozone on vegetation are related to direct toxic or harmful interactions with essential physiological functions of plants and subsequent reductions in biomass production (reduced growth). Damages to plants are commonly manifested as stress specific symptoms such as necrotic spots of plant leaves, acceleration of leaf aging, and reduced photosynthesis. Ozone damages at the community and ecosystem-level vary widely depending upon numerous factors, including concentration and temporal variation of tropospheric ozone, species composition, soil properties and climatic factors. In most instances, responses to chronic or recurrent exposure are subtle and not observable for many years. These injuries can cause stand-level forest decline in sensitive ecosystems (EPA, 1996; McBride et al., 1985; Miller et al., 1982).

Species that are particularly sensitive to ozone can be found among all groups of plants. Although many visible injuries have occurred in conifer species (e.g., Ponderosa and Jeffrey pine), a variety of deciduous trees and shrubs are also sensitive to ozone. Black cherry, many poplars (genus *Populus*) and many fruit trees including almond (*Prunus amygdalis* Batsch), peach (*Prunus persica*), and plum (*Prunus domestica*) trees are all

[2]Bioconcentration factors (BCFs) are calculated based on laboratory experiments. BCFs represent the ratio between the chemical's concentration in the organism to its concentration in the water, but unlike bioaccumulation factors (BAFs), they measure only how much of a chemical an organism accumulates as a consequence of its exposure to contaminated water. BCFs do not measure contaminant uptake as a function of exposure to contaminated food or sediments (EPA 1993).

[3]Because dioxins have such low solubility, accurately measuring their concentrations in water is extremely difficult. For this reason, any reported BCFs and BAFs should be examined carefully.

affected by elevated levels of ozone (EPA 1996a). In annual species, effects of ozone on production occur through changes in allocation of carbohydrates and can result in reduced seed production. Many annual plant species, including commercial crops, are among the most sensitive species. The National Crop Loss Assessment Network (NCLAN 1988), a multi-year program of the EPA, established that ambient ozone levels cause physical damages to crop plants and statistically significant reductions in agricultural yields.

Ecosystems with known damages that are attributed to ozone include the San Bernardino Mountains of Southern California, the Sierra Nevada Mountains, and sites in the vicinity of urban areas throughout the country. According to EPA (1996a), the San Bernardino Mountain range is by far the most severely ozone-impacted ecosystem. This mixed-conifer forest ecosystem has been exposed to chronically elevated ozone levels over a period of 50 or more years. This exposure has resulted in major changes of ecosystem characteristics, including species composition, nutrient cycling and energy flow. The first indications of ozone damages to the ecosystem were observed on the more sensitive members of the forest community: individual Ponderosa and Jeffrey pines. Direct injuries included visible foliar damage, premature needle senescence, reduced photosynthesis, altered carbon allocation, and reduction of growth rates and reproductive success. Changes in the energy available to trees (i.e., changes in carbohydrate production and allocation) influenced interactions with predators, pathogens and symbionts. Subsequently, the accumulation of weakened trees resulted in heavy bark beetle attack that significantly elevated mortality rates and extensive salvage logging during the 1960s and 1970s (Miller and McBride, 1998). Alterations in the composition and population density of the fungal microflora weakened soil microbial organisms and slowed the rate of decomposition, leading to the accumulation of a thick needle layer under stands with the most severe needle injuries and defoliation. Reduced production of seeds and fruits also affected the amount of food available to small vertebrates in the ecosystem, thereby affecting the local food chain (EPA 1996a). Similarly, ozone concentrations capable of causing injury to the Sierra Nevada Mountains have been occurring for many years, but injury to sensitive trees has never reached the same proportions as in the San Bernardino Forest. Significant differences in both the forest stand composition (e.g., the presence of fewer conifers and more hardwoods), and site dynamics have probably played an important role in determining the different ecosystem responses.

In each of these areas, ozone may act synergistically with other stress factors to induce further damages to vegetation. In the eastern United States, for example, regionally elevated levels of tropospheric ozone co-occur with high deposition rates of nitrogen, sulfur and acids. These multiple stress factors may have acted synergistically in injuring many high elevation forests throughout the eastern United States.

### *Multiple Stresses and Patterns of Exposure*

Although air pollutants can be grouped into classes according to their effects, as described above, it is recognized that one pollutant (or one class of pollutants) does not solely impact most ecosystems. Many environmental damages are the result of the combined action of multiple stress factors, including several types of air pollution and other anthropogenic or natural stress factors.

The recognition of interactions between several types of pollutants and between pollutants and other kinds of stress has introduced a new level of complexity in air pollution research. In many cases, various stress factors act synergistically to induce damages to ecosystems. These multiple stress factors can include: (1) various kinds of air pollutants (e.g., acidic deposition, ozone and nitrogen deposition); (2) other anthropogenic stress factors such as harvesting, overfishing or habitat disruption (e.g., the disruption of ecosystems by roads or urban areas); (3) environmental factors including availability of water, nutrients, light, or temperature (including heat and frost); and (4) biological factors such as animals feeding on plants, pathogens, and the status of micro-organisms in the soil (Taylor et al., 1994;

Winner, 1994; Smith, 1990). In the eastern US, for example, elevated deposition rates of nitrogen, sulfur and acids co-occur with regionally elevated levels of tropospheric ozone and climatic stress factors.

In addition, air pollutants have indirect effects that are at least as important as direct toxic effects on living organisms. Indirect effects include those in which the pollutants(s) alter the physical or chemical environment (e.g., soil properties) the plant's ability to compete for limited resources (e.g., water, light), or its ability to withstand pests or pathogens. Examples are excessive availability of nitrogen, soil depletion caused by acidic deposition, and changes in the ability to adapt to cold temperatures induced by acidic deposition (Taylor et al., 1994; NAPAP, 1991). Unfortunately, few mechanisms of interactions between various stress factors are known, and interpretations of scientific findings are usually associated with a high degree of uncertainty.

The situation is further complicated by the fact that the specific temporal and spatial patterns of pollutant exposures play a significant role in the response of organisms and ecosystems to air pollution. Temporal patterns include timing, duration and patterns of recurrent exposure to a specific pollutant or pollutants. For example, plant response to peak concentrations of ozone during daylight can be more severe (compared to exposure to the same level of ozone at night) because uptake of ozone is often higher during the day (EPA, 1996a). Spatial patterns include proximity of ecosystems to various pollution sources and the identification of specific source-receptor relationships between ecosystems and pollution sources (Taylor et al., 1994). For example, the deposition of most pollutants occurs after long-range atmospheric transport, with deposition rates depending upon climate, land-use and geology.

### Summary of Ecological Impacts from Air Pollutants Regulated by the CAAA

We summarize major examples of air pollution interactions with various levels of biological organization in Table E-2 through E-4. We organize these interactions according to classes of pollutants and injuries they cause, the various levels of biological systems, and types of affected ecosystems. It is important to note that interactions listed are intended to illustrate the range of possible adverse effects. These effects are examples for a wide variety of interactions but do not cover all aspects of air pollution-environment interactions.

**Table E-2**
**Interactions Between Acid Deposition and Natural Systems**
**At Various Levels of Organization**

| | | Examples of Interactions | |
|---|---|---|---|
| **Spatial Scale** | **Type of Interaction** | **Acidification of Forests** | **Acidification of Streams and Lakes** |
| Molecular and cellular | Chemical and biochemical processes | Damages to epidermal layers and cells of plants through deposition of acids. | Decreases in pH and increases in aluminum ions cause pathological changes in structure of gill tissue in fish. |
| Individual | Direct physiological response | Increased loss of nutrients via foliar leaching. | Hydrogen and aluminum ions in the water column impair regulation of body ions. |
| | Indirect effects: Death due to ionoregulatory failure. Acidification can indirectly affect response to altered environmental factors or alterations of the individual's ability to cope with other kinds of stress. | Cation depletion in the soil causes nutrient deficiencies in plants. Concentrations of aluminum ions in soils can reach phytotoxic levels. Increased sensitivity to other stress factors like pathogens and frost. | Aluminum ions in the water column can be toxic to many aquatic organisms through impairment of gill regulation. Acidification can indirectly affect submerged plant species, because it reduces the availability of dissolved carbon dioxide ($CO_2$). |
| Population | Change of population characteristics like productivity or mortality rates. | Decrease of biological productivity of sensitive organisms. Selection for less sensitive individuals. Microevolution of resistance. | Decrease of biological productivity of sensitive organisms. Selection for less sensitive individuals. Microevolution of resistance. |
| Community | Changes of community structure and competitive patterns | Alteration of competitive patterns. Selective advantage for acid-resistant species. Loss of acid sensitive species and individuals. Decrease in productivity. Decrease of species richness and diversity. | Alteration of competitive patterns. Selective advantage for acid-resistant species. Loss of acid sensitive species and individuals. Decrease in productivity. Decrease of species richness and diversity. |
| Local Ecosystem (e.g., landscape element) | Changes in nutrient cycle, hydrological cycle, and energy flow of lakes, wetlands, forests, grasslands, etc. | Progressive depletion of nutrient cations in the soil. Increase in the concentration of mobile aluminum ions in the soil. | Measurable declines of decomposition of some forms of organic matter, potentially resulting in decreased rates of nutrient cycling. |
| Regional Ecosystem (e.g., watershed) | Biogeochemical cycles within a watershed. Region-wide alterations of biodiversity. | Leaching of sulfate, nitrate, aluminum, and calcium to streams and lakes. Acidification of aquatic bodies. | Additional acidification of aquatic systems through processes in terrestrial sites within the watershed. |

**Table E-3**
**Interactions Between Nitrogen Deposition and Natural Systems At Various Levels of Organization**

| | | Examples of Interactions | |
|---|---|---|---|
| **Spatial Scale** | **Type of Interaction** | **Eutrophication and Nitrogen Saturation of Terrestrial Landscapes** | **Eutrophication of Coastal Estuaries** |
| Molecular and cellular | Chemical and biochemical processes | Assimilation of nitrogen by plants and microorganisms | Assimilation of nitrogen by plants and microorganisms. |
| Individual | Direct physiological response. | Increases in leaf- size of terrestrial plants. | Increase in growth of marine plants. |
| | Indirect effects: Response to altered environmental factors or alterations of the individual's ability to cope with other kinds of stress. | Decreased resistance to biotic and abiotic stress factors like pathogens and frost. Disruption of plant-symbiont relationships with mycorrhiza fungi. | Injuries to marine fauna through oxygen depletion of the environment. Loss of physical habitat due to loss of sea-grass beds. Injury through increased shading. Toxic blooms of plankton. |
| Population | Change of population characteristics like productivity or mortality rates. | Increase in biological productivity and growth rates of some species. | Increase in biological productivity. Increase of growth rates (esp. of algae and marine plants). |
| Community | Changes of community structure and competitive patterns | Alteration of competitive patterns. Selective advantage for fast growing species and individuals that efficiently use additional nitrogen. Loss of species adapted to nitrogen-poor environments. | Excessive algal growth. Changes in species composition. Decrease in sea-grass beds. |
| Local Ecosystem (e.g., landscape element) | Changes in nutrient cycle, hydrological cycle, and energy flow of lakes, wetlands, forests, grasslands, etc. | Magnification of the biogeochemical nitrogen cycle. Progressive saturation of microorganisms, soils, and plants with nitrogen. | Magnification of the nitrogen cycle. Depletion of oxygen, increased shading through algal growth. |
| Regional Ecosystem (e.g., watershed) | Biogeochemical cycles within a watershed. Region-wide alterations of biodiversity. | Leaching of nitrogen from terrestrial sites to streams and lakes. Acidification of aquatic bodies. Eutrophication of estuaries. | Additional input of nitrogen from nitrogen-saturated terrestrial sites within the watershed. |

Note: See *Overview of Ecological Impacts of Air Pollutants Regulated by the 1990 Clean Air Act Amendments* (IEc 1998) for sources.

**Table E-4**
**Interactions of Mercury and Ozone with Natural Systems**
**At Various Levels of Organization**

| Spatial Scale | Type of Interaction | Examples of Interactions: Mercury in streams and lakes | Examples of Interactions: Ozone |
|---|---|---|---|
| Molecular and cellular | Chemical and biochemical processes | Mercury enters the body of vertebrates and binds to sulfhydril groups (i.e. proteins). | Oxidation of enzymes of plants. Disruption of the membrane potential. |
| Individual | Direct physiological response. | Neurological effects in vertebrates. Behavioral abnormalities. Damages to the liver. | Direct injuries include visible foliar damage, premature needle senescence, reduced photosynthesis, altered carbon allocation, and reduction of growth rates and reproductive success. |
| | Indirect effects: Response to altered environmental factors or alterations of the individual's ability to cope with other kinds of stress. | Few interactions known. Damages through increased sensitivity to other environmental stress factors could occur, for example, through impairment of immune response. | Increased sensitivity to biotic and abiotic stress factors like pathogens and frost. Disruption of plant-symbiont relationship (mychorrhizae), and symbionts. |
| Population | Change of population characteristics like productivity or mortality rates. | Reduced reproductive success of fish and bird species. Increased mortality rates, especially in earlier life stages. | Reduced biological productivity. Selection for less sensitive individuals. Possibly microevolution for ozone resistance. |
| Community | Changes of community structure and competitive patterns | Loss of species diversity of benthic invertebrates. | Alteration of competitive patterns. Selective advantage for ozone-resistant species. Loss of ozone sensitive species and individuals. Reduction in productivity. |
| Local Ecosystem (e.g.,landscape element) | Changes in nutrient cycle, hydrological cycle, and energy flow of lakes, wetlands, forests, grasslands, etc. | Not well understood. | Alterations of ecosystem-wide patterns of energy flow and nutrient cycling. |
| Regional Ecosystem (e.g., watershed) | Biogeochemical cycles within a watershed. Region-wide alterations of biodiversity. | Not well understood. | Region-wide loss of sensitive species. |

Note: See *Overview of Ecological Impacts of Air Pollutants Regulated by the 1990 Clean Air Act Amendments* (IEc 1998) for sources.

Predicting ecological impacts of air pollution at the regional scale or for the United States as a whole would require an understanding of interactions at many temporal and spatial scales, where there is currently a general lack of data. Furthermore, there is limited transferability of existing information between various spatial and temporal scales and between geographic regions. However, we can reach several general conclusions, based on the existing literature.

- Although ambient concentrations of most air pollutants significantly decreased after the Clean Air Act of 1970, some pollutants still occur in concentrations high enough to directly injure living organisms. These direct injuries can be observed, for example, in areas with high ambient levels of tropospheric ozone or in some high-elevation ecosystems that are exposed to high levels of acid deposition (EPA, 1996a; NAPAP, 1991).

- Air pollutants have indirect effects that are at least as important as direct toxic effects on living organisms. Indirect effects include those in which the pollutant alters the physical or chemical environment (e.g., soil properties), the plant's ability to compete for limited resources (e.g., water, light), or the plant's ability to withstand pests or pathogens. Examples are excessive availability of nitrogen, depletion of nutrient cations in the soil by acid deposition, mobilization of toxic elements such as aluminum, and changes in winter hardiness (Taylor et al., 1994). As is true for other complex interactions, indirect effects are more difficult to observe than direct toxic relationships between air pollutants and biota, and there may be a variety of interactions that have not yet been detected.

- There is a group of pollutants that tend to be conserved in the landscape after they have been deposited to ecosystems. These conserved pollutants are transformed through biotic and abiotic processes within ecosystems, and accumulate in biogeochemical cycles. These pollutants include, but are not limited to, hydrogen ions (H+), sulfur (S) and nitrogen (N) containing substances, and mercury (Hg). Chronic deposition of these pollutants, can result in progressive increases in concentrations and cause injuries due to cumulative effects. Indirect, cumulative damages caused by chronic exposure (i.e., long-term, moderate concentrations) to these pollutants may increase in magnitude over time frames of decades or centuries with very subtle annual increments of change. Examples are N-saturation of terrestrial ecosystems, cation depletion of terrestrial ecosystems, acidification of streams and lakes, and accumulation of mercury in aquatic food webs (Pitelka 1994; Taylor et al. 1994; Likens et al. 1996; EPA 1997e).

- Damages to ecosystems are most likely caused by a combination of environmental stress factors with every interactive stress or else have a mechanistic model that incorporates interactions among pollutants. Unfortunately neither approach is yet possible. These include anthropogenic factors such as air pollution and other environmental stress factors such as low temperature, excess or limited water, and limited availability of nutrients. The specific combinations of factors differ among regions and ecosystems where declines have been observed (Taylor et al., 1994; Winner, 1994; Smith, 1990). To accurately predict the impacts of multiple acting stress factors we would have to build a catalogue of research results that defines the response of every plant species to every air pollutant, with every interactive stress or else have a mechanistic model that incorporates interactions among pollutants. Unfortunately neither approach is yet possible.

- Pollutant-environment interactions are complicated by the fact that biotic and abiotic factors in ecosystems change dramatically over time. Besides oscillations on a daily basis, and changes in a seasonal rhythm, long range successional changes occur over time periods of years, decades, or even centuries. These temporal variations occur in polluted and pristine ecosystems, and no single point in time or space can be defined as representative of the entire system.

Long-term impacts of air pollution are often manifested in interactions at the regional scale. The history of lead pollution may provide a useful illustration of impacts of air pollution long after deposition rates have declined significantly due to environmental regulations. Historically, scientists were concerned about lead deposition because of its high affinity to soil organic matter and its accumulation in the litter layers of soils. Starting around 1960, lead accumulated in forest soils in the northeastern United States as a result of human activities. Following a significant decline of combustion of leaded gasoline between 1970 and 1988, deposition rates dropped, and decreases in lead levels in soils and rivers have been observed throughout the United States. Apparently forest floors have responded rapidly to the decline of lead input, and instead of accumulating lead, forest soils are now slowly releasing lead to the underlying mineral horizon. It has been estimated that sometime in the middle of the next century, forests will begin to release anthropogenic lead deposited after 1960 to rivers and streams (Miller and Friedland, 1994), where it may cause unforeseen damages to aquatic ecosystems.

There is evidence that current air pollution is an important environmental stress factor over large areas of the United States and other countries, even if effects have not yet been fully documented. Actions taken now to reduce air emissions may have consequences far into the future and may affect ecosystems in ways that are not yet known. Because it is not yet possible to predict what long-term, continuous exposure to multiple pollutants might do to ecosystem structure and function, it may be concluded that the ecological benefits of air pollution control lie in the prevention of long-term damages to resources and the potential for increased recovery rates, as well as the more traditional prevention of acute injuries. Because it is not yet possible to predict what long-term, continuous exposure to multiple pollutants might do to ecosystem structure and function, it may be prudent to focus on the prevention of possible long-term damages to resources and preserve the potential for increased recovery rates, as well as preventing more traditional acute injures to ecosystems.

## Methodological Overview

In this section we describe the methods for characterizing the economic benefits of reducing several classes of ecological impacts through the regulations of the CAAA. As indicated in the previous section, it is not possible to characterize and quantify all impacts associated with air pollution. Instead, we select those impacts amenable to quantitative analysis, using two criteria:

*Criterion #1: The endpoint must be an identifiable service flow*

*Criterion #2: A defensible link must exist between changes in air pollution emissions and the quality or quantity of the ecological service flow, and quantitative models must be available to monetize these changes*

The use of these criteria greatly constrains the range of impacts that can be treated in this analysis. While the previous section identifies many pollutant-ecosystem interactions, only a handful are understood and have been modeled to an extent sufficient to reliably quantify their impact. We attempt to present both reliable quantitative information regarding the benefits of the CAAA while demonstrating the potential magnitude of the ecological benefits of the CAAA if all impacts were valued. A more detailed description of the choice of endpoints is found in

*Methods for Selecting Monetizable Benefits Derived from Ecological Resources as a Result of Air Quality Improvements Attributable to the 1990 Clean Air Act Amendments, 1990-2010 (IEc 1998b) and Characterizing Economic Benefits of Reducing Impacts to Ecosystem Integrity* (IEc 1998c).

### Using Service Flow Endpoints for Valuation

The theoretical basis of economic benefits assessment is that ecosystems provide services to humankind, and that those services have economic value. The application of this theory requires the isolation of service flows that have market values or are otherwise amenable to available methods for determining value in the absence of formal markets. Freeman (1997) provides one possible grouping of ecological service flows:

- Sources of material inputs to the economy, including fossil fuels, wood products, minerals, water, and animals;
- Life support services, including breathable air and a livable climate;
- Amenities that provide opportunities for active recreation and passive enjoyment of nature, including nonuse values; and
- Processing of waste products that are generated by economic activity and discharged into the environment.

Available methods do not exist to comprehensively value each of these service flows for all ecosystems. Generally, we are limited to those service flows that either are sources of material inputs or natural amenities that involve active recreation. Impacts to these service flows that can be valued tend to manifest themselves immediately and can be readily measured and assessed in terms of the proven cause and effect relationships. The result is that we can value only a small subset of the ecosystem benefits from environmental regulations in an analysis of national scope.

Based on the constraints of economic valuation methods and data, we select from the host of ecosystem impacts identified in the previous section a set of service flows as candidate endpoints for analysis. These endpoints are listed in Table E-5.

**Table E-5**
**Ecological Impacts with Identifiable Human Service Flows**

| Pollutant Class | Ecosystem Effect | Service Flow Impacted |
|---|---|---|
| Acidification ($H_2SO_4$, $HNO_3$) | High-elevation forest acidification resulting in dieback | Forest aesthetics |
| | Freshwater acidification resulting in aquatic organism (e.g. fish) population decline | Recreational fishing |
| | Changes in biological diversity and species mix in terrestrial and aquatic systems | Existence value for maintenance of biological diversity |
| Nitrogen Saturation and Eutrophication ($NO_x$) | Freshwater acidification resulting in aquatic organism (e.g. fish) population decline | Recreational fishing |
| | Estuarine eutrophication causing oxygen depletion and changes in nutrient cycling | Recreational and commercial fishing |
| | Changes in biological diversity and species mix in terrestrial and aquatic systems | Existence value for maintenance of biological diversity |
| Toxics Deposition (Mercury, Dioxin) | Terrestrial bioaccumulation of mercury and dioxin | Hunting, wildlife aesthetics |
| | Aquatic bioaccumulation of mercury and dioxin | Recreational and commercial fishing |
| | Changes in biological diversity and species mix in terrestrial and aquatic systems | Existence value for maintenance of biological diversity |
| Tropospheric Ozone ($O_3$) | Terrestrial plant foliar damage causing lower productivity and reduced competitiveness | Commercial timber productivity, forest aesthetics, existence value |
| Multiple Pollutant Stress | Ecosystem deterioration resulting in visual effects, habitat loss, and changes in biological diversity and species mix caused by synergistic action of several pollutants | Ecosystem aesthetics, ecosystem existence value |

### *Defensible Links and Quantitative Modeling Requirements*

The second criterion for endpoint selection is satisfied when complete data and model coverage is available to describe the impacts of air pollutants. We briefly describe the types of data and models necessary to accomplish quantitative benefits assessment, then identify those endpoints that we can pursue in this analysis.

In order to determine changes in ecological service flows, defensible links between pollution emissions and service flow changes must be quantitatively modeled. Described generally, five steps are necessary to complete a quantitative analysis; emissions characterization; environmental fate and transport assessment; exposure characterization; ecosystem effects characterization; and economic behavior models.

Emissions characterization requires models that project the level of air pollutants entering the atmosphere over the period of time in question for both factual and counterfactual scenarios under consideration in the analysis. In our analyses the factual scenario is the level of emissions in the United States generated between 1990 and 2010, as regulated by the CAAA (Post-CAAA). The counterfactual scenario is the level of emissions during the same

period without the regulations promulgated under the CAAA (Pre-CAAA)[4].

The geographic transport and deposition of air pollutants are estimated using models that consider multiple chemical and meteorologic factors. The section 812 prospective analysis of the CAAA uses three models, detailed in Appendix C and *Air Quality Modeling to Support the Section 812 Prospective Analysis* (prepared for EPA by Systems Applications International, Inc., 1999)[5].

In cases where the presence of a pollutant in a geographic region, as estimated by dispersion models, is not an adequate measure of the exposure of biota to the pollutant, an exposure model is required. These models must take biotic and abiotic ecosystems processes into account.

Once the exposure of the biota in question is estimated, the physiological effect of that exposure must be estimated. Dose-response functions that describe the effects of varying levels of pollutants to specific organisms are derived from laboratory, field, and modeling experiments. The intensive nature of this research and the necessity of studying each species individually causes this link to be weak in most quantitative ecological assessments.

[4]For all analyses in this report, emissions under each scenario are based upon EPA's National Emissions Inventory (NEI) with modeling provided by the Emissions Reduction and Cost Analysis Model (ERCAM). See Appendix A for details.

[5]The three regional-scale air quality modeling systems applied include the regulatory Modeling System for Aerosols and Deposition (REMSAD), the Regional Acid Deposition Model (RADM), and the Urban Airshed Model (UAM-IV and UAM-V). In addition, this prospective ecological benefits assessment uses results from the Regional Lagrangian Model of Air Pollution (RELMAP) to estimate mercury and dioxin deposition.

**Table E-6**
**Model Coverage for Candidate Endpoints for Quantitative Assessment**

| Pollutant | Endpoint | Emissions Model | Transport and Deposition | Exposure Model | Dose-response Functions | Economic Model |
|---|---|---|---|---|---|---|
| Acidification ($H_2SO_4$, $HNO_3$) | Forest aesthetics | NEI, ERCAM | RADM | Not Required | Multiple available | Only site-specific models available |
| | Recreational fishing | NEI, ERCAM | RADM | MAGIC (region specific) | Multiple available | Only site-specific models available |
| | Biological diversity existence value | NEI, ERCAM | RADM | MAGIC (region specific) | Multiple available | Only site-specific models available |
| Nitrogen Saturation and Eutrophication ($NO_x$) | Recreational and commercial fisheries | NEI, ERCAM | RADM | Estuary-specific models available | None Available | Only site-specific models available |
| | Biological diversity existence value | NEI, ERCAM | RADM | Estuary-specific models available | Multiple available | None Available |
| Toxics Deposition (Hg, Dioxin) | Forest aesthetics | None Available | RELMAP, ISC3 | None Available | Multiple available | Only site-specific models available |
| | Hunting, wildlife aesthetics | None Available | RELMAP, ISC3 | None Available | Multiple available | Only site-specific models available |
| | Recreational and commercial fishing | None Available | RELMAP, ISC3 | IEM-2M (site specific) | Multiple available, or consumption advisory limits can be used | Only site-specific models available |
| | Biological diversity existence value | None Available | RELMAP | None Available | Multiple available | None Available |
| Multiple Pollutant Stress | Ecosystem aesthetics, ecosystem existence value. | NEI, ERCAM | | None Available | None Available | None Available |

NEI: National Emissions Inventory; ERCAM: Emissions Reduction and Cost Analysis Model; RADM: Regional Acid Deposition Model;REMSAD: Regulatory Modeling System for Aerosols and Deposition; RELMAP: Regional Lagrangian Model of Air Pollution; UAM: Urban Airshed Model; TAMM: Timber Assessment Market Model, developed and maintained by the U.S. Forest Service.

When pollutant doses are sufficiently high to cause physiologic responses in biota, ecological service flows may be affected. In order to monetize these impacts, a model of the economic behavior associated with the service flow must be developed. Economic models are specific to the service flow and the consuming population, and not all service flows have adequate economic models that describe their value. For example, recreational fishing models account for the preferences and geographic distribution of anglers as well as the site characteristics of the fisheries. These data are site specific, making the model specification fairly non-transferable.

Table E-6 describes the extent to which models are available to estimate the full chain of defensible links for the ecological endpoints identified in Table E-5. Each column must have an identified model in order to complete the required modeling steps to quantify changes in that endpoint. In cases where defensible links are not quantified, opportunities exist for qualitative analysis.

Table E-7 summarizes the quantitative and qualitative analyses that we propose based on the available model coverage. Geographic scope plays an important role in determining the level of analysis, such as a national assessment, a case study or a qualitative description that is possible given existing models. This exhibit demonstrates that, of the great number of known impacts of air pollution, only a subset can be assessed. In the next section we discuss the methods, results, and caveats of the analyses of these selected endpoints.

**Table E-7**
**Summary of Endpoints Selected for Quantitative Analysis**

| Endpoint | Analysis | Geographic Scope |
|---|---|---|
| Lake acidification impacts on recreational fisheries | Quantification of improved fisheries with monetization of recreational value | Case study of New York State |
| Estuarine eutrophication impacts on recreational and commercial fisheries | Quantification of improved fisheries with monetization of avoided costs of alternative eutrophication control methods | Illustrative calculations for case studies of Chesapeake Bay, Long Island Sound, and Tampa Bay (with extensions to East Coast estuaries) |
| Ozone impacts on commercial timber sales | Quantification of improved timber growth with monetization of commercial timber revenues | National assessment |
| Ozone impacts on carbon sequestration in commercial timber | Quantification of improved carbon sequestration | National assessment |
| Toxicity impacts on recreational fishing | Qualitative analysis of improved recreational fisheries | Qualitative regional case studies of New York and Tennessee |

### Extending Future Analyses

By focusing on the readily measured impacts identified in Table E-7, it is possible to lose sight of ecosystem-level changes that may threaten ecosystem integrity in ways that alter or increase the risk of changing ecosystem structure and function. The isolation of service flows may often imply an oversimplified cause and effect relationship between pollution and the provision of the service flow, when more often the service flow is affected by complex non-linear relationships that govern ecosystem structure and function. Economic analyses that focus on a narrow class of acute service-flow impacts will not cover larger ecosystem-wide impacts that may ultimately prove most relevant to environmental policy decision making. This analytical weakness becomes apparent when impacts to ecological functions such as nutrient cycling and biological diversity are assessed.

Issues on which to focus future analytic work in this field include:

- Major linkages of cause and effect between air pollution and subtle deterioration in ecosystem integrity are difficult to quantify;
- Degradation of ecosystem integrity most often does not cause immediate measurable declines in ecosystem service flows that are monetarily valued by society;
- The time-frame required for many ecological impacts to manifest themselves is such that the present value of these impacts discounts to negligible sums; and,
- Uncertainties associated with the scale of complex ecological impacts are too great to allow for reliable estimation of the economic implications.

Because of the weaknesses in the available methods and data, the benefits assessment in this appendix does not represent a comprehensive estimate of the economic benefits of the CAAA. Moreover, the potential magnitude of long-term economic impacts of ecological damages mitigated by the CAAA suggests great care must be taken to consider those ecosystem impacts that are not quantified here. Significant future analytical work must be performed to build a sufficient base of knowledge and data to allow the expansion of this benefits assessment.

## Eutrophication of Estuaries

This analysis considers the economic benefits of reduced nitrogen deposition and the effects on selected eastern estuaries attributable to the 1990 Clean Air Act Amendments (CAAA). Note that these estimates were not included in the primary benefits of the CAAA; these are presented here as an alternative calculation only. We present a description of how nitrogen deposition affects estuarine ecosystems, an explanation of the effects on ecological service flows, and an assessment of the benefits of reducing nitrogen deposition in the context of several case studies using avoided-damage and displaced-cost approaches as alternative estimates of benefits. A more comprehensive description of this analysis is found in *Benefits Assessment of Decreased Nitrogen Deposition to Estuaries in the United States Attributable to the 1990 Clean Air Act Amendments, 1990-2010* (IEc, 1999a).

### Impacts of Nitrogen Deposition on Estuaries

Atmospherically derived nitrogen makes up a sizable fraction of total nitrogen inputs in estuaries in the eastern United States. When atmospheric nitrogen enters estuaries it can cause *eutrophication*, or an increased nutrient load that, in excess, changes the ecosystem's structure and function and affects the provision of ecological service flows. The ecological effects and their associated service flows are listed in Table E-8.

**Table E-8**
**Service Flows Affected by Changes in Estuarine Ecosystems**

| Ecosystem Changes | Service Flow Impacts |
|---|---|
| Deterioration of breeding grounds for fisheries | Commercial fishing yields, species mix<br>Recreational fishing catch rate, species mix |
| Loss of habitat for aquatic and avian biota | Existence value of a healthy estuarine ecosystem<br>Wildlife viewing<br>Aesthetics |

Derivation of dose-response relationships between atmospheric nitrogen loading and ecological effects is complicated by the dynamic nature of ecological systems. In addition to being characterized by non-linear, "threshold" type responses, esturine ecosystems are simultaneously influenced by a variety of stressors (both anthropogenic and non-anthropogenic). This makes it difficult to quantify the nature and magnitude of ecological changes expected to result from a change in a single stressor such as nutrient loading. Further, if the state of the ecosystem has changed (as from oligotrophic[6] to eutrophic) the removal of the initial stressor does not necessarily mean a rapid return to the prior state. This complicates the quantitative benefits assessment of controlling nitrogen deposition through the CAAA.

## Economic Benefits of Decreasing Atmospheric Deposition of Nitrogen

EPA's analysis begins with a geographic information system (GIS) approach to estimate the total volume of nitrogen inputs that the CAAA would reduce to three major estuaries, the Chesapeake Bay, Long Island Sound, and Tampa Bay. Unfortunately, resource limitations prevented us from examining more than three estuaries at this point. The three estuaries were chosen for several reasons. First, each of these areas maintains an active research center, either under Clean-Water Act or National Estuary Program provisions, and has identified airborne nitrogen as a major factor in its efforts to limit eutrophication. Second, each has conducted some research into the level of damages associated with nitrogen eutrophication, although in the final analysis only the Chesapeake Bay Program's research was sufficient to establish some measure of avoided damages. Third, each of these estuaries has in place a binding commitment to meet its nitrogen reduction targets, a necessary condition for applying the avoided costs approach. In these estuaries, failure to reduce airborne nitrogen deposition (as would be the case if no CAAA were in place) would imply that additional nitrogen reductions would be necessary from other sources, such as point or nonpoint surface water discharges. Implementation of the CAAA therefore effectively avoids the imposition of costs to achieve those nitrogen discharge reductions.

For each of the three estuaries selected, we then conduct two types of analyses. First, we assess the avoided nitrogen deposition loadings to the watershed and avoided costs from reducing nitrogen deposition, using submerged aquatic vegetation as a key biophysical indicator. Second, we estimate the avoided cost of implementing planned alternatives to the CAAA for reducing nitrogen deposition.

## GIS-Based Deposition and Loadings Estimates

The first step toward calculating deposition-related nitrogen loadings to the three estuaries is to estimate the total deposition of nitrogen to the watersheds. Table E-9 presents our estimates of the quantity of nitrogen deposited to the Chesapeake Bay, Long Island Sound, and Tampa Bay

[6]Oligotrophy refers to a state of relatively low nutrient enrichment and low productivity of aquatic ecosystems. In contrast, eutrophy refers to a state of relatively high nutrient loading and higher productivity, sometimes leading to overenrichment and reduction in ecological service flows via water quality decline.

**Table E-9**
**Total Nitrogen Deposition Based on GIS Analysis**
**(millions of lbs.)**

| Watershed | Scenario 1: 1990 | Scenario 2: 2010 without CAAA | Scenario 3: 2010 with CAAA | Difference |
|---|---|---|---|---|
| **Chesapeake Bay** | 345.1 | 452.4 | 258.1 | **194.3** |
| **Long Island Sound** | 78.3 | 93.7 | 56.8 | **36.9** |
| **Tampa Bay** | 8.1 | 11.3 | 7.0 | **4.3** |

watersheds. We present data for three different scenarios. The first scenario is our estimate of the quantity of nitrogen deposited in 1990, prior to the introduction of the CAAA. Scenario 2 is our estimate of the quantity of nitrogen deposited in 2010 without the CAAA, and Scenario 3 is the quantity deposited with the CAAA. The difference between Scenarios 2 and 3 represents the potential future impacts of the CAAA on nitrogen deposition.[7]

As the exhibit indicates, the CAAA are likely to have a significant impact on the quantity of nitrogen deposited to each of the three watersheds. For the Chesapeake Bay watershed, nitrogen deposition is expected to be nearly 195 million pounds less in 2010 (43 percent) than it would have been without the CAAA. For the Long Island Sound and Tampa Bay watersheds, this figure is approximately 37 million pounds (39 percent) and four million pounds (38 percent), respectively.

We also estimate the prevalence of major categories of land use in each of the three watersheds. Land use is a critical component of our analysis because the quantity of nitrogen runoff that eventually reaches the estuary varies according to the type of land that receives the atmospheric deposition. For example, the fate of atmosphericly deposited nitrogen will differ if the nitrogen falls on forest versus urban land, because forest land generally retains a greater percentage of nitrogen than urban land. Our analysis uses distinct nitrogen "pass-through" figures for each category of land use. Pass-through represents the percentage of atmospherically deposited nitrogen that is ultimately transported to surface water rather than retained by the land.

Table E-10 presents the land use prevalence and the pass-through factors that we use for each of the three watersheds in our analysis.[8] As the exhibit indicates, forests (53 percent) and agricultural lands (32 percent) represent the majority of the land use in the Chesapeake Bay watershed. In the Long Island Sound watershed, forests (67 percent) again dominate land use; however, urban lands account for as much territory as agricultural lands (11 percent). For the Tampa Bay watershed, agricultural lands constitute the largest land use (33 percent), while rangelands (19 percent), urban land (15 percent), and wetlands (10 percent) represent a much greater proportion of the land use than in the other two watersheds.

[7] These data are derived from IEc's spatial analysis of the watersheds and RADM nitrogen deposition modeling. The RADM modeling is described in Appendix C of this report.

[8] Pass-through estimates were derived from EPA's analysis of the relevant literature - see IEc (1999).

**Table E-10**
**Land Use Prevalence and Pass-Through Figures**

| Watershed | Forest | Agricultural | Urban | Water | Wetlands | Other* |
|---|---|---|---|---|---|---|
| **Chesapeake Bay** | | | | | | |
| **Land Use** | 53% | 32% | 6% | 7% | 1% | 1% |
| **N Pass-Through** | 20% | 30% | 50% | 100% | 20% | 30% |
| **Long Island Sound** | | | | | | |
| **Land Use** | 67% | 11% | 11% | 9% | 2% | 0% |
| **N Pass-Through** | 20% | 30% | 50% | 100% | 20% | 30% |
| **Tampa Bay** | | | | | | |
| **Land Use** | 5% | 33% | 15% | 15% | 10% | 22% |
| **N Pass-Through** | 20% | 30% | 50% | 100% | 20% | 30% |

* "Other" areas in Tampa Bay include rangeland (19%) and barren land (3%).

We use the pass-through figures and land use prevalence in conjunction with deposition quantities to estimate nitrogen loadings to each estuary. Table E-11 displays our nitrogen loadings estimates for the three watersheds under the three scenarios. As the exhibit indicates, loadings from atmospheric deposition decrease significantly due to the CAAA. For Chesapeake Bay, for example, we estimate that nitrogen loadings with the CAAA will be approximately 79 million pounds in 2010, approximately 58 million pounds less than our estimate for loadings in 2010 without implementation of the CAAA. For the Long Island Sound and Tampa Bay, the difference between the two scenarios is approximately 13 million pounds and 1.8 million pounds, respectively.

**Table E-11**
**Nitrogen Loadings from Atmospheric Deposition**
**(millions of lbs.)**

| Watershed | Scenario 1: 1990 | Scenario 2: 2010 without CAAA | Scenario 3: 2010 W/CAAA | Difference |
|---|---|---|---|---|
| **Chesapeake Bay** | 105.2 | 137.5 | 79.4 | 58.1 |
| **Long Island Sound** | 26.7 | 31.9 | 19.1 | 12.8 |
| **Tampa Bay** | 3.4 | 4.7 | 2.9 | 1.8 |

## *Displaced Costs from Reducing Atmospheric Deposition to Estuaries*

It is possible to use a displaced cost approach to determine the benefits associated with reduced nitrogen emissions. To reduce excess nutrient loads (including nitrogen) to local estuaries, many coastal communities are pursuing costly abatement options. These options include point source controls as well as urban non-point and agricultural non-point source controls. We estimate the marginal costs of abatement associated with these controls as implemented in the three case study estuaries. To the extent that nitrogen deposition can be controlled more cost effectively than point-source discharges, the control expenditures displaced by the CAAA represent a benefit to society.

Ideally, a nitrogen management program would result in the least expensive abatement possible,

thereby minimizing the resources society expends on nitrogen control. The lowest marginal cost pollution reduction is exploited first, and pursued to its limit before the next least costly alternative is exploited, and so on until the required nitrogen reduction is met (represented by the dark columns in Figure E-1).

With the CAAA, a portion of the resources society committed to or would have committed to reducing a quantity of waterborne nitrogen may be unnecessary. Following the cost minimizing strategy, society will forego the most expensive control cost option first, pursue it to its limit before the next most expensive alternative is foregone, and so on until the nitrogen reduction benefits from the CAAA are exhausted (represented by the lightly shaded columns in Figure E-2). The level of nitrogen reduction remaining will therefore be accomplished at the lowest cost (represented by the dark columns in Figure E-2).

**Figure E-1. Nitrogen Reduction Without CAAA**

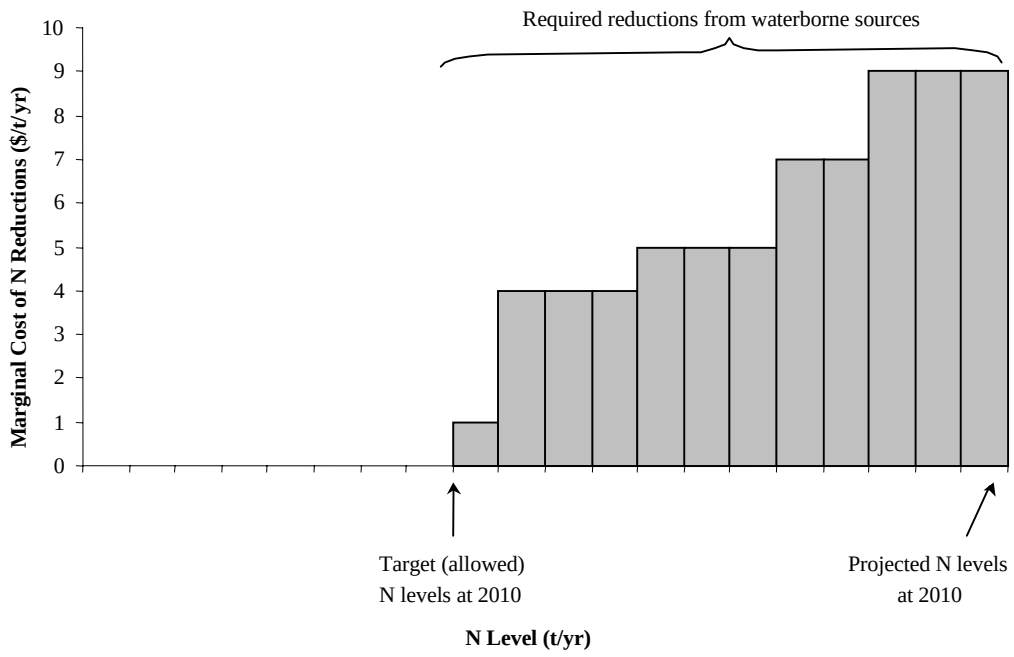
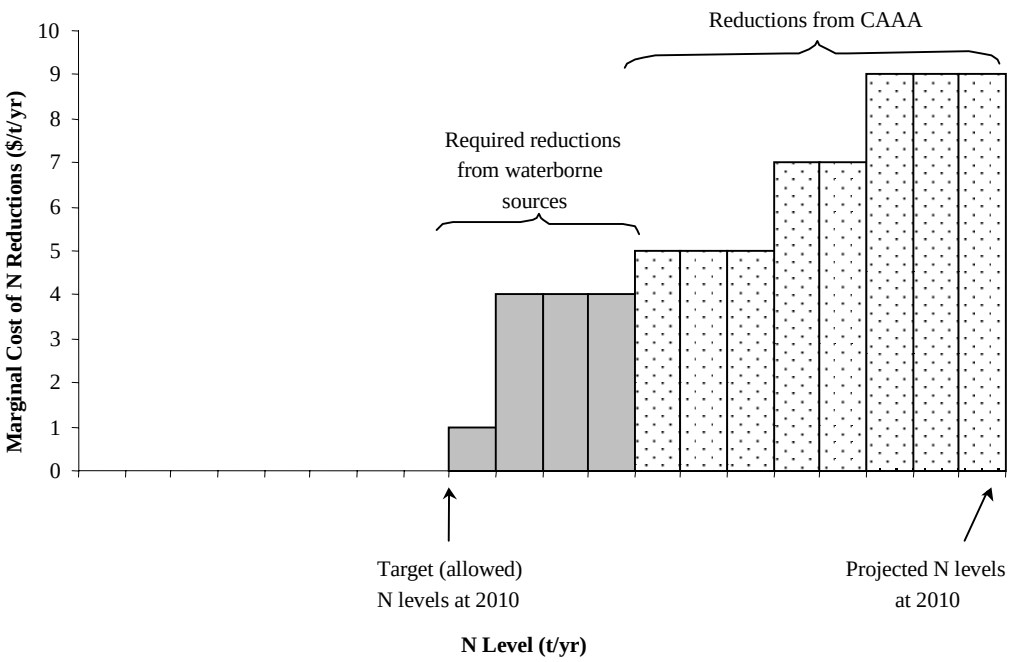



**Figure E-2. Nitrogen Reduction With CAAA**



We develop our avoided-cost estimate by assuming that decision makers will choose to forego the most costly nitrogen abatement projects first. That is, we assume that reduced deposition and the resulting loadings reduction will eliminate the need for additional point or non-point source controls at the high end of the marginal cost curve.

To estimate the economic benefits of reduced nitrogen deposition from the CAAA, we require site specific information from the watershed level. A justifiable avoided cost analysis relies upon the existence of realistic and enforceable nitrogen reduction goals for each estuary. Without specific targets or reduction goals, it is not possible to suggest that there are any control costs to be avoided. As described earlier, we have chosen case study estuaries that fit this criterion. These areas have established nitrogen reduction programs that rely primarily on reductions of effluent from point sources as well as reductions in non-point source discharges. Information on the reduction goal and potential abatement options for meeting those goals allow us to estimate the portion of the goal that can be met by the CAAA, as well as the associated cost savings.[9] We summarize those results in Table E-12.

Next, we need to know the annual quantity of atmospheric nitrogen deposited on the watershed. Last, we need to understand details about the different nitrogen reduction programs that could be implemented in the watershed. This includes the quantity of nitrogen reduced through a particular control option (e.g., agriculture best management plans[BMPs]), and the unit cost of reducing that nitrogen (i.e. dollars per pound or ton of nitrogen reduced).

The benefits valuation derived using the avoid-costs approach should be interpreted cautiously for two reasons. First, it is an estimation of capital costs that serve more purposes than mitigating nitrogen inputs into the estuaries of concern. Water treatment works are intended to provide waste water treatment for a variety of pollutants and may be required even in the absence of air deposition of nitrogen. Second, the nitrogen loading targets for the estuaries are not concrete, strictly enforced limits, based on certain knowledge of the capacity of the estuaries to accept nitrogen inputs. Instead, the targets may change over time as knowledge of the effects of nitrogen to these estuaries change. For these reasons, we do not include these estimates in the primary benefits estimates for the CAAA.

[9]With increasing populations, controls of alternative sources (e.g., automobile and utility emissions) may be needed simply to meet the original target or goal; if the CAAA amendments are necessary just to achieve the target reductions, then we are actually measuring alternative costs and not displaced costs.

**Table E-12**
**Estimated Avoided Costs For Three Estuaries**

| Estuary | Reduced N Deposition in 2010(millions of pounds) | Lower-BoundMarginal Cost($/lb/yr.) | Upper-Bound Marginal Cost ($/lb/yr.) | Estimated Annual Avoided Costs in 2010 ($millions) |
|---|---|---|---|---|
| Long Island Sound | 12.8 | $2 | $8 | $25.6-$102.4 |
| Chesapeake Bay | 58.1 | $6 | $22 | $349-$1,278 |
| Tampa Bay | 1.8 | $6 | $38 | $11 - $68 |

## Results for Case Study Estuaries

- Under the *Chesapeake Bay* Agreement, the signatories (EPA, Maryland, Virginia, Pennsylvania, and the District of Columbia) have agreed to reduce nutrient loadings to the Bay by 40 percent by the year 2000, relative to 1985 levels. This goal translates to a nitrogen reduction of about 186 million pounds per year. A great deal of progress toward this goal has already been made, although success differs across sub-regions in the watershed (Chesapeake Bay Program 1997). Nitrogen loadings reductions achieved thus far are the result of both point and non-point source controls. Since 1985, 33 of the 315 major municipal treatment plants in the region have upgraded to biological nutrient removal (BNR), an advanced treatment technology specifically focused on nitrogen removal. These upgrades have reduced annual loadings by about 13 million pounds (as of 1996). Approximately 60 additional facilities are expected to implement BNR in the future. In addition, agricultural and urban best management practices have reduced non-point source loadings by about 16 million pounds per year, with additional implementation of BMPs planned for coming years (Chesapeake Bay Program, 1997). Because both point and non-point source controls play a role in anticipated future nitrogen reductions in Chesapeake Bay, we develop marginal and avoided cost estimates that incorporate both types of control. As reflected in Exhibit 15, we estimate avoided cost benefits for Chesapeake Bay ranging from about $349 million to $1.3 billion.

- *Long Island Sound* has established a goal of reducing nitrogen loadings by approximately 48 million pounds by 2015. Point source controls are anticipated to be the primary source of these reductions. Numerous sewage treatment plant upgrades are slated for the region, many of which are currently under construction. We use data from the Connecticut Department of Environmental Protection and New York Department of Environmental Conservation. The marginal cost figures yield an estimate of avoided costs that ranges from about $26 to $102 million per year.

- In 1996, the *Tampa Bay* Estuary Program (TBEP) adopted a five-year nutrient management goal that caps annual nitrogen loadings at 1992-1994 levels. Nitrogen loading to Tampa Bay is expected to increase seven percent by the year 2010 as a result of population growth and related commercial and residential development. To offset this growth and maintain current nitrogen levels, the TBEP has asked local governments, agencies, and industries to reduce total nitrogen loadings to the Bay by approximately 84 tons (168,000 pounds) per year by the year 2000. The result of this planning effort is the Nitrogen Management Action Plan (TBEP, 1998). This plan lists the projects undertaken or planned by industry, local governments, and agencies

that will contribute to meeting the nitrogen management goal for 2000 and beyond. These projects, which together surpass the nitrogen reduction goal for Tampa Bay, are a combination of point and non-point source control measures. The non-point source control projects include urban stormwater retention ponds, wetlands restoration, and land acquisition. The point source projects focus on advanced treatment technologies such as BNR at (publically owned treatment works (POTWs). For example, one project, proposed for implementation after the year 2000, will involve additional treatment of effluent from a POTW prior to reuse in the regional water supply. We estimate annual avoided costs for Tampa Bay ranging from about $11 million to $68 million.

These three estuaries represent only a portion of the total estuarine area affected by nitrogen deposition in the United States. The Chesapeake Bay and Long Island Sound account for roughly 20 to 25 percent of the East Coast estuarine watershed area addressed by the National Estuary Program, and Tampa Bay is a small fraction of the total Gulf Coast estuarine watershed area. As a result, our estimates reflect only a partial analysis of the national impact of nitrogen deposition.

## Results for Total East Coast Estuarine Area

To extrapolate the results of this analysis to all East Coast estuaries, we assume all estuaries along the Atlantic Coast have binding nitrogen budgets. We then use the same geographic information system (GIS) approach we used in our analysis of Chesapeake Bay, Long Island Sound, and Tampa Bay to estimate total nitrogen deposition and the associated loadings to estuaries located along the Atlantic Coast. This approach allows us to estimate nitrogen loadings in the year 2010 with and without CAAA emissions controls. Since watershed specific nitrogen control program information is not available for each watershed, we extrapolate BMP cost and nitrogen reduction data from the three case studies across all East Coast watersheds.[10]

Although we simplistically assume that each estuary has a nitrogen budget, the total East Coast displaced cost analysis does not include all estuaries along the Atlantic Coast, since some estuaries are not sensitive to nitrogen loadings. Certain estuaries are able to process large amounts of nutrients (nitrogen and phosphorus) without problems, while others are unable to process even low amounts of nutrients. Rather than rely on nitrogen levels to determine which estuaries to include in the displaced cost analysis, we use a measure of eutrophic susceptibility developed by Bricker et al. (1999-DRAFT), to determine how sensitive estuaries are to nitrogen loadings. Using their eutrophic susceptibility classification (low, medium, and high) we then exclude estuaries with low eutrophic susceptibility from the displaced cost analysis.[11]

The total displaced costs for the East Coast is simply the sum of the displaced costs for each estuary along the Atlantic Coast classified as moderately or highly susceptible to eutrophication (see Table E-13).[12] The lower-bound estimate of $261.5 million represents a point source control strategy. The upper-bound estimate of $2,766 million represents a strategy

[10] The control cost and nitrogen reduction data is available in the IEc memorandum to EPA dated August 26, 1999.

[11] This index is based on a variety of factors influencing sensitivity of estuaries to eutrophication, including water surface area of the estuary, estuary volume, freshwater inflow, tidal cycles, and vertical stratification.

[12] In the North Atlantic Region, Blue Hill Bay, Casco Bay, Englishman Bay, Kennebec/Androscoggin River, Merrimack River, Musconguc Bay, Narraguagus Bay, Penobscot Bay, and Saco Bay have a low susceptibility to eutrophication and are excluded from the displaced cost analysis. All estuaries in the Mid Atlantic Region are moderately to highly sensitive to eutrophication. In the South Atlantic Region, New River, Ossabaw Sound, Savannah River, and St. Helena Sound have a low susceptibility to eutrophication and are not included in the displaced cost analysis.

**Table E-13**
**Avoided Cost for Atlantic Coast**

| Watershed | Reduction through CAAA, Millions of Pounds (2010) | Avoided Cost, 100 Percent from Point Source ($1000/yr.) | Avoided Cost, from Nonpoint Source First, then Point Source on Difference ($1000/yr.) |
|---|---|---|---|
| North Atlantic | | | |
| Cape Cod Bay | 0.51 | 1,282 | 7,915 |
| Great Bay | 0.57 | 1,446 | 19,130 |
| Massachusetts Bay | 1.19 | 3,015 | 66,887 |
| Sheepscot Bay | 0.13 | 340 | 2,397 |
| Sub-Total | 13.02 | 6,083 | 96,329 |
| | | | |
| Mid Atlantic | | | |
| Barnegat Bay | 0.48 | 1,202 | 19,688 |
| Buzzards Bay | 0.78 | 1,976 | 14,429 |
| Chincoteaque Bay | 0.33 | 824 | 3,690 |
| Delaware Bay | 12.11 | 30,640 | 384,002 |
| Delaware Inland Bays[1] | 0.18 | 459 | 4,592 |
| Gardiners Bay | 0.64 | 1,624 | 13,929 |
| Great South Bay | 1.30 | 3,294 | 78,682 |
| Hudson River/Raritan Bay | 11.15 | 28,197 | 460,659 |
| Narragansett Bay | 1.65 | 4,176 | 65,841 |
| New Jersey Inland Bays | 1.12 | 2,825 | 28,314 |
| Sub-Total[2] | 29.74 | 75,219 | 1,073,826 |
| | | | |
| South Atlantic | | | |
| Albemarle Sound | 16.29 | 41,223 | 272,772 |
| Altamaha River | 7.24 | 18,315 | 182,591 |
| Biscayne Bay[3] | 0.78 | 1,973 | 12,136 |
| Bogue Sound | 0.30 | 756 | 9,426 |
| Broad River[3] | 0.46 | 1,160 | 7,137 |
| Cape Fear River | 6.59 | 16,674 | 171,805 |
| Charleston Harbor | 10.58 | 26,764 | 306,698 |
| Indian River | 0.76 | 1,926 | 36,045 |
| North/South Santee Rivers | 0.32 | 821 | 3,671 |
| Pamlico Sounds | 9.58 | 24,225 | 191,289 |
| St. Andrew/St. Simons Sounds[3] | 1.18 | 2,974 | 18,297 |
| St. Catherines/Sapelo Sounds | 0.28 | 721 | 6,525 |
| St. Johns River | 4.72 | 11,940 | 152,909 |
| St. Marys River/Cumberland Sound | 0.53 | 1,338 | 8,886 |
| Winyah Bay[1] | 11.63 | 29,415 | 215,612 |
| Sub-Total | 81.07 | 180,225 | 1,595,799 |
| | | | |
| **Total East Coast** | **123.82** | **261,527** | **2,765,954** |

[1] CAAA N reductions met with agriculture, forestry, and urban BMPs (point source reductions not required).
[2] Excluding Long Island Sound and Chesapeake Bay.
[3] CAAA N reductions met with agriculture BMPs (further reductions not required).

of nonpoint source BMPs, with further nitrogen reductions from point sources, if required.[13]

This estimate is based on an extrapolation of nitrogen abatement costs from representative watersheds. Although this is a broad assumption, it does provide a gross estimation of the range and magnitude of the CAAA benefits for the Atlantic Coast as a whole and its component watersheds.
Due to our general assumptions, a high degree of uncertainty is associated with this range. First, we do not know enough about the nature of the nitrogen budgets for each estuary and if those budgets would be binding. If nitrogen budgets are not binding, these regions may have little incentive to reduce nitrogen loadings. Furthermore, because of the lack of watershed specific cost information, we rely on available abatement cost data from the Chesapeake Bay and Long Island Sound to represent point source and nonpoint source unit abatement costs for all watersheds along the Atlantic Coast. The tightness of the range and accuracy of the displaced cost analysis is dependant on an accurate understanding and representation of the nitrogen abatement costs associated with the different point source control options and nonpoint source BMPs in each individual watershed. Lastly, we use marginal cost figures that represent averages of the costs associated with certain control measures. For example, our agriculture BMP cost figure is a simple average of six different agriculture BMPs, each with a different level of nitrogen reduction and a different cost per pound of nitrogen reduced. While beyond the scope of this effort, a more refined analysis would use marginal cost estimates based on the precise mix of agriculture BMPs to be implemented in each watershed and the cost of each, as determined by local factors within the watershed.

[13] In our analysis of the total displaced costs for the Atlantic Coast, several watersheds meet their CAAA nitrogen reduction levels without relying on point source controls.

## Avoided Damages to Estuarine Ecosystems

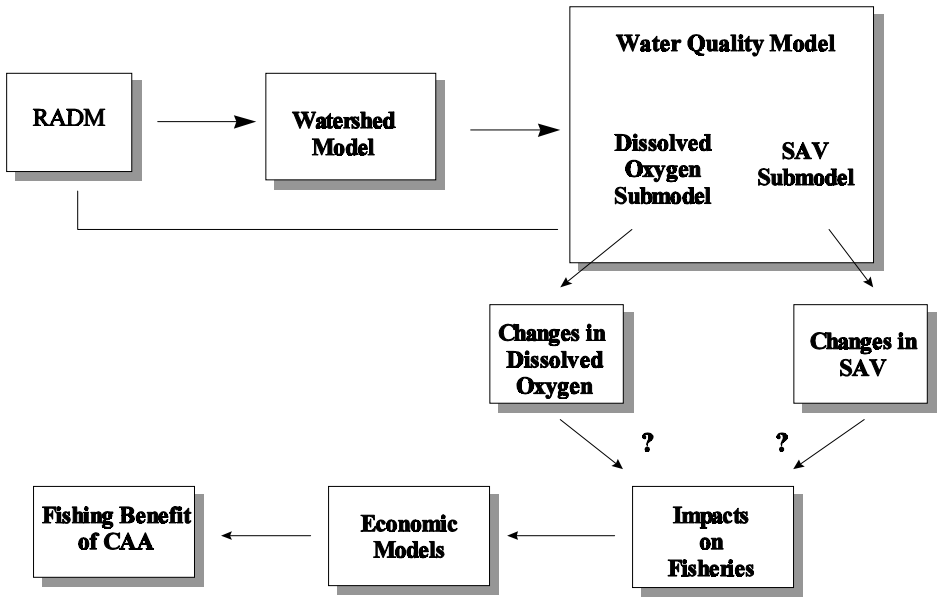
Theoretically, a modeling system that describes water quality changes, including fish population dynamics as a function of nitrogen input, would provide an assessment of the avoided damages from mitigating nitrogen deposition (Figure E-3). Because of current modeling and data constraints, however, the only means to quantify the damages of eutrophication from nitrogen deposition is through the use of specific biophysical indicators of estuarine health. Changes in an indicator, such as aerial extent of seagrass beds, can measure habitat damage. Based on changes in habitat, the change in ecosystem service flows associated with that habitat can be estimated.

From an economic perspective, this approach is useful in cases where habitat is closely related to the provision of ecological service flows, such as commercial and recreational fishing yields. Using seagrass beds as an indicator, we describe the potential for avoiding estuarine damages through the CAAA.

Submerged aquatic vegetation (SAV) is a group of angiosperms (plants that bear seeds, as opposed to algae, which reproduce via cell division) that form extensive meadows, providing habitat, breeding grounds, and nursery for a variety of organisms including fish, birds, shellfish, and invertebrates (Jacobs et al. 1981; Bell et al. 1989; Howard et al. 1989; Burkholder et al. 1992; Orth et al. 1994). SAV meadows give considerable three-dimensional structure to the seabed that provides small organisms with a place to hide from predators, acts as a sediment trap, and functions as a breakwater offering natural shoreline protection (Vermaat et al. 1998). Along with aerial extent, the density of SAV may be important in defining the health of the SAV community.

Though a universal nitrogen-SAV relationship has not been derived, field data show that increased nitrogen loading has been accompanied by extensive decline in SAV in a variety of estuaries (Valiela et al. 1997; Burkholder et al., 1992; Coastlines, 1994; Vermaat, 1998). In the Chesapeake, SAV acreage declined from more than 76,000 to about 40,000 acres

**Figure E-3**
**Estuary Models and Ecological Impacts of Concern**




between 1870 and 1950, concurrent with increased nutrient loading (Coastlines, 1994). From 1950 to 1980, when nutrient loading took a sharp upward turn, the decline continued to a low point of about 21,000 acres. This decline is quite likely due to shading by excessive algal growth and increased growth of epiphytic plants that also respond positively to increased nitrogen availability (Coastlines, 1994).

Empirical evidence from Tampa Bay and the Chesapeake Bay show that SAV populations will likely benefit from reduced nitrogen inputs to these estuaries. In Tampa Bay, there is a notable correlation between SAV and nitrogen inputs, as described in Figure E-4.[14]

In Chesapeake Bay, it is not possible to identify a statistically significant relationship between these two variables in isolation because of the large and heterogenous nature of the Chesapeake Bay. Embayments vary greatly in physical characteristics including depth and salinity, complicating the relationship. Nonetheless, the general trend has been an increase in seagrass acreage and a decrease in nitrogen loading. Figure E-15 shows the change in SAV acreage over the past two decades.

Because the relationships we describe from existing data are not sufficiently robust to allow projections of SAV coverage as a function of nitrogen deposition in our scenarios, we utilize alternative methods to provide quantitative benefits estimates in the following section.

[14] Two factor analysis of variance (ANOVA) analysis confirms this correlation; see EPA 1999 for more details.

**Figure E-4**

**Nitrogen Load vs. Seagrass Acreage in Tampa Bay**

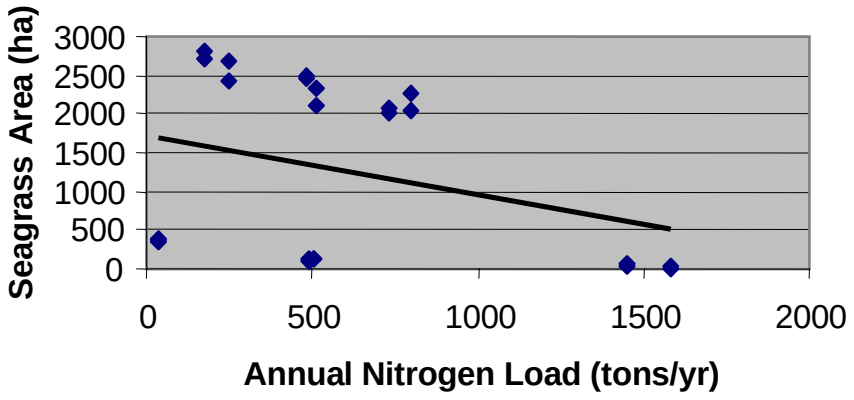
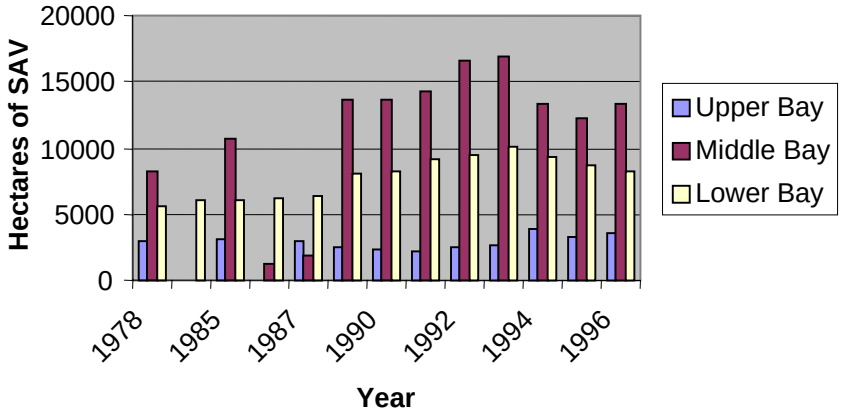



**Figure E-5**

**Chesapeake Bay SAV**
**1978-1996**



### Caveats and Uncertainties

Though it is difficult to directly predict the nature and magnitude of the ecosystem impacts of eutrophication, we conclude that continued nitrogen inputs at current levels will result in deleterious effects. The major caveats and uncertainties associated with these analyses follow.

- Our nitrogen loading estimates are derived using a highly simplified approach that takes into account total deposition and the nitrogen retention characteristics of different land uses. Some factors suggest that we may overstate loadings because we do not consider the effects of nitrogen travel through varied distances and heterogeneous geography, such as rivers and streams. For example, an additional 20 to 75 percent of nitrogen is retained during transport in rivers and streams (Hinga, et al., 1991).

  Other factors suggest that we may understate loadings. Most significantly, the Geographic Information Retrieval Analysis System (GIRAS) land use data in our GIS analysis were compiled in the early 1980s. It is likely that the current amount of urbanized land is greater than these data indicate. Furthermore, continued development of forest and other open land suggests that land uses will change significantly in the period between now and 2010. Because nitrogen removal in urban land is low, more refined land use data for future years would likely lead to greater estimates of nitrogen loadings. We compare our nitrogen loading estimates with those of estuary programs and published literature for 1990 to verify whether our approach generates results that correspond with existing estimates. For the Chesapeake our estimate of 150 million tons falls in the middle of existing estimates ranging from 58 to 159 million tons (EPA 1997e; Patwardhan and Donigian 1997; Fisher et al. 1998; Tyler 1998) for Long Island Sound our estimate of 27 million tons corresponds with the existing estimate of 26 million tons (Stacey 1998), and for Tampa Bay our estimate of 3 millions tons is close to the existing estimate of 2 million tons (TBEP 1998; Zarbuck et al. 1994).

- We base our estimates of avoided costs on simplified assumptions regarding the control measures that would be eliminated as a result of reducing atmospheric nitrogen. The mix of nitrogen controls that could be displaced will be influenced by state regulations affecting treatment plants and non-point sources as well as by pollution reduction goals for different sub-basins in each watershed. For example, water quality objectives for pollutants other than nitrogen may require controls that we assume could be eliminated.
- Similarly, we use marginal cost figures that represent averages of the costs associated with control measures. For example, we apply generic non-point source control cost estimates based on a mix of agriculture, forestry, and urban best management practices. While beyond the scope of this effort, a more refined analysis would use marginal cost estimates based on the precise mix of best management practices to be implemented in each watershed and the cost of each, as determined by local factors such as levels of nitrogen in soils, evolving agricultural practices, and changing development patterns.

## Acidification of Freshwater Fisheries

During the 1970s and 1980s, "acid rain" came to be known to the public as a phenomenon that injures trees, forests, and water bodies throughout Europe and in some areas of the United States and Canada. One of the goals of the CAAA was to address the problem of acidification of terrestrial and aquatic

ecosystems caused by acidic deposition. In this section we evaluate economic benefits accruing to society as a consequence of reductions in emissions of sulfur and nitrogen oxides mandated by the CAAA. In particular, we focus on a quantitative analysis of benefits derived from a reduction in acidification of aquatic bodies as they relate to recreational fishing in the Adirondacks region of New York State. Our analysis indicates that by mitigating acidification with the regulations promulgated under the CAAA, cumulative benefits between 1990 and 2010 can be accrued in the range of $67 to $465 million. Using the results from the acidification estimates, it is also plain to see that the CAAA may be preventing further ecological impacts from acidification that are not quantifiable in economic terms using available methods. A more comprehensive description of this analysis is found in *Economic Benefits Assessment of Decreased Acidification of Fresh Water Lakes and Streams in the United States Attributable to the 1990 Clean Air Act Amendments*, 1990-2010 (IEc, 1999b).

## Acidification of Surface Waters and Ecological Impacts

Acidification of surface waters is frequently described using two measures. One measure of acidification is pH, which is based on the hydrogen ion (H+) concentration found in surface waters.[15] The pH of a water sample can range from 1 to 14 on a logarithmic scale, with pure water having a pH of seven. The term acidic usually refers to a pH below seven, indicating high concentrations of hydrogen ions (H+). Rain water that is unaffected by anthropogenic factors (natural rain) is weakly acidic (pH 5.0 - 6.0), due to the presence of natural weak acids. With addition of acids from human activities, however, the pH of rain can range from 3.5 to 5.0 (NAPAP, 1991, p.15). Most freshwater lakes and streams have a pH between 6.5 and 8, indicating that surface waters can be naturally acidic. Only a small percentage of aquatic ecosystems are naturally acidic with pHs below 6.5, and concerns about anthropogenic acidification focus on the effects that may occur with decreases in pH below pH 6.5 (EPA, 1995a p. 9).

The second commonly used measure of acidification is Acid Neutralizing Capacity (ANC), which describes a water body's ability to neutralize acids added to the water column. Surface waters with higher ANC are generally more resistant to acidification and empirically tend to have higher pH levels. ANC is measured in micro-equivalents per liter (μeq/L). Surface waters with an ANC of less than 200 μeq/L are considered to have a low capacity for neutralizing acids. Water bodies with an ANC of 50 μeq/L or less have a very low capacity for neutralizing acids, and water bodies with an ANC of 0 μeq/L or less have no ability to neutralize acids and are acidic. These water bodies have no ability to neutralize acids and tend to be the most sensitive for long-term pH depressions below 6.0, which can produce the most severe effects on aquatic life (EPA, 1995a p. 9, NAPAP 1991 p.15).

Acidic deposition can lead to two kinds of acidification processes, depending on the duration of acidifying events. First, chronic acidification describes a situation in which acidic deposition leads to long-term changes in soil and water characteristics, causing chronically toxic environmental effects. Second, episodic acidification is a phenomenon in which surface waters experience short-term (hours to weeks) decreases in pH, usually during extreme hydrological events such as storm discharge or snowmelt (EPA, 1995a, p.9, NAPAP, 1991 p.18). For acid-sensitive fish species in some lakes or streams, for example, episodic events can cause complete spawning or recruitment failures (EPA, 1995a p.10).

The most comprehensive survey of surface waters comes from the National Surface Waters Survey (NSWS), which was conducted as part of the National Acid Precipitation Program (NAPAP). Based on the results from the NSWS surveys of lakes throughout the United States, an estimated 4.2 percent (1,180) of the NSWS lakes were acidic, defined as having ANC less than 0 μeq/l with pH levels in the range of 5.0 to 5.5. Nearly all of these lakes were in eastern portions of the United States, located in six "high-interest

[15] The pH of a water sample is equal to the negative log of its hydrogen ion concentration: pH= -log[H+].

regions". The six areas identified are New England, the Adirondacks, the mid-Atlantic Coastal Plain, the mid-Atlantic Highlands, Florida, and the upper Midwest. The NSWS found that acidic deposition is the dominant source of acid anions in about 75 percent of the acidic lakes and 50 percent of the acidic streams in their sample. Figure E-6 depicts the geographic ranges of known acidification.

The effects of acidity on aquatic organisms are determined by a number of different water quality variables, the most important of which are pH, inorganic aluminum, and calcium. The combined effects of these variables can adversely affect the physiology of individual organisms as well as population-level parameters. The direct effects may be classified as involving either recruitment failure or reduced adult survival. The outcomes on these impacts are declining acid-sensitive fish populations and a consequent decline in species richness (SOS/T 13 p.13-126).

## Modeling Acidification

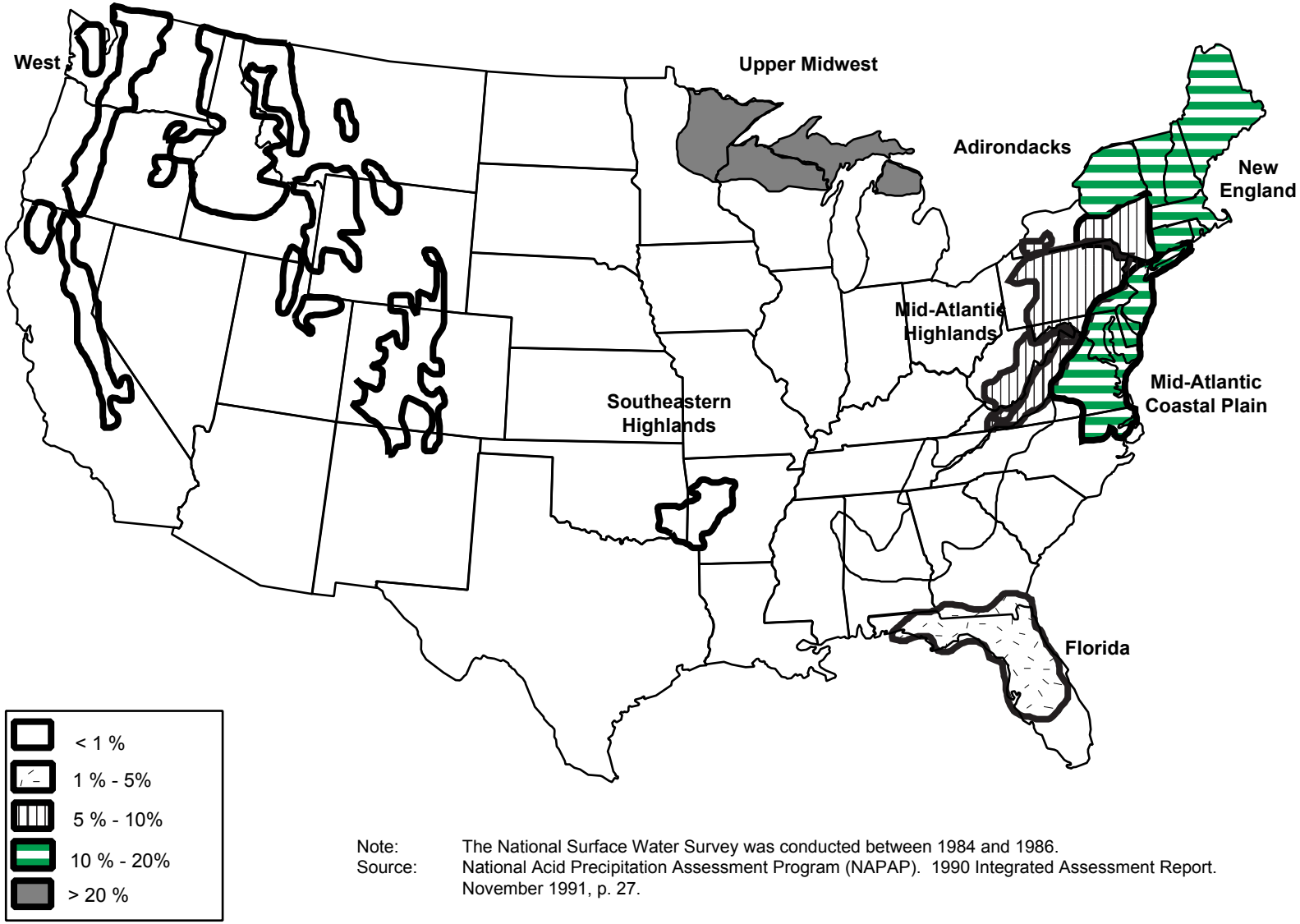
Figure E-7 shows the stages of modeling the ecological and economic impacts of acidification. This analysis uses RADM deposition data for the year 2010, and an extended deposition scenario that we develop for the year 2040, to demonstrate the possible lagged effects of the CAAA. We use these data in an acidification model that generates an estimate of the acidity of lakes in the Adirondacks. Lake acidity is input to an economic model that estimates the costs to anglers of diminishing lake water quality, and consequently declining fish populations.

We use the same emissions and deposition data for the period 1990 to 2010 as in each of our other endpoint analyses. The primary difference is that we extend the deposition scenarios to 2040 in order to accommodate for the lagged physical effects of acid deposition. This lag is a function of multiple watershed and water body characteristics influencing recovery from prolonged acidic deposition. Results of various efforts to model freshwater acidification showed that recovery of acidified water bodies can take over 50 years, even with substantial (up to 70 percent) reductions in sulfate deposition (Jenkins et al. 1990; Wright et al. 1994), while watershed soils may require 150-200 years for full recovery (Cosby et al. 1985).

Because appropriate data are lacking to simulate the emission and deposition of acidic pollutants between the years 2010 and 2040, we use two deposition scenarios for the period 2010-2040:

- Constant deposition from 2010 to 2040 at levels projected for 2010 under the regulations of the CAAA; and,
- Constant deposition from 2010 to 2040 at levels projected for 2010 without the regulations of the CAAA.

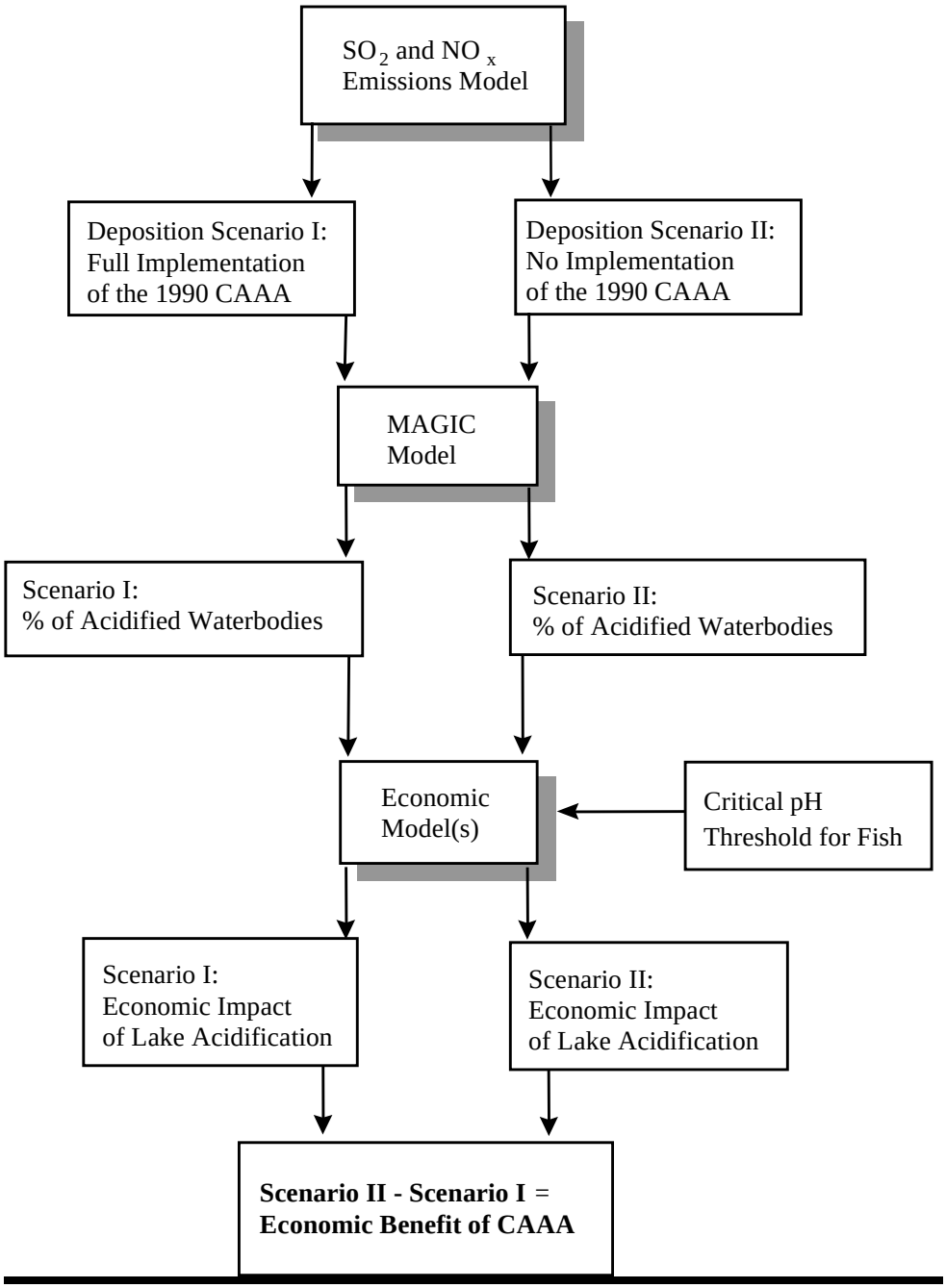
**Figure E-6**
**Percentage of Acidic Surface Waters in the National Surface Water Survey Regions**




Note: The National Surface Water Survey was conducted between 1984 and 1986.
Source: National Acid Precipitation Assessment Program (NAPAP). 1990 Integrated Assessment Report. November 1991, p. 27.

**Figure E-7**

## Acidification of Freshwater Ecosystems



Using these deposition scenarios, we estimate the extent of acidification in the Adirondacks. To do this, we use the Model of Acidification of Groundwater in Catchments (MAGIC). MAGIC is a lumped parameter model that was originally developed to project the long-term effects (i.e., decades to centuries) to surface water caused by acidic deposition (Cosby et al, 1985a, 1985b). NAPAP and the Acid Rain Program of EPA use MAGIC extensively in analysis of acidification in the Eastern United States (Church et al, 1989; Church et al, 1992; EPA, 1995), the results of which have been rigorously peer-reviewed and used in previous policy analyses. The data that MAGIC produces describe the extent of acidification (i.e., pH and ANC levels) that will occur in a sample of sensitive lakes in the Adirondacks as a function of acidic deposition levels.

As mentioned earlier, capacities of watersheds to retain deposited sulfur or nitrogen containing compounds are among the most important factors influencing surface water acidification. Because increasing stages of nitrogen saturation are likely to lead to decreasing nitrogen-retention capacities, it is necessary to consider these effects in our modeling approach. MAGIC, however, currently does not explicitly represent detailed cycling or processes affecting the rate of nitrogen uptake and release because processes that control the transition of a watershed to a state of nitrogen saturation leading to surface water acidification are not well understood (Van Sickle and Church, 1995). Instead, we use a sensitivity analysis of two boundary conditions, representing a nitrogen-saturated watershed and a watershed where terrestrial ecosystems continue to utilize the majority of deposited nitrogen.

Our means to assess the economic impact of acidification is to measure the change in social welfare that results from reducing the quality of available lakes for recreational fishing. In order to accomplish this, we must determine the water chemistry parameters at which declines in recreational fish populations are perceptible by anglers. As mentioned earlier in this memorandum, the toxicity of low levels of pH to fish species depends on a variety of factors, including the concentration of inorganic aluminum and calcium in the water column as well as specific sensitivities of locally occurring fish species. The level of pH is not a precise indicator of the habitability of a lake for fish. But because the full complement of relevant water chemistry variables for lakes in the region is not available, we are forced to work with pH as a proxy for habitability. In order to accommodate for the variable effects of pH due to other water chemistry variables, we derive a range of pH values where negative effects on fish species are empirically known to occur. We summarize these results in Table E-14.

**Table E-14**
**Summary of pH-Based Effects Threshold**

| | pH Effects Threshold (Low End) | pH Effects Threshold (High End) |
|---|---|---|
| Range for All Fish Species | 4.2 | 5.8 |
| Range of Mean Values for All Species | 4.8 | 5.3 |
| Range of recreationally important species (weighted average) | 4.6 | 5.4 |

Our review of the empirical effects literature demonstrates the difficulty in discerning a single pH threshold that could ever adequately characterize the ability of a water body to sustain recreational fishing. The most rigorous use of the available data might employ species-specific thresholds and apply these thresholds to individual lakes in the economic modeling domain. Unfortunately, information on the prevalent recreationally important species present in the sample of lakes modeled by MAGIC, as well as in the larger domain of lakes to which these results would be extrapolated, is not currently available. We therefore adopt a range of estimates of a pH threshold for acidification of a lake of 5.0 to 5.4. The range is consistent with a reasonable approximation of effects noticeable to anglers. Knowledge of effects with certainty would imply a more conservative assumption of a lower pH, perhaps consistent with the low end weighted average for recreationally important species, reported in the last row of Table E-14. We therefore report acidification results for an extreme low end threshold estimate of pH 4.6, but do not interpret those results as providing useful central estimates for a study specifically concerned with recreational fishing.

The final step in our analysis is to use an economic model that monetizes the impacts to recreational fishing under each of the acidification scenarios. This involves the selection of an economic model that appropriately covers the effects of acidification of multiple sites over the geographic area that is impacted, and the proper integration of the water quality information. The ideal for this application is a random utility model (RUM) that allows for the substitution among sites and fisheries as water quality parameters change, an essential feature when estimating recreational benefits.

Very few models of this type exist, and fewer cover a region of high acidification that is impacted by the CAAA. Efforts by Englin *et al* (1991), Mullen and Menz (1985), and Morey and Shaw (1990) advance this line of inquiry by relating regional acid deposition to recreational fishing damages, but Montgomery and Needelman (1997) are the first to use direct water quality measures in conjunction with a random utility model[16]. The estimation proceeds in three steps. First, a site-choice model determines the impact of water quality and other lake characteristics on the choice of a fishing site among the set of all potential sites. The model estimates the value of the available set of lakes to each New York resident. In the second step, a model predicts whether a New York resident will choose to fish on a particular day. Third, based on the results of the site-choice and fishing decision models, it is possible to estimate the change in economic welfare caused by altering water quality of the lakes available to New York residents.

We use the Montgomery and Needelman model by inputting MAGIC acidification estimates and simulating the impact on anglers. We do not re-estimate the econometric model's parameters for this application due to resource constraints, though it is important to note that the model was originally estimated to describe angler response to acidity at pH 6.0, while we assume that anglers respond at a lower pH level. Data from MAGIC are input to the model in the form of a percentage estimate of the lakes in the Adirondacks that fall below a chosen pH level. The effect of acidification has a negative impact on the utility of anglers that might wish to use that resource. By simulating the effect on anglers' utility of acidifying a percentage of lakes within a region, the model can compute the economic impact of a specific level of acidification. Subtracting the economic value of fishing at our baseline level of acidification from that which would occur if the CAAA were promulgated provides an estimate of the benefits accrued to recreational fishermen from reducing acidification in the region.

## Acidification Results

We summarize the results of our acidification projections from MAGIC in Exhibit 19. Each scenario described in these tables provides an estimate of the percentage of lakes in the Adirondacks likely to suffer from acidification given the deposition and

[16]The Montgomery and Needelman model applies a technique developed by Morey, Rowe, and Watson (1993).

nitrogen saturation parameters assigned to that scenario. The exhibits present the level of acidification expected given a specific threshold level of acidity (pH 4.6, 5.0, or 5.4), covering uncertainty associated with the impacts of acidity on a range of aquatic species.

Table E-15 shows that in the year 2010, the CAAA can be expected to reduce the number of lakes whose pH falls below 4.6 by zero percent, below 5.0 by one to four percent, and the percentage of lakes falling below a pH of 5.4 by five percent. These results are obtained by subtracting the acidification estimates for the year 2010 without the CAAA from the acidification estimate for 2010 with the CAAA. Note that we only compare scenarios with consistent nitrogen saturation parameters. One can not reliably compare the impacts of acidification on a nitrogen-saturated watershed in one deposition scenario with a non-saturated watershed in another deposition scenario.

**Table E-15**
**Acidification Results - 2010**

| Year | Status of CAAA | Level of N Saturation | Percentage of Lakes Acidified at Selected pH Levels | | |
|---|---|---|---|---|---|
| | | | **pH 4.6** | **pH 5.0** | **pH 5.4** |
| 1990 (Base Year) | No CAAA Regulations Promulgated | | 0% | 5% | 20% |
| 2010 | With CAAA | No Saturation | 0% | 2% | 18% |
| | | Saturated | 0% | 5% | 17% |
| | Without CAAA | No Saturation | 0% | 6% | 23% |
| | | Saturated | 0% | 6% | 22% |
| **Range of Benefits from CAAA in 2010** | | | **0%** | **1%-4%** | **5%** |

Acid deposition between 1990 and 2010 also contributes to lagged acidification impacts after 2010. MAGIC estimates that a significant amount of acidification between 2010 and 2040 is avoided by the CAA. This is an area that requires further research in order to fully quantify these impacts.

## *Economic Results*

As acidification of the Adirondacks is reduced by the CAAA, economic benefits accrue to society. In annual terms, the economic benefits of the CAAA in 2010 are summarized in Table E-16. Following the presentation used for the acidification data, the range of annual benefits from the CAAA is $12 million to $49 million using an effects threshold of pH 5.0, and $82 to $88 million for an effects threshold of pH 5.4. These results correspond to previous analyses (Englin et al, 1991; Mullen and Menz, 1985; Morey and Shaw, 1990) that find annual benefits to the Adirondacks of halving utility emissions in the millions to tens of millions of dollars. We do not provide an economic assessment of acidity in 2040, as the behavioral model is not sufficiently robust to estimate economic impacts 50 years into the future.

**Table E-16**
**Annual Economic Impact of Acidification in 2010**
**(Millions of 1990 Dollars)**

| Year | Status of CAAA | Level of N Saturation | Economic Impact of Acidification at Each pH Threshold | |
|---|---|---|---|---|
| | | | pH 5.0 | pH 5.4 |
| 1990 (Base Year) | No CAAA Regulations Promulgated | | $61 | $320 |
| 2010 | With CAAA | No Saturation | $24 | $281 |
| | | Saturated | $61 | $261 |
| | Without CAAA | No Saturation | $73 | $363 |
| | | Saturation | $73 | $349 |
| **Range of CAAA Benefits in 2010** | | | **$12-$49** | **$82-$88** |

We calculate the cumulative economic benefits from the CAAA by summing the difference between the discounted annual economic impact of acidification with and without the CAAA for every year from 1990 to 2010.[17] We perform this calculation for the minimum and maximum values for each of the three pH thresholds for survival of recreational fish species, assuming a straight line increase in the level of acidification between 1990 and the 2010 level for each scenario. We present the results as a cumulative net present value calculated with 1990 as the base year (i.e. costs in 2010 are discounted over 20 years). As indicated in Table E-17, the range of cumulative potential benefits from the CAAA between 1990 and 2010 is from $67 to $465 million.

[17]The formula is $\sum (c_t - f_t)/(1+r)^t$
Where: c = economic impact in the baseline, or counterfactual case;
f = economic impact with the CAAA, or the factual case;
t = the year, where 1990 is year 1;
r = the social discount rate, in this case we use 5%.

**Table E-17**
**Cumulative Economic Benefits of Acidification from 1990 to 2010**
**(Millions of 1990 Dollars)**

| | Economic Impact of Acidification at Each pH Threshold | |
|---|---|---|
| | pH 5.0 | pH 5.4 |
| CAAA Benefit Minimum | $67 | $433 |
| CAAA Benefit Maximum | $271 | $465 |

## *Avoided Cost of Liming*

An additional factor that must be considered in light of the context of economic damages from acidification is the possibility of mitigating these damages by local means. In the case of the Adirondacks, acidic lakes are systematically limed in order to raise pH and improve habitability for recreational fish species, which can be stocked after liming. This alternative is costly for local resource managers, and difficult to conduct in most Adirondack lakes with limited access, but it does serve to locally mitigate the damages caused by acid deposition. Naturally, damaged aquatic ecosystems can not be entirely replaced by liming and restocking, but impacts to recreational fishing may be minimized. Currently a limited number of lakes are limed in the Adirondacks. In this section we examine the economic implications of this practice and demonstrate that liming will be necessary both with and without the CAAA.

The goal of liming in the Adirondacks is to mitigate the effects of acidification by the addition of acid neutralizing products in selected waters to maintain and/or restore brook trout populations. Waters may be considered for liming and re-stocking, if the pH drops below 6.0.[18] The present liming program is limited in scope due to policy constraints, environmental regulations, and factors affecting the economic feasibility. With a few exceptions, lakes are typically not limed, if any of the following applies: the water is considered naturally acidic; the flushing rate is greater than two times a year; the water will not support brook trout regardless of the pH of the water; or, liming of the water would be too expensive, due to its remote location.[19]

It is not possible to predict the number of lakes that the New York Department of Environmental Conservation (NYSDEC) would choose to lime and restock in 2010 based on the available data, but we can estimate the potential costs and impacts if the program remains constant from 1990 to 2010, or if it grows at NYSDEC's proposed rate of two additional lakes per year. In 1990 approximately 25 percent of the region's lakes suffered a pH below 6.0, according to MAGIC, and NYSDEC limed and monitored 32 lakes and restocked 30. MAGIC estimates that the same percentage of lakes will maintain a pH below 6.0 in 2010, both with and without the CAAA. Table E-18 presents the cumulative costs associated with liming lakes in the region from 1990 to 2010.

[18]Personal communications with Larry Straight, Rick Costanza (NYSDEC, Region 5), and Bill Gordon (NYSDEC, Region 6).

[19]NYSDEC, 1990 and personal communications with Larry Straight, Rick Costanza (both at NYSDEC, Region 5), and Bill Gordon (NYSDEC, Region 6).

**Table E-18**
**Cumulative Cost of Ph Stabilization from 1990 to 2010**
**(Millions of 1990 Dollars)**

| | Number of Lakes | Cost of Liming | Cost of Monitoring | Cost of Stocking | Total Cost |
|---|---|---|---|---|---|
| **Program Remains Constant** | 32 | $0.11 | $0.07 | $0.23 | $0.40 |
| **Program Grows by Two Lakes per Year** | 72 | $0.16 | $0.10 | $0.35 | $0.61 |

Under the current plan to lime lakes with a pH below 6.0, this practice will continue under both scenarios of our analysis - with and without the CAAA. Therefore, liming costs will be incurred regardless of regulatory efforts. What we can not determine is the impact that liming may have on our avoided damages analysis. It is possible that liming lakes with the greatest recreational potential will offset the majority of economic impacts of acidification, though due to the structure of our economic modeling approach it is not possible to test this hypothesis at present. On the other hand, it is important to note that liming is feasible only on lakes where access is very easy, and therefore is limited in the scope of its impact. Furthermore, as previously stated, liming is a stop-gap measure that is both temporary and not a complete substitute for restoring natural ecosystem conditions.

## Caveats and Uncertainties

The impacts of acid deposition in the eastern United States include both terrestrial and aquatic ecosystem damages. Many of these effects are difficult to measure, and most are impossible to monetize given current methods. The result is that our analysis treats a very narrow definition of the impact of acidification. A far more broad definition would include costs associated with damaging the integrity of terrestrial and aquatic ecosystems, many of which are not quantifiable at this time. Nevertheless, our case study of the Adirondacks region demonstrates that the CAAA is generating substantial economic benefits in just the narrow scope of recreational fishing. Our analysis states that by mitigating the impacts to recreational fisheries from acidification with the regulations promulgated under the CAAA, benefits can be accrued in the hundreds of millions of dollars.

The limitations that affect these estimates are caused by data and computational constraints at each stage of the simulation process. We detail each of these limitations below, and indicate the directional bias these limitations may create in our final benefits estimates.

## Emissions, Deposition, and Acidification Estimates

Each of the models that contribute to the acidification estimates (i.e. the emissions model, RADM, and MAGIC) has been rigorously tested. For example, MAGIC estimates of acidification have been tested extensively including the following procedures: individual process formulations in the model have been tested against laboratory experiments with soils; model hindcasts of historical lake chemistries in the Adirondacks have been made and compared with values inferred from lake sediment records; and, predictions of the effects from whole-watershed manipulations have been compared to observed effects. Nevertheless, it is well documented that MAGIC estimates suffer from unquantified uncertainty, parameterization, and validation problems (EPA, 1995).

It is beyond the scope of this report to dissect MAGIC, RADM, or the emissions projections to identify factors that might affect their results. Furthermore, it is not possible to quantify the cumulative uncertainty that propagates in the linking

of these models to provide acidification estimates. It is sufficient to note that the acidification estimates generated by MAGIC should be treated with proper caution, applying sensitivity analysis to any further modeling work that uses these data as input. Several limitations are detailed below.

- We consider the potential effects of nitrogen saturation on lake acidification by performing sensitivity analysis of two boundary conditions, representing a nitrogen-saturated watershed and a watershed with complete nitrogen uptake. While this approach allows us to estimate the range of effects nitrogen saturation may have on acidification of surface waters, it does, however, not account for the fact that nitrogen saturation is a continuous process leading to increased leaching of nitrogen compounds as a watershed progresses through the various stages of nitrogen saturation (see for example: Stoddard, 1994).

- The sample of lakes simulated by MAGIC must be extrapolated to the entire population of lakes in the region. In order to simulate the complex hydrological, biological, and chemical dynamics of lakes, intensive data collection is required, forcing the developers of MAGIC to limit the number of simulated lakes to only 33. This sample represents lakes with an ANC of less than 400 microequivalents per liter (μEq/L). Lakes with greater ANC are believed not to be vulnerable to acidification from acid deposition. The results from MAGIC therefore are only applicable to those lakes with ANC less than 400 μEq/L, but we have no assurance that the sample of 33 lakes is representative of the distribution of lake ANC levels below 400 μEq/L in the total population. In addition, we are forced to use pH 7 as a proxy for ANC 400 μEq/L where ANC data is not available. Though pH and ANC are correlated, there is significant variance in this relationship.

## Ecological Factors

In the ecological assessment two major limitations arise. It is not possible to address either of these limitations with sensitivity analyses, so it is important to keep in mind that results may be biased by these factors.

- Acidic episodes may significantly affect fish populations. They are, however, not considered in our analysis due to significant limitations of our ability to model episodic events.

- It is well documented that pH is not the only factor that determines fish survival, although we do use it as the single indicator for ecological health. This overlooks the importance of other components of water chemistry such as aluminum and calcium concentrations. This is necessary because there is insufficient data for our geographic region to develop a sufficiently sophisticated ecological-economic model that would consider all these variables. Because we test several pH thresholds at which anglers might perceive declining fish populations, this simplification of ecosystem dynamics should not bias our final economic estimates.

## Economic Estimates

The economic model is also subject to some uncertainty that may bias our monetary results. Again, it is not possible to address all of these limitations with sensitivity analyses, so it is important to keep in mind that results may be biased by these factors.

- This analysis includes only day trips to sites within three hours of the angler's home. We take no account of people who would come and spend several days fishing at a site. By excluding these people we likely understate the costs of increased acidity.

- This model treats every day as a potential fishing day, and assumes that each fishing occasion is independent of all others. Neither assumption is realistic. The implication is that our measure of the seasonal costs are likely biased upward.

- Montgomery and Needelman do not compute a confidence interval for the value estimates of lake acidification in their study. This could result in an overestimate of the economic impact of acidification as we can not determine that the value estimates are significantly different from zero.

- Even though this study offers a much more comprehensive set of alternative fishing sites than most recreational fishing studies, we were unable to account for rivers and streams, or for lakes and ponds in nearby states. To the extent that these alternative sites are substitutes for New York lakes, our welfare measures may overstate the costs of acidification.

- We assume that anglers perceive the effects of acidification at a pH threshold lower than that at which the random utility model was estimated. This is a conservative approach which potentially underestimates the total impact of acidification, but overestimates the benefits of the CAAA because the difference between the percentage of lakes that are acidified in our baseline and CAAA scenarios is larger at lower threshold pH levels.

## Timber Production Impacts from Tropospheric Ozone

The purpose of this section is to evaluate the prospective benefits of improved commercial timber growth through the reduction of tropospheric ozone concentrations attributable to the CAAA. Tropospheric ozone ($O_3$) is a secondary pollutant that is created in the atmosphere by a photochemical reaction among nitrogen oxides (NOx) and volatile organic compounds (VOCs). Documented scientific evidence suggests that elevated ozone concentrations in the troposphere disrupt ecosystems by damaging and slowing the growth of vegetation. We examine one aspect of these impacts in this analysis, reduced commercial timber growth, and find the cumulative impacts from 1990 to 2010 to be $1.87 billion.

### *Ecological Effects of Ozone*

In terms of forest productivity and ecosystem diversity, ozone may be the pollutant with the greatest potential for regional-scale forest impacts (NAPAP, 1991). Studies have demonstrated repeatedly that ozone concentrations commonly observed in polluted areas can have substantial impacts on plant function (see U.S.EPA 1996; De Steiguer 1990; Pye 1988 for summaries).

Like carbon dioxide ($CO_2$) and other gaseous substances, ozone enters plant tissues primarily through apertures in leaves in a process called stomatal uptake. To a lesser extent, ozone can also diffuse directly through surface layers to the plant's interior (Winner and Atkinson 1986). Once ozone reaches the interior of plant cells, as a highly reactive substance, it inhibits or damages essential cellular components and functions, including enzyme activities, lipids, and cellular membranes, disrupting the plant's osmotic (i.e., water) balance and energy utilization patterns (U.S.EPA 1996; Tingey and Taylor 1982). Damage to plants is commonly manifested as stress specific symptoms such as chlorotic or necrotic spots, increased leaf senescence (accelerated leaf aging) and reduced photosynthesis. All these factors reduce a plants' capacity to form carbohydrates (U.S.EPA 1996), which are the primary form of energy storage and transport in plants. Reduction of carbohydrate production and disruption of carbon allocation patterns in turn can impact the growth rates of trees, shrubs, herbaceous vegetation and crops.

In this section we focus on the economic impacts of reducing commercial timber growth on the U.S. economy. Timber supply is a direct ecological service flow affected by tropospheric ozone and is therefore

an ideal quantitative example of the benefits of controlling tropospheric ozone in the U.S. Nevertheless, it is important to note that this benefit represents only a small portion of the overall ecological benefits of reducing the impacts of tropospheric ozone on ecosystems across the nation.

## Modeling Timber Impacts from Ozone

In this section we describe our methods for quantifying the impacts of tropospheric ozone on commercial timber production. The assessment of the benefits of regulating tropospheric ozone involves three major steps:

- Estimation of ambient ozone concentrations under a regulatory and a non-regulatory scenario;
- Estimation of the growth changes from ozone exposure on commercial forests;
- Estimation of the economic impact of changes in commercial timber growth.

We describe the completion of each of these steps, the models we use and their input data. Upon completion of these steps it is possible to compare the tropospheric ozone concentrations, ecological effects, and resultant economic impacts over the period 1990-2010 both with and without the CAAA. The net difference between ecosystem effects and economic impacts with and without the CAAA represents the benefits accrued to society from the implementation of the CAAA.

### *Step 1: Estimating Ambient Ozone Concentrations*

In order to simulate the impacts of ozone on commercial forest productivity we must estimate the ambient ozone concentrations at which forests are exposed both with and without the regulations of the CAAA. We accomplish this using historical ambient ozone data for 1990, and projected ozone data for the years 2000 and 2010. We use historical hourly ozone concentrations from EPA's Aerometric Information Retrieval System (AIRS).[20] AIRS is a comprehensive database that contains ambient air quality monitor data for the contiguous U.S. To estimate future year concentrations of ozone we use the Urban Airshed Model (UAM-V), a three-dimensional photochemical grid model that calculates concentration of pollutants by simulating the physical and chemical processes in the atmosphere.

### *Step 2: Ecological Effects*

We use the PnET-II model to estimate the impacts on timber growth of elevated ambient ozone. The model assesses the average change in productivity for softwood and hardwood forests in each of nine timber growing regions defined by the U.S. Forest Service (see following section). The strength of PnET II is that it provides a means to use a geographically transferable method to assess forest stand-level estimates of the impacts of ambient ozone on productivity. For the purposes of a national assessment, this provides a significant advantage over alternative existing methods based on plant-level models (e.g. the tree grow model (TREGRO)) or expert opinion surveys (e.g. . Pye et al. 1998; deSteiguer 1990). The disadvantages of the model include: the use of photosynthetic rates as an indicator of ozone impacts rather than a mechanistic measure of respiratory change; potential bias created when scaling net primary productivity (NPP) changes in plants to the forest stand level; and the use of an ozone measure that may be overly sensitive to changes in ambient concentrations (D40[21] ). We discuss the major facets of the model's construction below.

The PnET-II model is a monthly time step, canopy- to stand-level model of forest carbon and water balances based on several generalized relationships (e.g. maximum net photosynthesis as a function of foliar nitrogen content). Carbon and water balances are linked in that potential evapotranspiration is determined as a function of leaf gas exchange rates and the atmospheric vapor

[20] See "www.epa.gov/airs",

[21] The D40 measure represents the cumulative ozone dose above a threshold concentration of 40 ppb.

pressure deficit (i.e. humidity). Actual evapotranspiration is determined from a comparison of potential evapotranspiration with available soil water, which is affected by precipitation, snow melt, direct evaporation from canopy surfaces, soil water holding capacity and a fast flow fraction that represents macropore flow to below the rooting zone.

The model simulates a multi-layered forest canopy that includes gradients in available light, specific leaf weight and hence, leaf level carbon gain. Annual, whole-canopy carbon gain is allocated to leaves, wood and roots after calculations for growth and maintenance respiration costs. The model has been successfully validated for forest production and water balances at a number of temperate and boreal forest sites. For a full description of model algorithms, inputs, assumptions, sensitivity analyses and validation exercises, see Aber and Federer (1992), Aber et al,.(1995) and Ollinger et al. (1998).

PnET-II uses an algorithm to allow prediction of ozone effects on forest growth that relates ozone-induced reductions in net photosynthesis to cumulative ozone uptake (Ollinger et al. 1997). Uptake is determined for ozone concentrations above 40 ppb and is affected by ozone exposure levels and leaf gas exchange rates. Application at sites located across the northeastern US show an interesting interaction between ozone and water availability whereby the occurrence of drought stress reduced ozone damage via reductions in stomatal conductance, and hence, ozone uptake.

### *Step 3: Economic Impacts*

To monetize the ecological effects of elevated ambient ozone on commercial timber production it is necessary to estimate the market changes that result from reduced timber growth rates. We use the USDA Forest Service Timber Assessment Market Model (TAMM) to analyze the changes in timber inventories that would result under each of our ozone exposure scenarios, and the consequent changes in harvests, prices and regional demand for timber. Using the inventory and market computations, TAMM estimates the overall economic welfare impact of changes in forest growth rates, in terms of changes in consumer and producer surplus. Previous peer-reviewed EPA analyses of changes in timber productivity (U.S.EPA 1997) use this same model.

There are three stages to the economic estimation. First forest growth rate information generated by PnET-II is provided to the forest inventory tracking component of TAMM, called Aggregate Timber Land Assessment System (ATLAS). Growth rate information is provided for each of the forest production regions defined by TAMM. (We do not simulate changes in the Canadian regions for this analysis.) Second, ATLAS generates an estimate of forest inventories in each major region, which in turn serves as input to the market component of TAMM. In the third stage, TAMM estimates the future harvests and market responses in each region. A detailed description of TAMM's structure is found in Adams and Haynes (1996).

## *Ecological Results*

P-Net II partitions NPP of forest trees according to tissue type. Changes in NPP for wood tissue result in changes in tree growth rates. On the whole, P-Net II estimates that commercial timber growth rates are improved as a result of reduced tropospheric ozone exposure attributable to the CAAA. The improvement in growth rates by the year 2010 ranged from negative 0.56 percent to 10.91 percent. Table E-19 summarizes the estimated changes in growth rates, by region, for the entire U.S.

**Table E-19**
**Difference in Commercial Timber Growth Rates With and Without The CAAA**

| Region | Difference in 2000 | | Difference in 2010 | |
|---|---|---|---|---|
| | Softwoods | Hardwoods | Softwoods | Hardwoods |
| PN W-E | 1.68% | 1.58% | 2.11% | 1.25% |
| PN W-W | 1.17% | 0.42% | -0.56% | 1.13% |
| S. West | 0.84% | 1.77% | -0.14% | 1.59% |
| N.Rocky | 2.67% | 0.40% | 4.46% | 2.05% |
| S. Rocky | 4.77% | 2.25% | 4.14% | 3.88% |
| S. Central | 4.54% | 4.80% | 7.93% | 8.41% |
| S. East | 5.40% | 5.65% | 10.38% | 10.91% |
| N. Central | 1.80% | 5.74% | 4.36% | 9.22% |
| N. East | 4.27% | 6.68% | 9.58% | 11.49% |

It is important to note that the difference in growth rates gradually grows from zero percent in 1990 to the values presented for 2000, and then 2010. In other words, the difference in growth rate estimated for 2010 is not experienced over the entire 1990-2010 modeling period.

## *Economic Impacts*

TAMM estimates that there is a measurable difference in timber harvests attributable to ozone exposure under our two scenarios. At the outset of our modeling period, early 1990s, virtually no change is measured in forest harvest volumes. This is an expected result because increases in growth rates should not substantively affect timber volume over so short a period of time. By the end of our modeling period, late 2000s, increased growth rates over the previous decade(s) begin to affect overall forest yields in the form of harvestable timber. This is observed in Figure E-8 as an increasing annual benefit estimate over the modeling period.

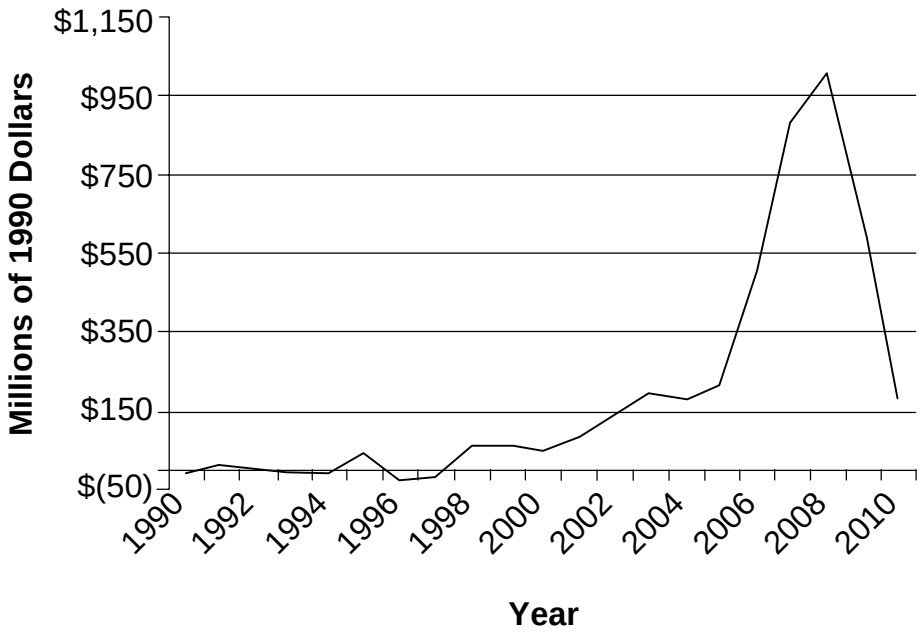
The shape of the benefits time-series reveals a production shift in one region of the United States as a result of increased timber availability. This shift produces a spike in economic surplus for a period of three years. Although this change is small in percentage terms relative to total economic surplus generated by the timber sector it contributes to a large portion of the benefits estimate over the 1990-2010 period.

The cumulative value of annual benefits is calculated as the sum of the annual differences in consumer and producer surplus from commercial timber harvests under our CAAA and no CAAA ozone exposure scenarios from 1990 to 2010. We discount annual benefits to 1990 dollars using a five percent discount rate. The total cumulative benefits estimate is $1.87 billion.

## *Caveats and Uncertainties*

In interpreting results from this analysis, several points should be considered. First, large-scale analyses of complex ecosystem processes are typically conducted with simulation models because it is impossible to conduct large-scale manipulation experiments that would provide similar predictive capabilities. This brings with it inherent uncertainties in that there may be little or no data with which to validate model predictions. In the case of ozone effects on forest production, the absence of controlled, whole-forest fumigation studies across the range of climatic, vegetation and pollution conditions experienced across the U.S. makes it presently impossible to validate all model predictions. In this

**Figure E-8**
**Annual Economic Welfare Benefit of Mitigating Ozone Impacts on Commercial Timber: Difference Between the Pre-CAAA and Post-CAAA Scenarios**




analysis, we have combined established empirical relationships between ozone exposure and plant physiological function in a peer-reviewed model that is based on sound forest growth processes. As such, the resulting model predictions should be viewed as a set of refined hypotheses, but nevertheless, hypotheses that have not been thoroughly tested.

Second, while ozone has repeatedly been identified as an important environmental stress agent affecting forest vegetation, it is not the only such factor to which forests are currently exposed at regional to global scales. Human activities have profoundly affected global cycles of carbon, nitrogen and a number of other elements in ways that may be at least as important as ozone. Because a number of changes (e.g. elevated $CO_2$ and increased atmospheric nitrogen deposition) have significant potential to cause large-scale fertilization effects, growth predictions that include ozone effects alone should be viewed as incomplete.

***Ozone Modeling***

- Because it is not possible to model ozone levels throughout the country during the months of October through April during future years, it is necessary to employ another method to obtain estimates for ozone levels during these months. We assume that ozone levels during these months for 2000 and 2010 will be identical to the levels during the same months of 1990. Thus, any differences in timber production under the two scenarios of CAAA promulgation and no CAAA promulgation will be driven solely by ozone differences during the warmer part of the year that comprises the majority of the growing season.

- It is important to note that ozone monitoring is not complete, with coverage especially low in forested regions of the United States.

Only two percent of ozone monitors are in forested areas (U.S.EPA 1996). We work with the best possible estimates of tropospheric ozone concentrations but identify this as a significant area of uncertainty in this analysis.

### *Ecological Modeling*

- Preliminary model results revealed an interesting and unexpected interaction between ozone, drought stress and carbon allocation. On moist, productive sites, ozone resulted primarily in decreased wood growth because the simulated trees can afford to lose wood without reducing more important tissues which are given higher allocation priority in the model (leaf and root). On progressively colder or drier sites, ozone exposure causes reductions in all plant tissues because plants are already stressed enough that additional reductions in carbon gain must come from all plant pools. Complex interactions among ozone, drought and this carbon allocation dynamic produced unexpectedly variable results, which, in some cases caused an increase in growth in response to ozone. Although these are interesting and biologically feasible interactions, in the absence of any real data in this area, it is impossible to determine the extent to which they actually occur.

### *Economic Modeling*

- There are two important caveats to the economic modeling. First, we generalize changes in growth rates for entire forest types across potentially heterogeneous regions. TAMM is capable of modeling timber growth and harvest with greater precision, breaking down forests into many species and age-classes and by county. We do not anticipate that increasing the precision of growth rate data on a national scale would substantially alter our results.
- The second caveat is economic benefits may be underestimated by using so short a modeling period. It is evident from the data we present that improved growth takes years to affect actual harvests. Therefore, the complete benefits of improved growth during 1990 to 2010 will not be accrued until after 2010. By not including these years in our analysis we can not fully account for the commercial timber benefits of ozone mitigation over the period of our analysis.

## Carbon Sequestration Effects

It is possible to extend the analysis of timber growth rates to account for the differences in temporary and long-term carbon sequestration under each of our ozone scenarios. This is accomplished by linking two USDA Forest Service Models to TAMM/ATLAS to generate estimates of carbon sequestered in standing forest, and carbon sequestered in commercial forest products. We briefly summarize those steps here.

TAMM/ATLAS provides an estimate of the standing timber stock and the commercial timber harvests that will occur under each of our ozone exposure scenarios. Using this information we estimate the respective volumes of carbon sequestered in each scenario using the forest carbon model (FORCARB) and harvested carbon model (HARVCARB).

FORCARB contains a set of stand level carbon budgets that relate the timber growth and yield output from ATLAS to trends in total ecosystem carbon over the course of stand development. These include carbon sequestered in trees, woody debris, understory vegetation, and the forest floor. Using these data, FORCARB estimates the total carbon sequestered in commercial forests at any point in time. This information provides a useful baseline for the rate of forest carbon sequestration that can be expected under different ozone exposure scenarios. For a complete description of FORCARB and its application see Turner et al. (1993) and Turner et al. (1995).

The age of natural forests and the management regime of commercial forests largely determine the fate of forest carbon. In natural forests, carbon sequestration is temporary, with sequestered carbon eventually returning to the nutrient cycle. Alternatively, harvested timber is transformed into commercial products that alter the life cycle of sequestered carbon. Using the HARVCARB model, we use harvest information from TAMM to track the lifecycle of timber. The ultimate fate of this sequestered carbon depends on the efficiency of timber conversion (i.e. how much timber becomes a product), and the durability of that product. HARVCARB estimates the long-term carbon sequestration resulting from timber harvests under each of our scenarios. A full description of HARVCARB is found in Row and Phelps (1990).

Forest ecosystems help mitigate increasing anthropogenic carbon dioxide emissions by sequestering carbon from the atmosphere, converting atmospheric carbon into biological structures or substances needed in physiological processes. Some air pollutants, however, may adversely affect the potential of forests to sequester carbon by slowing down the rate of biomass accumulation of sensitive forest tree species. This may affect the global carbon cycle and may contribute to anthropogenically induced changes in the earth's climatic conditions.

Using output from TAMM/ATLAS, timber inventories can be converted into estimates of carbon sequestered in commercial forests by a forest carbon model (FORCARB). FORCARB estimates the carbon storage in each of four ecosystem components: trees; forest understory, forest floor, and soil. The model uses forest carbon storage and flux estimates based on ecological analyses of each of the forest ecosystem components. The details of these studies and their synthesis into the FORCARB model can be found in Birdsey (1992a, 1992b) and Heath and Birdsey (1993). Heath and Birdsey (1995) provide a technical description of integrated simulations using TAMM/ATLAS and FORCARB. Of the carbon sequestered in forests, some portion is subsequently harvested as timber and processed into wood products, paper, and biomass fuel. We use a harvest carbon model (HARVCARB) to estimate the life-cycle of harvested forest timber and thereby adjust the forest carbon sequestration estimates of FORCARB. HARVCARB relies on a range of assumptions approximately 50 percent of harvested wood ultimately becomes a wood or paper product, the remainder becomes waste from the production process. Of the final wood and paper products, a small percentage become durable products or are landfilled and decompose at a rate of less than one percent a year (Row and Phelps, 1990). Wood that is either manufactured into a durable product (e.g. permanent building construction material, furniture) or materials that are landfilled (e.g. paper) contribute to long-term carbon sequestration. The remainder of the harvested wood mass (e.g. biomass fuel, non-durables that are not landfilled) is re-released to the environment and therefore is not included in the volume of carbon estimated to be sequestered in forests.

We find that forest carbon sequestration increases with improved air quality under the CAAA. This result corresponds with the intuition that forests tend to grow faster when tropospheric ozone exposure is reduced. Carbon flux, or annual forest carbon sequestration minus forest harvest losses (excluding long-term carbon sequestration in forest products) is also greater under the CAAA than under our No-CAAA air quality scenario. We summarize our results in Table E-20.

**Table E-20**
**Differences in Carbon Flux (millions of metric tons/year)**

| | 1990-1999 | 2000-2010 |
|---|---|---|
| Forest Flux | 8 | 28 |
| Land Use Change | > -1 | > -1 |
| Cumulative Fate of Removals | > 1 | > 1 |
| **TOTAL FLUX** | **8** | **29** |

Forest carbon flux attributable to the CAAA represents approximately four to sixteen percent of anticipated total carbon flux in U.S. forests between 1990-2010.

In the event of a binding international carbon mitigation agreement, the implication of this result is that substantial costs of carbon mitigation may be avoided by improved forest growth attributable to the CAAA. Though it is not possible to evaluate the monetary value of the avoided cost at this time due to uncertainty regarding the actual cost of carbon mitigation, it will be possible to estimate the value using the data in this analysis once reliable carbon mitigation costs become available.

### *Caveats and Uncertainties*

Additional caveats and uncertainties associated with the estimation of carbon sequestration in U.S. commercial forests include the following:

- FORCARB estimates are based on a synthesis of a variety of empirical studies of the four ecosystem components (soil, forest floor, understory, and trees). The total error of the composite of these studies is not treated explicitly as a modeling output.

- FORCARB also estimates the carbon storage and flux for a variety of forest types based on a synthesis of empirical studies. The error associated with extrapolating these data across a variety of forest ecosystem types is not explicitly treated.

- HARVCARB utilizes data on the life span of durable wood products that is over 50 years old, originally compiled by the Internal Revenue Service for purposes of calculating depreciation of these products. Though the authors of HARVCARB state that this data continues to be reliable, changes in construction, product and their uses most likely biases these data. No estimate is made of the magnitude or direction of this bias.

## Aesthetic Degradation of Forests

The purpose of this section is to evaluate the prospective benefits of forest aesthetic improvements associated with improved air quality attributable to the CAAA. In order to assess these benefits, we first evaluate the known changes in visible injuries over time. Available scientific methods and data on the visual appearance of forest stands and their impact on perceived forest aesthetics, however, make it difficult to precisely describe changes in forest aesthetics. Nevertheless, it is possible to describe a range of visual impacts that may be caused by air pollutants and their potential effect on forest aesthetics. Second, we assess the economic value associated with such aesthetic changes. The focus of much of this work tends to be site-specific, describes the aesthetic impacts of a number of causal factors, and utilizes a variety of experimental methods making it difficult to generalize results. We conclude that air quality improvements attributable to the CAAA should result in improved forest health, possibly providing aesthetic

value to society in the range of billions of dollars. A more detailed description of this analysis is found in *Characterizing the Forest Aesthetics Benefits Attributable to the 1990 Clean Air Act Amendments, 1990-2010* (IEC, 1999c).

## Forest Aesthetic Effects from Air Pollutants

Air pollution can cause a wide variety of visual injuries to forest stands, ranging in severity from subtle injuries (e.g., minor leaf discoloration) to severe forest decline (e.g., extensive defoliation and death of trees). The severity of symptoms depends on many factors including the atmospheric concentration of air pollutants, the sensitivity of tree species to air pollution and the presence of other environmental stress factors (Fox and Mickler, 1995; Eagar and Adams, 1992; Olson et al., 1992; Smith, 1990).

Many CAAA-regulated air pollutants are associated with visual symptoms, including, but not limited to, tropospheric ozone, sulfur dioxide and hydrogen fluoride, the three major pollutants known to have caused significant visual injuries to forest trees in the past (NAPAP, 1987). Other air pollutants known to potentially cause visual injuries to plants are strong mineral acids, precursors of which are also regulated by the CAAA (NAPAP, 1987). In addition, there are a variety of other air pollutants potentially affecting the visual appearance of plants, including heavy metals such as lead and mercury (EPA, 1997d; Gawel et al., 1996; Smith, 1990; NAPAP, 1987); nitrogen oxides; ammonia; peroxyacetyl nitrate; chlorides; and ethylene (Smith, 1990; NAPAP, 1987; Jacobson and Hill, 1970). However, very limited information is presently available on visual damages caused by these pollutants. Tables E-21 and E-22 summarize the known visual impacts of air pollutants on forests and their geographic extent.

As a consequence of complex natural forest dynamics, lack of extensive long-term monitoring networks, and difficulties in establishing cause and effect relationships, it is not possible to quantify the extent of visual forest injuries caused by air pollutants or changes that may have occurred since the implementation of the CAAA. In addition, mechanisms that induce threats to forests may operate on such a long-term scale that benefits in the visual appearance of forests may not be exhibited during the period of our analysis.

**Table E-21**
**Typical Impacts of Specific Pollutants on the Visual Quality of Forests**

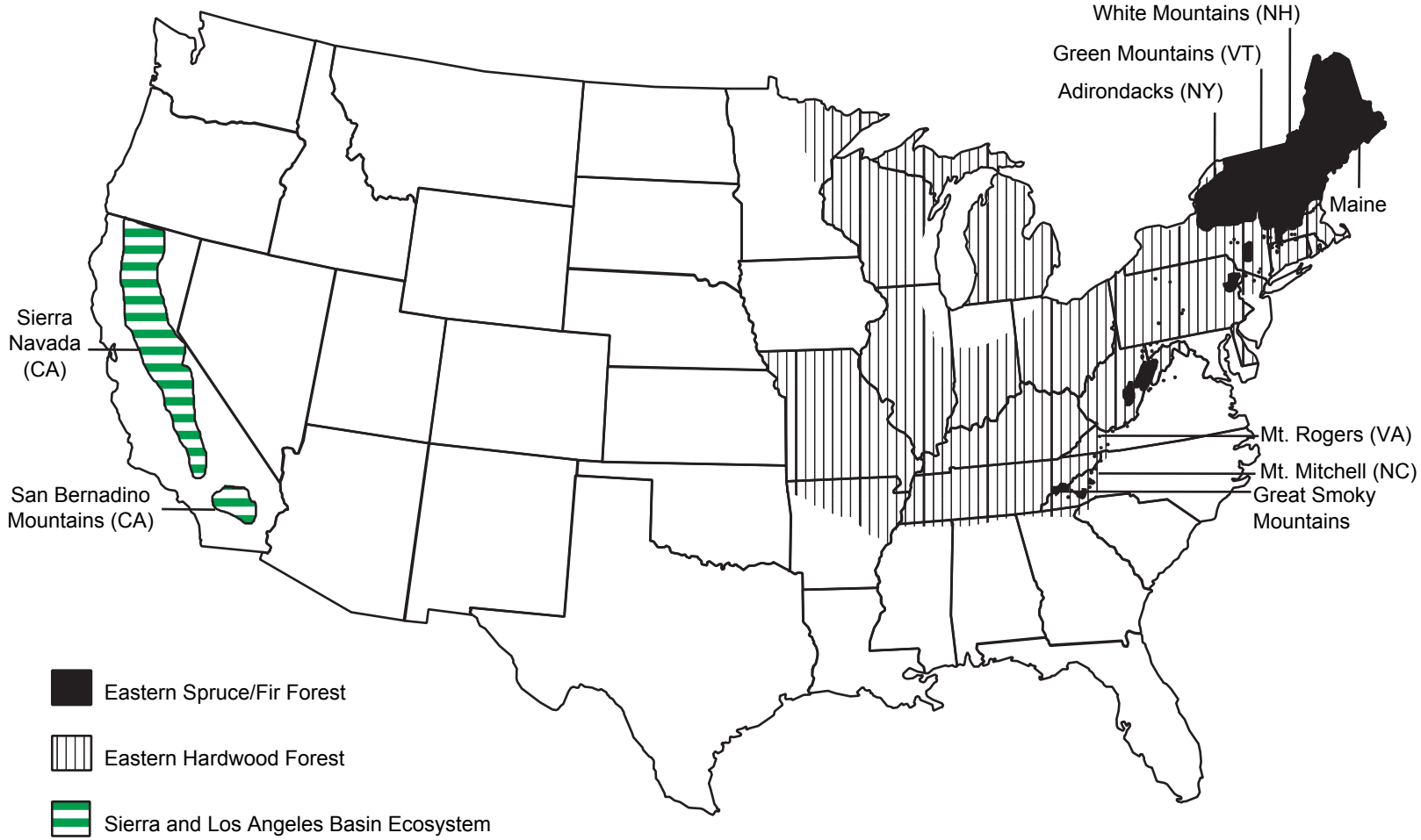
| | Geographic Extent | Direct/Indirect Injuries | Major Types of Visual Injuries |
|---|---|---|---|
| Ozone | Area or regional effects | Direct injuries | Foliar injuries (e.g., pigmented stipple), increased needle/leaf abscission, premature senescence of leaves. Pattern, size, location, and shape of foliar injuries to indicator species can be specific for ozone. |
| | | Indirect Injuries | Increased susceptibility to visual injuries that may result from other adverse environmental factors, such as insect attacks. For example, increased needle/leaf abscission, elevated mortality rates, and/or changes in species composition. |
| Acidic Deposition | Area or regional effects | Indirect Injuries | Increased susceptibility to visual injuries that may result from other adverse environmental factors, such as climatic factors. For example, increased needle/leaf abscission, elevated mortality rates, and/or changes in species composition.<br><br>Acidic deposition can also cause direct foliar injuries. Acids are, however, more likely to indirectly affect the visual appearance of forest trees, unless exposure levels are very high. |
| Sulfur Dioxide | Point source pollution | Direct Injuries | Foliar injuries including leaf/needle discoloration and necrosis. Pattern, size, location, and shape of foliar injuries to indicator species can be specific for sulfur dioxide. At high concentrations, elevated mortality rates of sensitive species and changes in species composition may occur.<br><br>Sulfur dioxide may also cause indirect injuries. Indirect injuries, however, are not well documented. |
| Hydrogen Fluoride | Point source pollution | Direct Injuries | Foliar injuries including leaf/needle discoloration and necrosis. Pattern, size, location, and shape of foliar injuries to indicator species can be specific for sulfur dioxide. At high concentrations, elevated mortality rates of sensitive species and changes in species composition may occur.<br><br>Hydrogen fluoride may also cause indirect injuries. Indirect injuries, however, are not well documented. |

**Table E-22**
**Forests Affected by Regional Pollution**

| Affected Forest Type / Species | Region | Major Air Pollutants | Documented Visual Injuries | Suspected Mechanisms of Injury | Sources |
|---|---|---|---|---|---|
| Mixed Conifer Forest / Ponderosa and Jeffrey Pines | San Bernardino Mountains, California | Ozone and nitrogen containing substances | Foliar injuries include chlorotic mottle, tip necrosis, premature senescence of needles, and increased needle abscission. Elevated mortality rates and changes in species composition have occurred. | Direct ozone-induced foliar injuries. Heavy bark beetle attacks facilitated by drought, ozone, and nitrogen containing air pollutants.<br><br>Ponderosa and Jeffrey pine have shown air pollution-related symptoms of decline probably since the mid 1950s. | EPA 1996a; Miller, 1992; Stolte et al., 1992; NAPAP, 1991; Miller and McBride, 1998 |
| | Sierra Nevada, California | Ozone | Foliar injuries include chlorotic mottle, tip necrosis, premature senescence of needles, and increased needle abscission. | Direct ozone-induced foliar injuries.<br><br>The Sierra Nevada contains the largest forest area in the world with documented damage from a non-point source pollutant but ozone exposure and injuries are not as severe as in the San Bernardino Mountains. Visible ozone-induced foliar injuries were first documented in the early 1970s. | EPA, 1996a; Peterson and Arbaugh, 1992; NAPAP, 1991; Miller and Millecan, 1971 |

| Affected Forest Type / Species | Region | Major Air Pollutants | Documented Visual Injuries | Suspected Mechanisms of Injury | Sources |
|---|---|---|---|---|---|
| Spruce-Fir Forest / Red Spruce | High elevation areas in the northern Appalachians. | Acidic deposition (esp. acidic cloud water), and ozone | Foliar dieback, bud injury, foliar loss, Elevated mortality rates. | Acidic deposition increases the susceptibility of red spruce to winter injury (freezing).<br>A dramatic increase in the frequency of winter injury in red spruce stands occurred in the late 1950s and 1960s, coincident with a significant increase in the emissions of precursors of acidic deposition. | EPA, 1995a; Johnson et al., 1992; DeHayes, 1992; NAPAP, 1991 |
| | High elevation areas in the southern Appalachians. | Acidic deposition (esp. acidic cloud water) and ozone | Crown thinning and pockets of high red spruce mortality have been detected on a few mountain sites. Ozone-induced foliar injury. | Acidic deposition leads to nutrient imbalances through accelerated foliar leaching and soil acidification. Soil acidification is characterized by a loss of soil nutrient cations and occurrence of toxic aluminum levels. Also: direct foliar injuries caused by ozone. | EPA, 1995a; Johnson et al., 1992; Johnson and Fernandez, 1992; Cook and Zedaker, 1992; NAPAP, 1991 |
| Eastern Hardwood Forest / Sugar Maple | Northeastern US and Canada | Acidic deposition and ozone | Crown thinning, branch dieback, elevated mortality rates | Acidic deposition leads to nutrient imbalances through accelerated foliar leaching and soil acidification. Soil acidification is characterized by a loss of soil nutrient cations and occurrence of toxic aluminum levels.<br>During 1980s sugar maple declined in many stands in the northeastern US and Canada. Involvement of acidic deposition in sugar maple decline has not been demonstrated but cannot be ruled out. | USFS, 1995b; EPA, 1995a; NAPAP, 1991 |

**Figure E-9**
**U.S. Major Forest Types Affected by Air Pollution-Induced Visual Injuries**




Note: Only areas affected by non-point pollution are shown. Scientific certainty varies with location. Direct ozone-induced injuries also occur in several other locations not indicated (e.g., Southern Forests, Berraug et al, 1995).
Sources: NAPAP, 1991 and White and Cogbill, 1992.

Despite limitations in detecting trends in forest health and associated causal agents, it is possible to identify areas in the US that contain forests known or suspected to experience visual injuries. Forests affected by high concentrations of air pollutants in the vicinity of point sources may provide useful case studies because cause and effect relationships may be easier to establish and visual injuries can be severe enough to cause significant aesthetic impacts. In particular, point sources can lead to well-defined concentration gradients in the prevailing downwind direction causing corresponding gradients of visual injuries (Smith, 1990; NAPAP, 1987).

In contrast, concentrations of regionally distributed air pollutants (e. g., ozone and acidic deposition), can be fairly uniform over large geographic areas. Visual symptoms can be more intense in the vicinity of urban areas or industrial sites but may not be limited to these regions (NAPAP, 1987) making it more difficult to establish cause-and effect relationships. Despite difficulties in establishing cause-and-effect relationships, all identified forest ecosystems likely to have experienced air pollution-induced visual injuries in recent history are affected by regionally distributed air pollutants.

## Economic Value of Changes in Forest Aesthetics

Though studies that attempt to estimate the value of changing aesthetics are limited in number and scope, they do suggest that people value forest aesthetics and change outdoor recreational behavior according to the quality of forest health in recreational areas. The shear volume of forest-based recreation in the United States suggests that improvements in forest aesthetics could result in substantial benefits. For example, the United States Forest Service reports that recreation visitor days to national forests have increased over the last ten years from 250 million to over 350 million. With the potential magnitude of aggregated individual preferences in mind, we review several studies that relate individual preference for forests with respect to overall appearance and attempt to extend these analyses to those regions where forests are most affected by air pollution.

Peterson *et al.* (1987) used the contingent valuation (CV) method and a hedonic property valuation model to estimate willingness to pay to avoid ozone-induced forest damage in the Los Angeles area. This contingent valuation survey involved two samples: one made up of recreationalists in the greater Los Angeles area, and the second made up of individuals who owned property within the boundaries of the San Bernardino and Angeles National Forests. Each group was shown a set of photographs depicting varying degrees of vegetative damage. Mean WTP by recreationalists and residents were found to be approximately $43 and $137 per household per year, respectively. The hedonic analysis revealed a significant and positive WTP to avoid homes located in forested areas exhibiting ozone damage. Using these two methods, total damages resulting from the current levels of ozone induced forest injury were estimated to be between $31 and $161 million per year. The study authors rejected a significant percentage of responses as "protest" or "inconsistent" bids (40 percent), which would indicate that many respondents may not have understood or accepted the scenario and the commodity being valued. Apart from this, the study also does not address a series of concerns related to the application of CV to assess nonuse values. First, the survey instrument did not include reminders of budget constraints or substitute goods and services. Second, the survey did not clearly define the commodity. The WTP scenario did not clearly indicate how forest damages were to be mitigated.

Walsh *et al.* (1990) interviewed 200 individuals representing the general population of Colorado and were shown three color photographs representing three levels of forest quality. The mid-level quality was said to represent the present state of the forest (100 to 125 live trees measuring more than six inches in diameter at breast height (dbh) per acre). Respondents were asked their WTP to prevent the lowest state (zero to 50 live trees measuring more than six inches dbh per acre) and attain the highest state (125 to 175 trees per acre in this size class). All respondents were informed beforehand that the damage being valued was due to pine beetle and spruce budworm infestations. Mean WTP per respondent was estimated to be $47. An evaluation of the Walsh et al (1990) study reveals several notable

strengths. The survey included reminders of budget constraints, and the authors ensured that respondents were familiar with the commodity being valued and were accustomed to paying for access to recreation sites with good forest quality. Only five percent of the responses were rejected as "protest" or "large" bids. Weaknesses of the study include a small sample size (198), inconsistency between results solicited using different question formats (iterative bidding vs. direct question), and potential biases attributable to framing the question as one of the most important issues affecting Colorado residents and the possibility of a "warm glow" affect concerning payment for a social cause.

Holmes et al (1992) used a CV survey to determine WTP to protect threatened spruce-fir forests in Southern Appalachia from insect and air pollution damage. In this study, residents within 500 miles of Asheville, NC were surveyed about their willingness to pay to eliminate damages to regional spruce-fir forests. The authors used two survey formats, discrete choice and payment cards. The mean willingness to pay for protecting the spruce-fir forests was $20.86 using the payment card method, and $99.57 using the discrete choice method. The study ensured that the sample had adequate knowledge of the commodity being valued, and the overall sample size was large. Unfortunately, several weaknesses arise from the fact that the sample was divided into two groups in order to test different survey formats. The study used a small sample size for each of the tested methods (232 and 236, respectively). The number of protest bids was small (7 to 10 percent), indicating that the respondents understood the function of the survey, but the final results generated by the two different methods were substantially different. This study was later revised in Holmes and Kramer (1996), where the results were published as mean willingness to pay of $36.22 for forest users, and $10.37 for nonusers.

## Extending Economic Estimates to a Broader Area

These studies provide an incomplete picture of the total benefits that could be obtained by eliminating visual damages to forests associated with air pollution in the country. As an illustrative calculation, we extend the range of valuation estimates provided in Peterson (1987); Walsh et al. (1990); and Holmes and Kramer (1996) to the major regions of affected landscape in the United States. We do not estimate aesthetic value as a function of forest damage from varying levels of air pollution, but rather provide an estimate of the values placed on avoiding damages characteristically experienced during the 1980s in the United States.

In Table E-23 we present the results from the three studies. We base our calculations of benefits on the value per household of avoiding forest damages multiplied by the number of households in the study region.

In Table E-24 we present the results of an illustrative calculation that extends the "market" for this commodity to a broader group of households. The annual value of avoiding the forest damages is the product of the range of household values in Table E-23 and the total number of households in the states most affected by air pollution.

**Table E-23**
**Summary of Monetized Estimates of the Annual Value of Forest Quality Changes**

| Study | Aesthetic Change Valued | Value of Change per Household (Current Dollars) | Value of Change per Household (1990 Dollars)[i] | Total Annual Value of Change for Region (Current Dollars) | Total Annual Value of Change for Region (1990 Dollars)[i] |
|---|---|---|---|---|---|
| Peterson et al. (1987) | Ozone damage to San Bernardino and Angeles National Forests | $6.31-$32.70[ii] | $7.26-$37.62 | $27-$140 million | $31-$161million |
| Walsh et al. (1990) | Visual damage to Colorado's Front Range | $47 | $61.68 | $55.7 million | $73.09 million |
| Holmes and Kramer (1996) | Visual damage to spruce-fir forests in southern Appalachia | $10.81 nonusers<br>$36.22 users | $10.37 nonusers<br>$34.76 users | NA | NA |

Note: i.) Values adjusted using all item Consumer Price Index, Economic Report of the President, 1998. Years for current dollar estimates: Peterson et al, 1987; Walsh et al, 1983; Holmes et al, 1991.
ii) Based on 4.3 million households in Los Angeles, Orange, and San Bernardino counties.
iii) Assumes 2.5 million households in North Carolina and 1.8 million in Tennessee.

**Table E-24**
**Illustrative Value of Avoiding Forest Damage in the United States**
**(1990 Dollars)**

| Affected System | States Included | Value per Household | Households[i] | Estimated Total Annual Value[ii] | Cumulative Value (1990-2010)[iii] |
|---|---|---|---|---|---|
| Sierra Nevada and Los Angeles Basin | CA | $7.26-$37.62 | 10.4 million | $75.5 million -$391.2 million | $1.02 billion - $5.27 billion |
| Eastern Spruce Fir and Selected Eastern Hardwood | ME, VT, NH, MA, NY, PA, WV, TN, KY, NC, VA | $7.26-$37.62 | 23.2 million | $168 million - $872.8 million | $2.27 billion - $11.75 billion |

Notes: i.) Household data from 1990 Census; ii) Total Value = Households x Value per Household; iii)Assumes a 5 percent real discount rate.

The results of existing work in this area suggest that improvements in air pollution controls result in positive changes in the aesthetic quality of forest stands. Pollutant control provisions of the 1970 CAA and the 1977 CAAA, for example, may have resulted in a significant decrease or elimination of forests visually affected in the vicinity of emission sources. Further reductions in air pollution emissions mandated by the 1990 CAAA should result in additional improvements in forest health and associated economic benefits derived from improved forest aesthetics.

Our illustrative calculation of the regional effects of improving the aesthetic quality of forest stands (Table E-24) likely overstates the extent of market for this commodity. Estimates presented in Table E-23, however, based on a more conservative application of the extent of market for this commodity, provide a better basis to estimating the order of magnitude of

this category of effects of air pollution on ecosystem health. Considering only the Peterson et al. and Walsh et al. studies, conducted in two areas that have been shown in previous assessments to be affected by accumulated air pollution damages, estimates of the total annual value of improvements in the aesthetic quality of forests are in the $100 million to $250 million range.

### Caveats and Uncertainties

To quantitatively assess the effects of air pollution emission reductions on forest aesthetic benefits, considerable amounts of high-quality data are required. These data include extensive long-term monitoring networks producing consistent and comparable information over time frames as long as several decades. In addition, injuries captured by monitoring networks have to be linked to the causal agent(s), a task that is currently associated with high factors of uncertainty. Only rarely, if ever, is air pollution the only factor negatively affecting forest health. Typically, a variety of adverse environmental factors act synergistically to induce injuries, considerably limiting our ability to detect air pollution as one of the factors causing injury and to quantitatively assess the amount of injuries attributable to air pollutants.

There are caveats to the use of benefits transfer in this context. The application of this method is intended to provide an order of magnitude estimate of the benefits associated with avoided aesthetic damages to forests in the United States. More sophisticated estimation methods will be required if a truly accurate estimate of value, especially the marginal value of incremental changes, is to be derived. Following is a summary of the caveats to using this approach.

- The impacts that we value are not equivalent to those avoided through the implementation of the CAAA, they are historical effects. A comprehensive assessment of forest aesthetics-related benefits associated with improvements in air quality is limited by significant factors of uncertainty occurring in both the natural science component of the assessment and the economic analysis. Factors of uncertainty in natural sciences include difficulties detecting trends in forest health in general, attributing changes in forest health to specific factors such as air pollution, and establishing valid dose-response relationships of forest exposure to air pollutants and resulting visual injuries.

- The types of aesthetic deterioration in the original studies are not necessarily the same as those experienced in other regions. The nature of forest aesthetic deterioration will vary (e.g. the yellowing of conifer needles vs. gypsy moth defoliation of hardwoods) as will the intensity.

- We do not fully assess the range of potential substitutes for the aesthetic health of regional forests to each household. Having ready substitutes could lower the value a specific household might place on aesthetic quality of regional forests.

- The distinction between marginal values for forest health and average value is not made. As marginal values for changes in forest health diverge from the assumed average value in this analysis, the estimates develop bias.

- We assume that differences in average regional income do not affect estimates.

## Toxification of Freshwater Fisheries

The purpose of this section is to assess, from 1990 through 2010, the ecological benefits likely to accrue as a consequence of reductions in the emissions of hazardous air pollutants (HAPs), as mandated by the CAAA. Title III of the CAAA lists 189 chemicals considered to be HAPs. Ideally, a comprehensive economic analysis of the ecological benefits of CAAA-mandated reductions in HAP emissions would include analyses for all service flows potentially affected by the emissions of HAPs. However, a broad quantitative analysis of all these benefits is not yet scientifically possible. What is

possible is a qualitative analysis of the likely benefits of reduced HAP emissions for recreational fishing. A more detailed description of this analysis is found in *Economic Benefits of Decreased Air Toxics Deposition Attributable to the 1990 Clean Air Act Amendments*, 1990-2010 (IEc 1998d).

## Impacts of Toxic Air Emissions

Five HAPs, mercury, PCBs, chlordane, dioxins, and DDT were responsible for nearly 95 percent of the fishing advisories extant in 1995 (EPA 1996b). The use of three of these compounds (PCBs, chlordane, and DDT) was effectively illegal in the United States prior to 1990 (EPA 1992a), and there are currently no plans for additional CAAA regulations of these compounds (Federal Register Unified Agenda 1998). The remaining two HAPs, mercury and dioxins, are therefore the focus of this analysis.

Because the ecosystem responses to toxic contamination are poorly understood, and observable service flow impacts are difficult to model, we use fishing advisories as a measure of the extent of toxic contamination. In addition, we can characterize the economic impact of HAPs emissions based on altered fishing behavior caused by toxic contamination of freshwater fisheries. It is important to note that fishing advisories alone do not provide a comprehensive view of impacts of toxic contamination on ecosystems, and more expansive measures should be examined in future research.

Fishing advisories are issued by state and tribal agencies when the levels of toxins in the tissue of fish exceed limits established by both state and federal authorities. Fishing advisories generally take one of four forms:

- Advisory for any consumption by the general population;
- Advisory for pregnant women, nursing mothers, and children;
- Advisory for limitation on consumption based on size of fish and frequency of consumption; and
- Advisory for limitation on consumption for specific sub-populations.

According to the U.S. Fish and Wildlife Service (1998), the total number of advisories in the U.S. in 1997 was 2,299, increasing five percent from 1996. The number of water bodies under advisory represents 16.5 percent of the nation's total lake acres and 8.2 percent of total river miles. In addition, 100 percent of the Great Lakes waters and their connecting waters and a large portion of the nation's coastal waters are also under advisory. The total number of advisories in the U.S. has steadily increased for mercury and dioxin.

Mercury is responsible for approximately 75 percent of all fish consumption advisories in effect in 1995 (EPA 1996b). Mercury from point sources, as opposed to mercury deposited from the atmosphere, may be responsible for many of these advisories. The lakes and streams with advisories are concentrated in the northern portions of Minnesota and Wisconsin, as well as in Florida, Missouri, Indiana, Ohio, North Carolina and New England (EPA 1997a, 1997d). Judging by fish consumption advisories, fish mercury levels do not appear to be a widespread problem in the remainder of the United States, and EPA (1997d) found that the typical consumer eating purchased fish is not at risk of methylmercury poisoning. Approximately three percent of fish consumption advisories in effect in 1995 were due to the presence of dioxins (EPA 1996b), and in 1996, 18 states had one or more water bodies under advisement because of dioxin levels in fish (EPA 1997a). Dioxins from point sources may be responsible for many of these advisories.

Several limitations to the fish advisory data exist. First, many lakes, rivers and streams have not been analyzed for toxicity, and it is possible that advisories eventually will be issued for these water bodies. Table E-25 summarizes the sampling intensity for toxicity through 1997. Second, current levels of toxics in watersheds may result in future toxification of healthy water bodies, even in the absence of additional future HAP deposition. Therefore, the current set of fish advisories underestimates the magnitude of toxification from air deposition to date. Third, the protocol for fishing advisory issuance may vary from

**Table E-25**
**Summary of National Data on Toxicity Sampling for Fishing Advisories**

| Water Body | Percentage of Water Bodies Assessed for Contaminants | Percentage of Assessed Water Bodies Under Advisory |
|---|---|---|
| Lakes (acres) | 11.36 | 78.61 |
| Rivers and Streams (miles) | 2.41 | 29.58 |

Source: EPA 1997a

state to state, removing any consistent basis on which to judge the levels and causes of fisheries' toxicity for each state.

## *Illustration of Economic Cost to Anglers*

The economic welfare implication of water quality changes to recreational fishing are well studied. Most literature in this field focuses on the impacts of deteriorating water quality in a specific fishery. More recently, economic models are appearing that address the social welfare cost of water quality deterioration in multiple fisheries within a region. Such an approach accounts for choices made by fishermen concerning travel to, and the attributes of (e.g., fish advisories), multiple fisheries. Random utility models (RUM) provide the computational method for these regional analyses.

Montgomery and Needelman (1997) were the first to use direct water quality measures in conjunction with a RUM approach to analyze the economic impacts of toxification on regional anglers. Using data from the New York Department of Environmental Conservation (NYDEC), Montgomery and Needelman identify 23 water bodies with toxicity advisories among 2,561 lakes and ponds in the state. Using water quality data and geographic location of both water bodies and anglers, the authors estimate the economic cost of the toxification within the state. The results are presented in Table E-26.

**Table E-26**
**Estimates of the Welfare Cost of Toxification in New York State (1990 Dollars)**

| Level of Toxicity | Compensating Variation per Trip | Compensating Variation per Capita per Day | Compensating Variation per Capita per Season |
|---|---|---|---|
| Toxic Contamination | $1.23 | $0.37 | $51.51 |
| Site Closed Due to Toxic Contamination | $1.69 | $0.50 | $70.92 |

Source: Montgomery and Needelman 1997

The results from Montgomery and Needelman indicate that the economic welfare implication of existing toxic contamination is substantial for New York State, as described below:

$0.37/person/day x 17,990,000 people x 140 fishing days/season = $931,882,000/season.

In perpetuity,[22] the value of eliminating toxicity in New York State, using a five percent discount rate, is calculated to be $18,637,640,000.

Clearly, the results using these assumptions are very large. Applications of this model for purposes of estimating the effects of air toxics deposition on recreational fishing requires further investigation of the assumptions in this model.

Jakus et al. (1997) conducted a similar RUM analysis of toxification of reservoirs in Tennessee. Data from the Tennessee Valley Authority showed fishing advisories for two of 14 reservoirs in central Tennessee, and six of 14 reservoirs in the eastern portion of the state. Again, using water quality data and the geographic locations of both water bodies and anglers, Jakus et al. (1997) estimated the economic impact of the fish consumption advisories. Anglers living in central Tennessee suffered a $17.92 per trip per season loss from the advisories, and anglers in eastern Tennessee suffered a $38.27 loss (1990 dollars). Therefore, considering an angler population of 146,450 individuals, the impact of this level of toxification into perpetuity, using a five percent discount rate, is approximately $65.96 million.

These results indicate that fish advisories impose substantial economic cost on anglers in the United States. Measuring the marginal changes in toxification that would occur in the absence of the CAAA is not possible, but it is plausible to state that continued HAP emissions impose a cost on society if they result in the issuance of additional fish advisories. Any efforts to minimize these emissions, including the CAAA, may generate corresponding benefits.

If air deposition of toxics results in statewide fishing advisories (e.g., Connecticut, Washington D.C., Illinois, Maine, Massachusetts, Missouri, New Hampshire, New Jersey, New York, North Carolina, Ohio, Vermont), substitution away from recreational fishing for other activities may begin to occur. No models are available to estimate the economic impact of a large-scale substitution away from recreational fishing. The RUM approach does not adequately capture the magnitude of ubiquitous toxification because the models measure only the choice to participate in the activity and not the welfare implications of participation in alternative activities, nor do they account for the industries that provide supplies and services to anglers in the region. However, the economic cost of statewide advisories could be substantial.

Although Montgomery and Needelman (1997) and Jakus et al. (1997) examined only two areas of the country - New York State and part of Tennessee - their work demonstrates that HAP emissions have a measurable economic cost when the consequence of these emissions is the issuance of fish advisories for recreational fisheries. While it is not possible to measure the differences in HAP deposition and the marginal ecological impacts that will result from the CAAA, it is clear that continued emissions of HAPs will result in further toxification of aquatic resources, and reductions in HAP emissions may provide economic benefits.

The toxification of freshwater ecosystems in the United States by mercury and dioxins is a problem, and emissions of mercury and dioxins to the atmosphere contribute significantly to the problem. Quantifying the magnitude of ecosystem effects of air toxics deposition is not yet possible, but it is clear that the deposition of air toxics to some ecosystems, such as freshwater recreational fisheries, can result in measurable economic costs.

[22] A perpetuity is a stream of benefits, accrued over an infinite time horizon. A simplified formula for calculating a perpetuity of equal benefits accrued annually, in which the first payment is received at the end of year one, the second payment is received at the end of year two, etc., is: (Nominal Value of Benefit) / (Discount Rate)

### Caveats and Uncertainties

Because of limitations in the currently available data and models, a comprehensive quantitative analysis of the ecological benefits of reduced mercury and dioxin emissions for recreational fishing is not possible. However, such an analysis may be possible in the foreseeable future.

- The potential for mercury and dioxins to persist for long periods of time in the environment is a confounding factor in this analysis. Because these pollutants can persist in aquatic ecosystems for decades, even though the CAAA may reduce their emissions, it is possible that the status of toxified ecosystems may not be significantly affected during the time frame of the analysis (i.e., through 2010).

- In addition, the persistent nature of toxification presents challenges with respect to how benefits are discounted over time. In those cases where recovery from toxification will take a number of years, the benefits accrued by society will be diminished in terms of their present value. In other words, if all air emissions ceased, many fish consumption advisories would remain in place until the fisheries recovered. If this recovery period were to extend for several decades, the present value of economic benefits from the eventual retraction of advisories could be reduced dramatically. In a cost-benefit decision analysis, these benefits might not justify the costs of HAP regulations. In this case, an inter-generational benefits assessment, where discounting is not applied, would be required.

- The global nature of mercury pollution is another confounding factor Because a significant portion of mercury deposited within the U.S. comes from the global pool, a decrease in U.S. emissions may be offset by increases in emissions in other countries. If this should occur, it might be difficult to detect or predict actual changes in the toxicity of U.S. aquatic ecosystems, despite reductions in U.S. emissions.

- To quantitatively assess the effects of mercury and dioxin emission reductions on recreational fishing, more and better data and models are required. The most pressing research needs in this area are a model that can predict the national fate and transport of dioxin, and models that can, on a national scale, convert mercury and dioxin deposition quantities to amounts of the contaminants in fish. Data to verify these models is also highly desirable.

- Even if it were currently possible to perform the analysis discussed here, it would likely capture only a fraction of all the benefits attributable to CAAA-mandated HAP emissions reductions. The analysis focused entirely on two HAPs and on one endpoint. Neither the potential benefits of reductions in the emissions of other HAPs nor other endpoints were considered.

## Conclusions and Implications

Our analysis has identified four major categories of air pollutants that affect ecological structure and function: sulfur compounds, nitrogen compounds, tropospheric ozone, and hazardous air pollutants. Each of these pollutants is scientifically documented as a cause of ecosystem degradation due to acute and chronic exposure. Sulfur and nitrogen compounds contribute to episodic and chronic acidification of aquatic and terrestrial ecosystems, while the chronic deposition of nitrogen compounds alone may cause harmful eutrophication to terrestrial and aquatic ecosystems. Tropospheric ozone disrupts the normal functioning of plants, leading to acute, visible damages to terrestrial ecosystems, and chronic exposure at levels that do not produce acute damages may result in reduced growth rates and eventually alter ecosystem nutrient cycling. Finally, hazardous air pollutants deposited across the landscape are accumulating in aquatic organisms and subsequently entering both

aquatic and terrestrial foodchains. Though the ecological impacts are not fully understood, the long-term effects of introducing hazardous air pollutants to ecosystems may be slow to manifest and irreversible in nature.

Ecological effects can occur at different levels of biological organization. Most effects that are currently quantifiable are understood at the individual or population level, perhaps because of the feasiblity of conducting controlled experiments at this level. For example, research on the effects of ozone on timber began with experimental research on the response of seedlings and leaves of mature trees to elevated levels of ozone. Only recently have modeling efforts begun to consider interactions of factors at the community level, taking into account the dynamics of competitive relationships among tree and plant species. Experimental research continues to progress toward a better understanding of the full range of ecological impacts including effects at the ecosystem level. Continued consideration of these higher-order effects of pollutants on ecological systems is necessary for a more complete understanding of the benefits of pollution control.

Because the chronic ecological effects of air pollutants may be poorly understood, difficult to observe, or difficult to discern from other influences on dynamic ecosystems, our analysis focuses on acute or readily observable impacts. Disruptions that may seem inconsequential in the short-term, however, can have hidden, long-term effects through a series of interrelationships that can be difficult or impossible to observe, quantify, and model. This factor suggests that many of our qualitative and quantitative results may underestimate the overall, long-term effects of pollutants on ecological systems and resources.

## *Summary of Quantitative Results*

Although the effects of air pollutants on ecological systems are likely to be widespread, many effects may be poorly understood and lack quantitative effects characterization methods and supporting data. In addition, many of our quantitative results reflect an incomplete geographic scope of analysis; for example, we generated monetized acidification results only for the Adirondacks region of New York State. As a result, quantitative results we generate for the purposes of estimating the benefits of the CAAA reflect only a small portion of the overall impacts of air pollution on ecological systems. Our quantitative overview of effects nevertheless suggests that the overall impacts of air pollution are far greater than those quantified.

**Table E-27**
**Summary of Monetized Ecological Benefits (millions 1990$)**

| Description of Effect | Air Pollutant | Geographic Scale of Economic Estimate | Range of Annual Impact Estimates in 2010 | Primary Central Estimate for 2010 | Primary Central Cumulative Impact Estimate 1990-2010 | Key Limitations |
|---|---|---|---|---|---|---|
| Freshwater acidification | Sulfur and nitrogen oxides | Regional (Adirondacks) | $12 to $88 | $50 | $260 | - Captures only recreational fishing impact<br>- Incomplete geographic coverage leads to underestimate of benefits |
| Reduced tree growth - Lost commercial timber | Ozone | National | $190 to $1000 | $600 | $1,900 | - Uncertainties in stand-level response to ozone exposure<br>- Uncertainty in future timber markets |
| **TOTAL MONETIZED ECONOMIC BENEFIT** | | | $200 to $1,100 | $650 | $2,200 | - Partial estimate that omits major unquantifiable benefits categories; see text |

Note: Estimates reflect only those benefits categories for which quantitative economic analysis was supported. A comprehensive total economic benefit estimate would likely greatly exceed the estimates in the table. Range of estimates for timber assessment is based on variation in annual point estimates for 2005 through 2010.

Despite these limitations, it is important to recognize the magnitude of the monetized ecological benefits that we could estimate and reflect those results in the overall estimates of benefits generated in the larger analysis. Table E-27 provides a tabular summary of the results documented earlier in this appendix. It is not possible to indicate the degree to which ecological benefits are underestimated, but considering the magnitude of benefits estimated for the select endpoints considered in our analysis, it is reasonable to conclude that a comprehensive benefits assessment would yield substantially greater total benefits estimates.

## *Recommendations for Future Research*

Previous sections of this appendix have discussed several areas for future research related to the individual research and analytic efforts conducted. From a broader perspective, there are three key research needs to improve benefits assessments of this type:

- Exemplary assessments that incorporate a greater emphasis on ecosystem structure and function rather than specific service flows;
- Assessments with broader geographic coverage of impacts categories assessed in this report; and
- More sophisticated treatment of uncertainty and complexity, including careful consideration of the irreversibility of ecosystem impacts.

### Assessing Changes in Ecosystem Structure and Funtion

A major limitation of our quantitative analysis is that by focusing on individual acute and chronic impacts it is possible to lose sight of ecosystem-level changes to structure and function. These ecosystem-level changes could eventually lead to large-scale impacts far greater in degree and geographic extent. Determining the appropriate ecological level of analysis is crucial to properly account for ecological benefits that may accrue from environmental regulations. While quantifying the decrease in impacts on species attributable to air pollutant control is analytically tractable, the impact of pollutant reductions on ecosystem structure and function may be a more appropriate measure that can be further explored in future analyses.

Changes in ecosystem structure and function may not be obvious to the lay person, and the ultimate effects of such changes in ecosystems are sometimes unpredictable in scale and nature. Ecosystems affected by humankind may respond in a discontinuous manner around critical thresholds that are boundaries between locally stable equilibria. Complexity in ecosystems prevents analysts from using linear methods to "add up" the discrete ecological effects of pollution. Understanding the complex cause and effect relationships between pollution and deterioration of ecosystem structure and function is fundamental to making adequate policy decisions that will protect ecological resources. The isolation of service flows may often imply an oversimplified cause and effect relationship between pollution and the provision of the service flow, when more often the service flow is affected by complex non-linear relationships that govern ecosystem structure and function. The result is that ecosystem impacts may not be adequately assessed by analyses that focus on specific service flows.

One potentially fruitful approach to assessing impacts on the ecosystem scale would be to more adequately model a wide range of ecosystem functions that do not necessarily contribute to human welfare. Assessments at the watershed scale might provide an appropriate level of detail to more adequately characterize some of these intermediate service flows. This type of research effort would require close cooperation between air pollution specialists, ecologists, and economists to be most useful within the context of benefit-cost analyses such as this one.

### Broader Geographic Scale

Several of the ecological analyses conducted to support the first prospective section 812 report are limited by their partial geographic coverage. For example, while nitrogen deposition is an important contributor to eutrophication in a wide range of Eastern and Gulf Coast estuaries, resource, time, and data availability constraints, as well as limitations in our ability to reasonably apply an avoided cost

approach, prevented EPA from conducting a national economic assessment for this category of impacts. In this and many other effects categories, extension of the methods applied here to new geographic areas could greatly enhance the comprehensiveness of the physical effects and economic impact estimates.

## Alternative Treatment of Uncertainty

At present a variety of economic schools of thought are converging on quantitative analysis of environmental impacts that integrate uncertainty, irreversibility and ecological complexity. Efforts within the field of "ecological economics" to develop structured appraisals of uncertainty associated with environmental management and procedural rationale for decision making have yielded a variety of theoretical proposals. Drepper and Mansson (1993) argue that most aspects of uncertainty are compressed into the discount rate for policy analysis, resulting in the inappropriate use of a constant positive discount rate for environmental existence values. These existence values, they argue, may be more appropriately assigned negative discount rates. Faucheux and Munda (1997) advance a similar criticism of the unified discount rate and posit that a differentiated discount rate be applied to multiple aspects of a policy decision according to the implied uncertainty of each aspect. This quantitative approach evolves into a multi-criteria decision framework that departs from conventional cost-benefit analysis. Alternatively, Hinterberger and Wegner (1997) abandon quantitative analysis as a futile exercise due to ecosystem complexity in favor of simply applying the precautionary principal of reducing any and all environmental impacts that have uncertain outcomes.

In the resource economics literature, discussion of alternatives to cost-benefit analysis when the magnitude of benefits or costs are uncertain have focused on the concept of quasi-option value (see Freeman 1993 for a summary). The term was coined by Arrow and Fisher (1974) to describe the potential welfare gain of altering the timing of development/preservation decisions under uncertainty and when at least one of the choices involves an inrreversible commitment of resources (either spent or preserved). While much of the quasi-option value literature suggests that adopting this type of framework would lead to greater environmental protection, Freeman (1993) argues that it is also possible that the information gained by some incremental development of ecological resources might be the only way to reduce uncertainty and gain information about the magnitude of the trade-offs involved in preventing ecological exposures. It is nonetheless important to recognize that option and quasi-option value should not be considered as additional components of willingness-to-pay, but rather a value of altering decision making practices (e.g., the value of moving from a benefit-cost framework based on expected value to a framework that better considers the value of information gained over time and the irreversibility of certain effects).

The main implication of this body of work is that cost-benefit analysis may well underestimate the value of both the costs and benefits of uncertain, irreversible environmental outcomes from public policy. From the cost perspective, regulating a pollutant that may have no environmental consequence may cause economic losses that reduce unknown investment and growth opportunities in the future. From the benefits perspective, the value of preserving ecosystem integrity may include the mitigation of irreversible damage to a variety of service flows previously not associated with simplified dose-response relationships between pollution and ecosystems. Applications of these principles in economic assessments, including more rigorous assessments of option and quasi-option value, probabilistic analysis of multiple scenarios, and value of information approaches have the potential to greatly increase the utility of uncertain ecological assessment results for the purposes of making environmental policies.

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# Effects of Criteria Pollutants on Agriculture

Appendix F

## 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 control or alter growth and reproduction, thereby potentially increasing or decreasing yields of agricultural crops. Possible physiological effects of pollutants include: 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.

Air pollutants that might damage plants include $SO_2$, $NO_x$, peroxyacetyl nitrate (PAN), 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 and/or acid deposition, both 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),[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 between 1990 and 2010 that is projected to result from passage of the 1990 CAA Amendments (CAAA). 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 for a variety of reasons, mostly related to the absence of a national level benefits model (for vegetables and specialty crops) and difficulties in quantifying the physical impacts of air quality changes and their associated effect on welfare (for ornamentals). To estimate the economic value of ozone reductions under the CAAA, agricultural production levels expected from post-CAAA scenario ozone conditions are first compared with those expected to be associated with ozone levels projected under the pre-CAAA 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

For this analysis, the SUM06 index – a cumulative index of ozone concentrations over a specified threshold (0.06 ppm) – was selected to conform with the recent EPA ozone NAAQS benefits analysis.[2] The SUM06 index is one of several cumulative statistics that emphasize peak concentrations (in this case by use of a threshold), and may correlate more closely to crop damage than do unweighted indices.[3]

[1] Shriner et al., 1990; NAPAP, 1991.

[2] Abt Associates, 1998.

[3] Lefohn et al., 1988.

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.

Three main steps are used in the process of estimating the county-level SUM06 values:

(1) 1990 hourly ozone concentrations obtained for all available monitors from EPA's AIRS system.[4]

(2) For each county centroid, the 1990 hourly data from the closest set of monitors are temporally- and spatially-adjusted using UAM-V modeling data (as described in Appendix C), and the SUM06 is calculated for each monitor for each month.

(3) A distance-weighted average SUM06 is estimated for each month from the temporally- and spatially-adjusted monthly values.

One difference between the agricultural analysis and the health analysis is the treatment of distance extrapolation. The health effects results in this 812 analyses are calculated first for the population living within 50 km of monitors, and then for the whole country by extrapolating the air quality modeling results to provide universal coverage. The air quality modeling results near to monitors are believed to be more certain than the modeling for more remote areas. The less certain air quality modeling results is a very important issue for the agricultural analysis, as the majority of the commodity crops are grown in locations some distance from ozone monitors. Because only a small portion of cropland is within 50km of an ozone monitor, the agricultural analysis is not conducted for the within 50km of a monitor locations. The agricultural analysis is only conducted using the full national extrapolation of ozone modeling results.

## Calculation of the SUM06 Statistic

The hourly ozone concentrations are screened to identify those that equal or exceed 0.06 ppm, and these values are summed to obtain a "raw" monthly SUM06 index:

$$\sum_{j=day1}^{day\,30} \sum_{i=8:00\,AM}^{7:59\,PM} ozone_{i,j}\,,\ for\ all\ ozone_{i,j} \geq 0.06\ ppm$$

In this analysis, the SUM06 statistic was calculated on a monthly rather than a daily basis, reflecting the same hours of the day as if daily statistics had been individually calculated. Although a completeness criterion had been used to select monitors, there were still missing data for some included monitors. Therefore, this "raw" statistic was adjusted by a completeness ratio, the proportion of hours with available data to total hours in the period (either 12 in a day or 360 in a 30-day month), in order to address missing data as follows:

$$raw\ statistic * \frac{maximum\ hours\ per\ month}{actual\ hours\ in\ month}$$

The assumption implicit in using a completeness ratio is that the distribution of hourly ozone values for the missing data is the same as the distribution for the available data.

## October to April Ozone Concentration Data

Agricultural crop seasons extend the May to September period used in the health analysis, and the SUM06 index is cumulative, requiring data for the entire agricultural season. To address the need for SUM06 indices in months between October and April, 1990 monitoring data from AIRS were used --

[4]The analysis reflects the application of a 50 percent completion criterion, ensuring that included monitors have at least 12 hours of data for at least half the days in the modeling season.

no temporal- or spatial-adjustments were made to reflect potential ozone conditions in future years outside of the modeling season.[5]

## 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 at 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 post-CAAA and pre-CAAA 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 post-CAAA scenario percentage relative yield loss (PRYL) is compared to the minimum and maximum PRYL for the pre-CAAA scenario. Each step is discussed in more detail below.

### *Exposure-Response Functions*

Yield impacts resulting from changes in from ozone concentrations are estimated using exposure-response functions that are specific to each crop being 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. Consistent with EPA's ozone NAAQS benefits analysis, we used National Crop Loss Assessment Network (NCLAN)-based exposure-response functions that were derived using a Weibull distribution for available data, and a 12-hour SUM06 ozone index.

[5]AIRS data for all U.S. monitors were screened using the 50 percent completeness criterion for each month. All hourly data was converted to parts per million and rounded to the nearest 0.0001 ppm.

### Minimum/Maximum Exposure-Response Functions

Experimental data to evaluate the response of crops to ozone has been collected for a limited number of crops under the NCLAN program. The objective of this program was to employ a consistent experimental methodology to provide comparable results across crops. The crops included in the NCLAN experiments are corn, cotton, peanuts, sorghum, soybeans, winter wheat, potatoes, lettuce, kidney beans, tomatoes, and hay. For many crops, the NCLAN program evaluated the effects of ozone on several different cultivars. Although not necessarily representative of the full range of variability in crop response, the results for different cultivars do permit identification of a range of responsiveness. The most tolerant and responsive response functions are used to represent minimum and maximum impacts, *within the limits of available data.*

Use of cumulative exposure-response functions is relatively recent, and few experiments have been designed or reported in terms of the SUM06 index. Because the NCLAN program used a consistent protocol and developed a database of experimental conditions and results for all of its studies, U.S. EPA's Environmental Research Laboratory (ERL) was able to use original data from NCLAN studies to develop SUM06 exposure response functions for most NCLAN crops[6] (Lee and Hogsett, 1996). In addition, the agricultural model used in this analysis does not reflect non-commodity crops such as lettuce, tomatoes, potatoes, alfalfa, tobacco, turnips, and kidney beans. Table F-1 presents the exposure-response functions used in this analysis. Finally, one commodity crop, spring wheat, was excluded because the NCLAN exposure-response function was only developed for winter wheat.

Estimated responsiveness of a given crop to ozone varies within the NCLAN data. This range of response is partially explained by the program's

[6]Data were not sufficient to develop functions for tomatoes or hay.

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 actual 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, ERL conducted an analysis to identify the ozone concentration required to reduce yields by 10 percent for each crop cultivar using its 12-hour SUM06 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.

In this analysis, the maximum and minimum yield change results are used to bound a uniform distribution of possible yield change, recognizing that this distribution reflects only *known* potential yield losses. Each percentile change in yield, including the minimum and the maximum, is used to estimate a distribution of possible changes in economic welfare (see below).

**Table F-1**
**Ozone Exposure-Response Functions for Selected Crops (SUM06)**

| Ozone Index | Quantity | Crop | Function | Median Experimental Duration (Days) | Median Duration (Months) |
|---|---|---|---|---|---|
| SUM06 | Max | Cotton | 1-exp(-(index/78)^1.311) | 119 | 4 |
| SUM06 | Max | Field Corn | 1-exp(-(index/92.4)^2.816) | 83 | 3 |
| SUM06 | Max | Grain Sorghum | 1-exp(-(index/177.8)^2.329) | 85 | 3 |
| SUM06 | Max | Peanut | 1-exp(-(index/99.8)^2.219) | 112 | 4 |
| SUM06 | Max | Soybean | 1-exp(-(index/131.4)^1) | 104 | 3 |
| SUM06 | Max | Winter Wheat | 1-exp(-(index/27.2)^1.0) | 58 | 2 |
| SUM06 | Min | Cotton | 1-exp(-(index/116.8)^1.523) | 119 | 4 |
| SUM06 | Min | Field Corn | 1-exp(-(index/94.2)^4.307) | 83 | 3 |
| SUM06 | Min | Grain Sorghum | same as max (see above) | 85 | 3 |
| SUM06 | Min | Peanut | same as max (see above) | 112 | 4 |
| SUM06 | Min | Soybean | 1-exp(-(index/299.7)^1.547) | 104 | 3 |
| SUM06 | Min | Winter Wheat | 1-exp(-(index/72.1)^2.353) | 58 | 2 |

Source: Lee and Hogsett (1996)

## Calculation of Ozone Indices

The SUM06 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. For each crop included in NCLAN ozone exposure-response experiments, the period of ozone exposure reflected only a portion of the crop's growing season. The duration of the NCLAN experiments was provided by ERL, and reflects the duration of the function that provides the median responsiveness to ozone exposure. 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 calculate the SUM06 index for the appropriate growing season, state-level data on planting and harvesting dates was used in this analysis.[7] To calculate the cumulative SUM06 index, the experimental duration for each crop was anchored on that crop's harvest date in each state in order to most closely approximate the relevant period of exposure for yield analysis. The harvest date was assumed to be the first day in the month of harvest, so that the SUM06 index includes the months up to but not including the harvest month. Because the baseline and regulatory ozone data were developed as monthly SUM06 values, for the first month of the duration period the proportion of remaining days to days in the month were used to adjust the monthly SUM06 value. The SUM06 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.[8]

The form of the exposure response functions is an exponential function based on a Weibull distribution of the original NCLAN data, estimated to predict a yield loss relative to conditions of "clean air" (charcoal filtered/zero ozone) , or a zero SUM06 value. The resulting equation is in the form of:

$$Y = 1 - e^{\left[-(SUM06/B)^{C}\right]}$$

where:

| | | |
|---|---|---|
| Y | = | predicted relative yield loss (PRYL), expressed as a decimal value (i.e., not multiplied by 100 to report as a percent loss), and relative to a zero SUM06 (or clean air) condition |
| SUM06 | = | cumulative SUM06 ozone statistic at a specified level of spatial representation, in ppm |
| B, C | = | statistically estimated parameters, unitless |

## Calculation 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 post-CAAA scenario, for both the minimum and the maximum yield responses. Weights based on 1997 crop production data[9] were used to represent all years in this analysis (1990 to 2010). Because weather and other conditions may change the proportion of counties' production to the total national production in each year, weights based

[7] 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. Peanut emergence and harvest dates were taken from the U.S. EPA Pesticide Root Zone Model-2 (PRZM) data, US EPA 1993.

[8] 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.

[9] USDA 1998a.

on a single year may bias the estimates to some extent. The weights were calculated by dividing the production level of a crop in a county[10] by the sum of all states' reported production for that crop.[11] These county weights were applied to the percent relative yield loss results for each county, as discussed below, to develop a national level yield change estimate.

## Calculation of Percent Change in Yield

There is an issue associated with applying the yield loss functions to analysis of alternative regulatory profiles. The functions provide a predicted yield loss relative to "clean" air, while policy analysis needs to compare policy options with a baseline, non-zero ozone condition. Therefore, the yield change resulting from the Clean Air Act Amendments is evaluated as the yield loss relative to clean air under the CAAA scenario being evaluated compared to the yield loss under baseline (no-CAAA) conditions.

The change in yields, relative to "clean air" is calculated as:

$$PRYL_{Post-CAAA} - PRYL_{Pre-CAAA}$$

and, if yield under clean conditions is 100 percent of possible yield, then baseline yield in this context is 1 minus baseline yield loss. Thus the change in yield under clean air conditions can be divided by the baseline yield, and the change in yields relative to the baseline can be given as:

$$PRYL_{Post-CAAA} - PRYL_{Pre-CAAA} / 1 - PRYL_{Post-CAAA}$$

To create the national percent change in yield for each crop, the results of this equation are multiplied by the county level weights and summed for each scenario (maximum and minimum) and for each year. Table F-2 presents the resulting percent yield changes that were used as inputs to the economic model.

[10] USDA, 1995.

[11] 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 Percent Yield Change**

| | | Corn | Cotton | Peanuts | Sorgham | Soybeans | Winter Wheat |
|---|---|---|---|---|---|---|---|
| 2000 | Minimum Response | 0.01% | 1.66% | 0.61% | 0.01% | 0.26% | 0.20% |
| | Maximum Response | 0.05% | 3.79% | 0.61% | 0.01% | 2.75% | 5.07% |
| 2010 | Minimum Response | 0.01% | 2.84% | 1.36% | 0.02% | 0.42% | 0.39% |
| | Maximum Response | 0.10% | 6.58% | 1.36% | 0.02% | 4.38% | 9.11% |

## Economic Impact Estimates

To estimate the economic benefits of controls on ozone precursor pollutants implemented pursuant to the 1990 CAAA Amendments, we evaluated the changes in yields resulting from additional, post-1990 controls 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).

The modeling approach used in this analysis is to use 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 used extensively to evaluate air pollution impacts, as well as a number of other environmental policy analyses. The version of AGSIM© used in this analysis reflects production conditions and projections for three discrete periods: 1990, 2000, and 2010. Projections of the 2000 and 2010 baseline are essentially those reported by USDA/ERS (USDA 1998b). A few endogenous variables in AGSIM© were not included in the USDA baseline. In those cases, the 1997 Food and Agricultural Policy Research Institute (FAPRI) baseline was used (FAPRI 1997).[12]

The AGSIM© baseline production and price data serve as the post-CAAA scenario baseline. Percent relative yield losses (PRYLs) between the post-CAAA and pre-CAAA 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.

Table F-3 presents the net *changes* in economic surpluses in nominal terms for our two target years, 2000 and 2010. The positive net surpluses are a result of the increase in yields associated with lower ozone levels than those predicted to occur under the pre-CAAA scenario. The annual value of the estimated agricultural benefits of the CAAA in 2010 ranges between $7.5 million in the minimum response case to approximately $1.1 billion in the maximum response case, with a median response of $550 million. It

[12] Documentation for this version of AGSIM can be found in Abt Associates, 1998.

should be reiterated that 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 post-CAAA of ozone precursors under the CAAA, but not the precise value of those benefits.

**Table F-3**
**Change in Net Crop Income, Consumer Surplus and Net Surplus Under the Post-CAAA Scenario (millions of 1990$)**

| | Change in Net Crop Income | | Change in Consumer Surplus | | Change in Net Surplus | |
|---|---|---|---|---|---|---|
| | Minimum | Maximum | Minimum | Maximum | Minimum | Maximum |
| 1990 | $0 | $0 | $0 | $0 | $0 | $0 |
| 2000 | -$320 | -$1,901 | $367 | $2,763 | $46 | $862 |
| 2010 | -$736 | -$4,555 | $743 | $5,643 | $7.5 | $1,088 |

## Conclusions

Agricultural benefits associated with post-CAAA levels 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 for those crops included in the analysis. 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 may be biased toward higher ozone responsiveness. Therefore, we anticipate that cumulative net present value agricultural benefits from the Clean Air Act Amendments over the period 1990 to 2010 are on the order of $4 billion dollars.

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# Stratospheric Ozone Assessment

Appendix G

## Introduction

This appendix describes the assessment of costs and benefits of Title VI of the Clean Air Act Amendments (CAAA). Provisions under Title VI limit emissions of stratospheric ozone-depleting chemicals. Cost and benefit estimates of Title VI provisions are derived from a secondary analysis that updates key economic valuation components of previous analyses conducted by EPA's Office of Air and Radiation, Stratospheric Protection Division. We chose to perform a secondary analysis for several reasons: (1) prior analyses suggest that the benefits of these programs far exceed the costs, suggesting a potentially low value of information from re-estimating costs and benefits in new primary analyses; (2) prior analyses were extensively peer-reviewed; (3) the costs and benefits of these provisions are largely separable from provisions implemented under other titles of the Clean Air Act Amendments; and (4) developing new primary estimates would involve considerable time and expense. We therefore provide a new assessment of the valuation of benefits to ensure consistency with other portions of the prospective analysis, but have not re-assessed the Agency's previous estimates of stratospheric ozone depleter emissions, stratospheric ozone loss, changes in exposure to UV-b radiation, changes in physical effects, or the costs of Title VI provisions.

Unlike estimates for other Titles of the CAAA, we present estimates for Title VI as net present values of the streams of annual costs and benefits. The rationale for this type of presentation of costs and benefits relates to the long-term nature of the mechanisms of stratospheric ozone depletion and measures taken to avoid depletion. Stratospheric ozone is a global resource, and its formation and depletion are governed by long-term processes that may take place over decadal or longer time scales. Attempting to parse the incremental effects of an annual reduction in emissions of ozone depleting substances and estimate its impact at the unit emissions level is an extremely difficult, if not impossible, task and was not attempted for this exercise.

For the same reasons, We conduct a longer time-scale of analysis than is used in the remainder of the study. We estimate cost over the period 1990 through 2075, and benefits are estimated over the period 1990 to 2175. The difference in time scales for costs and benefits reflects the persistence of ozone depleting substances in the atmosphere, the slow processes of ozone formation and depletion, and lags in the manifestation of physical effects in response to exposure to elevated levels of UV-b radiation. The full benefits of emissions reductions achieved during 1990 to 2075 accrue across many decades and several generations, requiring an extended time scale for benefits analysis.

In the next section of the appendix, we provide a brief history of Title VI and its amendments. Next we summarize the general approaches used to estimate the costs and benefits in previous analyses, and we describe our strategy for modifying several analytical parameters to ensure consistency with the assessments of other titles of the Clean Air Act. Finally, we present the adjusted cost and benefits from 1990 to 2165 and discuss the uncertainty associated with the analyses.

## History of Stratospheric Ozone Protection and the CAAA

The protection of stratospheric ozone is an international effort, resulting in several multinational agreements. The United States has both participated in the development of these international

agreements and has created its own reduction and phaseout schedules for ozone-depleting substances used within its borders. These reduction and phaseout schedules, codified under Title VI of the Clean Air Act Amendments, are often stricter than the international agreements that preceded them. Below we briefly describe the history of the international agreements and their relationship to the Clean Air Act Amendments.

The United States, the European Economic Community, and 23 other countries signed the "Montreal Protocol on Substances that Deplete the Ozone Layer" (1987 Montreal Protocol) on September 16, 1987. Thirty-four countries then ratified this protocol. The agreement prohibits the use of chlorofluorocarbons (CFCs) beyond 1986 usage levels starting in mid-1989 and establishes a schedule for reducing the production of CFCs (i.e., a 20 percent reduction in CFC production in 1993 and a 50 percent reduction in 1998). The protocol also forbids the production of halons beyond 1986 production levels starting in 1992. On August 12, 1988, the U.S. Environmental Protection Agency (EPA) published final regulations to protect stratospheric ozone and comply with the requirements of the 1987 Montreal Protocol.[1]

After ratification of the Montreal Protocol scientists determined that the loss of stratospheric ozone was greater than they had originally thought and that man-made compounds containing bromine and chlorine were responsible for this loss. Consequently, in June 1990 the countries that had signed the Montreal Protocol met in London to develop an accelerated CFC reduction schedule (i.e., a decrease in CFC production to 50 percent of 1986 production levels by 1995 and 15 percent of 1986 levels by 1997). According to this London Agreement, production of CFCs, halons, and carbon tetrachloride will cease by 2000 and methyl chloroform (MCF) production would end by 2005.[2]

In November 1990 President George Bush signed the Clean Air Act Amendments (CAAA), which include Title VI. This title consists of six major sections, of which the most important are sections 604 and 606. Section 604 accelerates the London Agreement's reduction schedules for CFCs, halons, and carbon tetrachloride and shortens the time allowed for methyl chloroform phaseout by three years. Section 606 allows Congress to accelerate the reduction and phaseout schedules of section 604 if necessary to protect human health and the environment. Together, sections 604 and 606 generate nearly all of the costs and benefits of Title VI. The remaining sections include the following: section 608 (which requires the EPA to establish standards regarding the use and disposal of ozone-depleting substances during the service, repair, or disposal of appliances and industrial refrigerators); section 609 (which requires the EPA to regulate the servicing of motor vehicle air conditioners); and section 611 (which stipulates that the EPA must establish labeling requirements for containers of ozone-depleting substances and for products containing these substances). This analysis does not examine the costs and benefits of section 605, which institutes the reduction and phaseout schedules for hydrochlorofluorocarbons (HCFCs), because the schedules of section 606 supersede section 605's timetables. Table G-1 provides a description of the principal sections of the CAAA's Title VI.

[1] ICF, Incorporated, *Regulatory Impact Analysis: Compliance with Section 604 of the Clean Air Act for the Phaseout of Ozone Depleting Chemicals,* Prepared for Global Change Division, Office of Air and Radiation, U.S. Environmental Protection Agency, Washington, D.C., July 1, 1992, page ES-1.

[2] Ibid, ES-1 and ES-2.

**Table G-1**
**Six Major Sections of Title VI**

| Section | Description |
|---|---|
| Section 604 - Class I Phaseout | Institutes the reduction and phaseout schedules for Class I substances (methyl chloroform, halons, chlorofluorocarbons, carbon tetrachloride). |
| Section 605 - Class II Phaseout | Institutes the reduction and phaseout schedules for Class II substances (hydrochlorofluorocarbons). |
| Section 606 - Accelerated Schedule | Permits Congress to accelerate the phaseout schedule of Class I and II substances if necessary for the protection of human health and the environment.<br><br>Also, if countries modify the Montreal Protocol to accelerate phaseout schedules of Class I and II chemicals, Congress can amend the Clean Air Act to reflect these modifications. |
| Section 608 - National Recycling and Emission Reduction Program | Requires the EPA to establish standards regarding the use and disposal of Class I and II substances during the service, repair, or disposal of appliances and industrial refrigeration units. |
| Section 609 - Servicing of Motor Vehicle Air Conditioners | Requires the EPA to regulate the servicing of motor vehicle air conditioners. |
| Section 611 - Labeling | Stipulates that the EPA establish labeling requirements for containers of Class I and II substances and for products containing these substances. |

In November 1992 the parties to the Montreal Protocol met in Copenhagen to establish an agreement that incorporates new scientific information on stratospheric ozone depletion.[3] For example, data from the National Aeronautics and Space Administration (NASA) indicated that ozone depletion was progressing more rapidly than expected. In addition, ozone depletion was extending further south in the United States than anticipated and lasting longer (late fall to late May).[4] In response to new data, the parties to the Montreal Protocol agreed to several changes in the reduction and phaseout schedules of ozone-depleting chemicals. First, they agreed to a 1999 phaseout deadline for hydrobromofluorocarbons (HBFCs), chemicals not regulated under the earlier London Agreement. Second, they called for a freeze on production of methyl bromide by stipulating that the chemical should not exceed 1991 levels starting in 1995.[5] Third, these countries decided to accelerate the reduction schedule for the production and consumption of hydrochlorofluorocarbons (HCFCs). Lastly, they hastened the phaseout of halons by agreeing to 1994 as the production and consumption phaseout deadline.[6]

[3] U.S. Environmental Protection Agency, http://www.epa.gov/ttn/oarpg/t6/fact_sheets/66.txt, March 25, 1998.

[4] Ibid, ES-3 and ES-4.

[5] U.S. Environmental Protection Agency, http://www.epa.gov/spdpublc/mbr/harmoniz.html, March 26, 1999.

[6] U.S. Environmental Protection Agency, http://www.epa.gov/ttn/oarpg/t6/fact_sheets/66.txt, March 25, 1998.

Under the Clean Air Act Amendments' section 606, the EPA also responded to the new scientific data by periodically accelerating the reduction schedules for MCF, CFCs, halons, carbon tetrachloride, and HCFs and by establishing earlier phaseout deadlines. In addition, the EPA targeted HBFCs and methyl bromide, chemicals not explicitly addressed by the 1990 Clean Air Act Amendments.

The most recent changes established under section 606 involve methyl bromide. In 1993 the EPA called for a freeze on production at 1991 levels starting in 1994 and established a phaseout deadline of 2001. In 1995 and 1997 the parties to the Montreal Protocol met in Vienna and Montreal, respectively, to address issues such as the phaseout of methyl bromide. In 1998 Congress directed the EPA to match the 1997 Montreal Amendment's reduction and phaseout schedule for this chemical. In 1998, Congress amended the Clean Air Act to establish a new methyl bromide reduction schedule, which establishes 2005 as the phaseout deadline, and helps to address the needs of American farmers who are currently waiting for methyl bromide alternatives that are in the research and development stage.[7] Table G-2 presents the most significant sections of the new and old phaseout schedules.

[7] U.S. Environmental Protection Agency, http://www.epa.gov/spdpublc/mbr/harmoniz.html, November 9, 1999.

**Table G-2**
**Phaseout Scenario in Clean Air Act Section 604 and**
**Phaseout Scenario in Amendments Added under Clean Air Act Section 606**

| Year | Section 604 (Original Schedule) | | Section 606 (Revisions to Original Schedule) |
|---|---|---|---|
| 1991 | • Methyl chloroform (MCF) production decreases to 1989 levels | | |
| 1993 | • MCF production decreases 10% from 1989 levels | | |
| 1994 | • MCF production decreases 15% from 1989 levels | ➜ | • MCF production decreases from 1989 levels by 50% |
| 1995 | • MCF production decreases 30% from 1989 levels | ➜ | • MCF production decreases from 1989 levels by 70% |
| 1996 | • MCF production decreases 50% from 1989 levels | ➜ | • 100% phaseout of MCF |
| 2000 | • MCF production decreases 80% from 1989 levels | | |
| 2002 | • 100% phaseout of MCF | | |
| 1989 | • Chlorofluorocarbon (CFC) production decreases to 1986 levels | | |
| 1991 | • CFC production decreases 15% from 1986 levels | | |
| 1992 | • CFC production decreases 20% from 1986 levels | | |
| 1993 | • CFC production decreases 25% from 1986 levels | | |
| 1994 | • CFC production decreases 35% from 1986 levels | ➜ | • CFC production decreases to 75% from 1986 levels |
| 1995 | • CFC production decreases 50% from 1986 levels | ➜ | • CFC production decreases to 75% from 1986 levels |
| 1996 | • CFC production decreases 60% from 1986 levels | ➜ | • 100% phaseout of CFC production |
| 1997 | • CFC production decreases 85% from 1986 levels | | |
| 2000 | • 100% phaseout of CFC production | | |

| Year | Section 604 (Original Schedule) | | Section 606 (Revisions to Original Schedule) |
|---|---|---|---|
| 1991 | • Halon production decreases 15% from 1986 levels | | |
| 1992 | • Halon production decreases 20% from 1986 levels | | |
| 1993 | • Halon production decreases 25% from 1986 levels | | |
| 1994 | • Halon production decreases 35% from 1986 levels | ➜ | • 100% phaseout of halons |
| 1995 | • Halon production decreases 50% from 1986 levels | | |
| 1996 | • Halon production decreases 60% from 1986 levels | | |
| 1997 | • Halon production decreases 85% from 1986 levels | | |
| 2000 | • 100% phaseout of halon production | | |
| 1991 | • Carbon tetrachloride production decreases to 1989 levels | | |
| 1992 | • Carbon tetrachloride production decreases 10% from 1989 levels | | |
| 1993 | • Carbon tetrachloride production decreases 20% from 1989 levels | | |
| 1994 | • Carbon tetrachloride production decreases 30% from 1989 levels | ➜ | • Carbon tetrachloride production decreases 50% from 1989 levels |
| 1995 | • Carbon tetrachloride production decreases 85% from 1989 levels | ➜ | • Carbon tetrachloride production decreases 85% from 1989 levels |
| 1996 | | ➜ | • Carbon tetrachloride production decreases 100% phaseout of carbon tetrachloride |
| 2000 | • 100% phaseout of carbon tetrachloride | | |

| Year | Section 604 (Original Schedule) | | Section 606 (Revisions to Original Schedule) |
|---|---|---|---|
| 1994 | | | • Freeze on production and consumption of methyl bromide at 1991 levels |
| 1999 | | | • 25% phaseout of methyl bromide |
| 2001 | | | • 50% phaseout of methyl bromide |
| 2003 | | | • 75% phaseout of methyl bromide |
| 2005 | | | • 100% phaseout of methyl bromide<br>(quarantine and preshipment uses exempt; critical agriculture uses allocated after 2005) |
| 1996 | | | • 100% phaseout of hydrobromofluorocarbons (HBFCs) |
| 2003 | | ➜ | • Production and consumption of HCFC-141b banned |
| 2010 | | ➜ | • Production and consumption of HCFC-142b and HCFC-22 decreases to 1989 levels |
| (2010-2020) | | ➜ | • Production and consumption of HCFC-142b and HCFC-22 permissible only for servicing equipment manufactured prior to 2010 |
| 2015 | • Freeze on HCFC production | ➜ | • Production and consumption of the remaining HCFCs decreases to 1989 levels |
| 2020 | | ➜ | • 100% phasout of HCFC-142b and HCFC-22 |
| 2030 | • Prohibition of HCFC production after January 1, 2030 | ➜ | • 100% phaseout of the final category of HCFCs |

## Cost and Benefit Approaches

To estimate the costs and benefits of Title VI, we rely primarily on past EPA regulatory impact assessments (RIAs), including the following:

- *Addendum to the 1992 Phaseout Regulatory Impact Analysis: Accelerating the Phaseout of CFCs, Halons, Methyl Chloroform, Carbon Tetrachloride, and HCFCs* (1993);
- *Regulatory Impact Analysis: Compliance with Section 604 of the Clean Air Act for the Phaseout of Ozone Depleting Chemicals* (1992);
- *Regulatory Impact Analysis: The National Recycling and Emission Reduction Program (Section 608 of the Clean Air Amendments of 1990)* (1993);
- *Section 609 of the 1990 Clean Air Act: Refrigerant Recycling for Mobile Air Conditioners: Cost-Benefit Analysis and Regulatory Flexibility Analysis* (1991);
- *Draft: Regulatory Impact Analysis of the Proposed Rule Requiring Labeling of Products Containing or Manufactured with Ozone Depleting Substances* (1991).

The major difference between this analysis and the RIA analyses involves the parameters used to value the costs and benefits. To ensure consistency with the larger Section 812 analysis, we adjust the discount rate in the costs calculations, and we adjust the value of statistical life, GDP per capita growth rates, and the discount rate in the benefits calculations.

### *Cost Approach in RIAs*

Existing regulatory impact assessments (RIAs) for individual provisions of Title VI provide the basis for the social costs of phasing out CFCs, halons, methyl chloroform, and HCFCs.[8] These social costs are the additional quantities of resources necessary to produce equivalent quantities of goods and services for consumers. To generate social cost estimates, the RIAs calculate the costs of replacing ozone-depleting chemicals (ODSs) with alternative technologies and materials, as well as the costs of recycling and storing unused ODSs. The estimates also include costs of training, labeling, and administration. The total cost estimate of Title VI comprises the costs of sections 604 and 606 and the incremental costs of the remaining sections (i.e., the cost estimates for sections 608, 609, and 611 do not include the costs of actions already required by sections 604 and 606). Table G-3 indicates the major costs associated with each section. We present summaries of the cost methodologies in this appendix; for more details, see the RIAs of the individual provisions of Title VI.

[8] Only a small percentage of carbon tetrachloride (less than three percent of the total produced in the U.S.) is subject to the CAAA, and the costs of phasing out carbon tetrachloride are likely insignificant compared to the costs of phasing out CFCs, halons, and MCFs. Consequently, the RIAs do not quantify the costs of phasing out carbon tetrachloride. In addition, this analysis does not assess the cost of methyl bromide because *Part 2: The Cost and Cost-Effectiveness of the Proposed Phaseout of Methyl Bromide* does not provide the corresponding benefits.

**Table G-3**
**Scope of Title VI Cost Estimates**

| Section | Costs |
|---|---|
| 604 & 606 | • Capital investment and variable costs associated with switching to alternative technologies and non-ozone-depleting substances;<br>• Possible elimination of products containing ozone-depleting substances (ODSs) if firms are unable to develop cost-effective manufacturing alternatives;<br>• Costs of recycling ODSs;<br>• Additional costs of switching from methyl bromide to other substances (e.g. purchases of more costly substitutes, incremental labor needs, and crop and throughput issues). |
| 608 | • Purchase of ODS recovery equipment;<br>• Training and certification of technicians;<br>• Filtration of refrigerants to remove impurities;<br>• Leak repair requirements;<br>• Storage of unused chlorofluorocarbons (CFCs);<br>• Paperwork. |
| 609 | • Training and certification of mobile air conditioner (MAC) service technicians;<br>• Recycling costs, including fees for off-site recycling or labor and capital costs for on-site recycling. |
| 611 | • Development and application of new labels;<br>• Administrative activities associated with compliance;<br>• Possible accelerated substitution of less harmful substances relative to the mandates of the rule codifying the phaseout of ODSs (thereby resulting in additional costs beyond those resulting from the phaseout rule). |

## Costs: Sections 604 and 606

To generate the costs of section 604, the *Addendum to the 1992 Phaseout Regulatory Impact Analysis: Accelerating the Phaseout of CFCs, Halons, Methyl Chloroform, Carbon Tetrachloride, and HCFCs* (1993) and the *Regulatory Impact Analysis: Compliance with Section 604 of the Clean Air Act for the Phaseout of Ozone Depleting Chemicals* (1992) use engineering analyses that involve several steps. First, the RIAs identify potential technical responses to section 604 from 1990 to 2075. For example, they examine data on the CFC reduction technologies available to each CFC-using industry (i.e., mobile air conditioners, household refrigeration, foam blowing, solvent cleaning, sterilization, rigid polyurethane foams, chillers, and process-12 refineries).[9] For halon the RIAs analyze 74 categories of fire extinguishing applications, the primary users of the substance.[10] With these data, the RIAs identify three potential technical responses: use of chemical substitutes for CFC and halon use in new and existing equipment, implementation of engineering controls that reduce use of ODSs through recycling, and use of product substitutes for ODS-containing products.

Second, the RIAs assess the costs of the responses. For halons and CFCs the RIAs examine the date at which an action is first available for adoption, the time necessary for the entire industry to evaluate the action, and the time required for

[9] ICF, Incorporated, *Addendum to the 1992 Phaseout Regulatory Impact Analysis: Accelerating the Phaseout of CFCs, Halons, Methyl Chloroform, Carbon Tetrachloride, and HCFCs*, Prepared for the Stratospheric Protection Division, Office of Air and Radiation, U.S. Environmental Protection Agency, September 10, 1993, page 6.

[10] ICF, Incorporated, *Regulatory Impact Analysis: Compliance with Section 604 of the Clean Air Act for the Phaseout of Ozone Depleting Chemicals,* Prepared for Global Change Division, Office of Air and Radiation, U.S. Environmental Protection Agency, July 1, 1992; page 4-7.

firms to adopt the action if it is cost-effective. They also study reduction potential (decrease in ODS use for all firms that have decided to take the action) and applicability to new and/or existing equipment. This CFC and halon cost approach is similar to the approach used in the U.S. Environmental Protection Agency's *Regulatory Impact Analysis: Protection of Stratospheric Ozone* (1988); the main difference is the expansion of the methods used to model the lifetimes of equipment using CFC and halons.[11] By contrast, for MCFs the RIAs do not focus on the lifetime of the equipment because firms can retrofit MCF-consuming end uses with MCF alternatives.[12] Using the relevant data for MCFs, halons, and CFCs, the RIAs estimate feasible schedules for implementation of reduction measures and estimate the following costs:

- Variable costs (e.g., materials, energy, labor, and operating expenses);
- Capital costs;
- One-time fixed costs (e.g., research and development or training); and
- Changes in energy efficiency.

For the last step of the cost analyses, the model selects technologies that minimize production cost increases and achieve the necessary ODS reductions. The final set of control plans must satisfy the following requirements: 1) the plans contain components that industry has already implemented or intends to implement in the near future; 2) they jointly ensure that the amount of CFC production over time does not exceed the maximum stipulated by the phaseout schedule; and 3) they collectively prevent CFC use after the phaseout deadline from exceeding feasible recycling.

Under section 606 the EPA accelerates the reduction and phaseout schedules of ODSs; for these substances the cost methodology is the same as the section 604 methodology for CFCs, halons, methyl chloroform, and carbon tetrachloride. For HCFCs the EPA calculates the costs of phaseout by multiplying the quantity of replaced HCFCs by the difference in price between the HCFC compound and its substitute. This analysis assumes that the replacement compounds will cost between 10 and 50 percent more than the HCFCs. Together, the costs of sections 604 and 606 are $55.9 billion (1990 dollars) with a two percent discount rate; these costs comprise nearly all of the costs of Title VI.

## Costs: Sections 608

For section 608, the *Regulatory Impact Analysis: The National Recycling and Emission Reduction Program (Section 608 of the Clean Air Amendments of 1990)* (1993) uses the section 604 cost model to forecast the timing of emissions controls, resulting prices, and recycling levels from 1994 to 2015. In particular, the model assumes that recovery efficiency is 95 percent and predicts that all users of chillers, industrial processes, cold storage, retail food, and refrigerated transport will either recover

[11] Specifically, the cost model improves upon the 1988 model in the following ways: tracking the size, age, and turnover of CFC-consuming equipment over time; simulating the lifecycle of the equipment in terms of manufacturing, operation, servicing, and disposal; estimating CFC use, CFC emissions, energy use, and costs for each point in the lifecycle; calculating CFCs potentially available through recycling from older equipment; and simulating the impact of CFC reduction measures by assessing the degree of emissions reductions, total costs, and total changes in energy use associated with implementing groups of controls over time for each end use. (ICF Incorporated, *Regulatory Impact Analysis: Compliance with Section 604 of the Clean Air Act for the Phaseout of Ozone-Depleting Chemicals*, Prepared for the Global Change Division, Office of Air and Radiation, U.S. Environmental Protection Agency, 1992, pages 4-2, 4-8, 4-9, and 4-16.)

[12] The EPA's *Costs and Benefits of Phasing Out Production of CFCs and Halons in the United States* (1989) and the EPA's *Regulatory Impact Analysis: Protection of Stratospheric Ozone* (1988) provide the basis of the cost methodology for the MCF phaseout. Note that the MCF model calculates energy costs as a part of operating costs, rather than as a separate component, because MCF end uses are not energy intensive. (ICF Incorporated, *Regulatory Impact Analysis: Compliance with Section 604 of the Clean Air Act for the Phaseout of Ozone-Depleting Chemicals*, Prepared for the Global Change Division, Office of Air and Radiation, U.S. Environmental Protection Agency, 1992, pages ES-8, 4-27.)

or recycle CFCs at service and disposal and that after the phaseout all household appliance users will recover CFCs at disposal.[13] With a two percent discount rate the cost estimate is $1.2 billion (1990 dollars).

## Costs: Section 609

Section 604 prompts service establishments to recycle CFC-12 in mobile air conditioners (MACs). Therefore, the section 604 cost estimate accounts for most costs associated with the recycling of CFC-12 from MACs. The costs of section 609 that are not included in the section 604 cost estimate are the costs of training and certifying MAC service technicians. The analysis of the social costs of Section 609 examines these costs from 1992 to 2008 under two scenarios: a lower bound scenario in which small and large shops recover CFC-12 and pay an off-site recycler to purify and return the used refrigerant and an upper bound scenario in which all shops recycle CFC-12 on-site.[14] The central cost estimate for section 609 with a two percent discount rate is $14.3 million (1990 dollars).

## Costs: Section 611

For section 611, the *Draft: Regulatory Impact Analysis of the Proposed Rule Requiring Labeling of Products Containing or Manufactured with Ozone Depleting Substances* (1991) evaluates two response options: companies label all products or they reformulate/redesign the products to eliminate the use of Class I ozone-depleting substances (fully halogenated CFCs, three halons, methyl chloroform, and carbon tetrachloride).[15] The RIA then assesses three associated costs: costs of implementing substitutes (for MCF-containing products) more rapidly than predicted under the phaseout schedule, administrative activity costs, and costs of labeling.[16] From 1994 to 2000 the costs of section 611 are $252 million with a two percent discount rate.

## *Benefits Approach in RIAs*

The RIAs' Title VI benefits analyses necessarily differ from the benefits analyses for other parts of our CAAA-analysis because, unlike most of the effects of criteria air pollutants, the effects of Title VI are global in scale and occur over several hundred years. The delay in effects occurs for several reasons. First, emissions often emanate from products that leak the ozone-depleting chemicals over a significant period of time. Second, ozone-depleting chemicals rise into the stratosphere and affect the ozone layer at a slow rate. Third, ozone-depleting substances can persist in the stratosphere for many years. Fourth, natural processes that replace stratospheric ozone are slow. To reflect the long time period during which stratospheric ozone depletion occurs, this analysis assumes that the benefits accrue from 1990 to 2165.

Figure G-1 is a simplified illustration of the relationships between the sets of data used in the existing benefits analyses. First, the EPA estimates change in ozone-depleting substance emissions. With these data the EPA calculates the extent of stratospheric ozone depletion and global warming. Then the EPA calculates the effects of stratospheric ozone on UV-b radiation, which in turn affects

[13] ICF, Incorporated, *Regulatory Impact Analysis: The National Recycling and Emission Reduction Program (Section 608 of the Clean Air Amendments of 1990)*, Prepared for the Stratospheric Protection Division, U.S. Environmental Protection Agency, March 25, 1993, page 4-2.

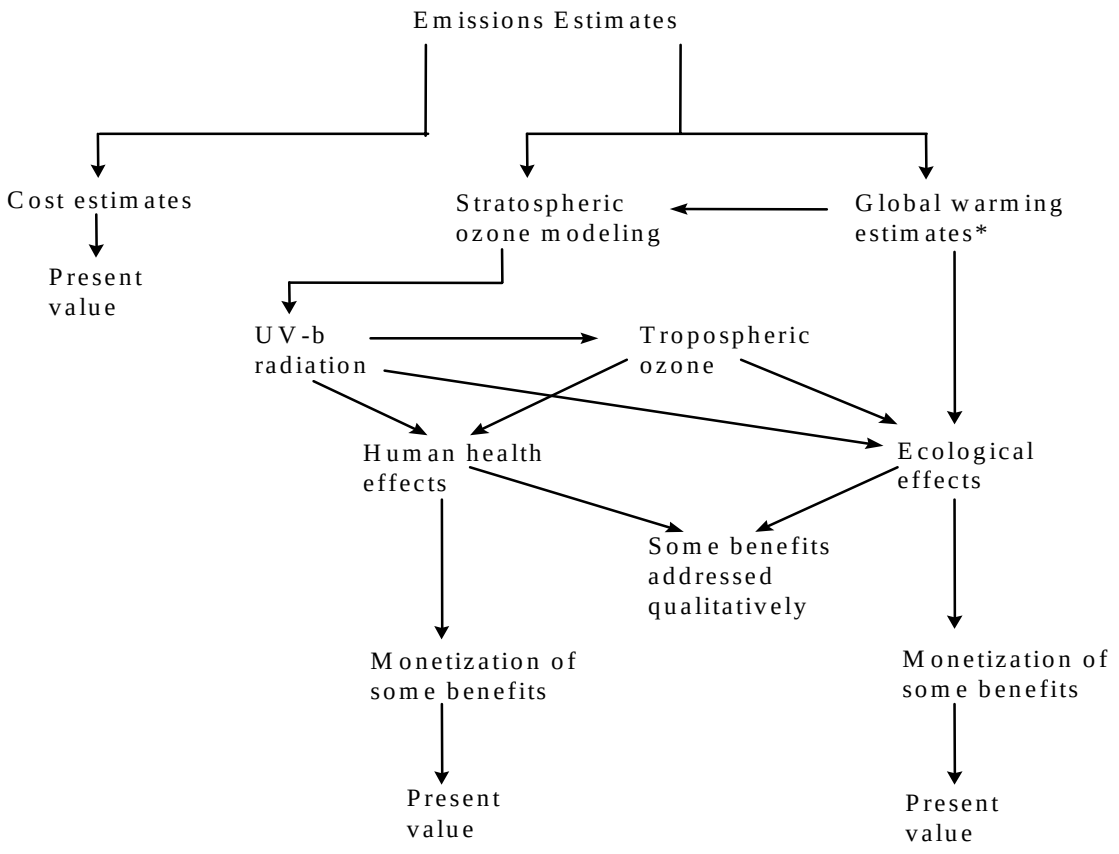
[14] ICF, Incorporated, *Section 609 of the 1990 Clean Air Act: Refrigerant Recycling for Mobile Air Conditioners: Cost-Benefit Analysis and Regulatory Flexibility Analysis*, Prepared for the Division of Global Change, U.S. Environmental Protection Agency, May 24, 1991, pages 4 and 8.

[15] The costs analysis does not account for the accelerated reduction and phaseout schedule of section 606. ICF, Incorporated, *Draft: Regulatory Impact Analysis of the Proposed Rule Requiring Labeling of Products Containing or Manufactured with Ozone Depleting Substances*, Prepared for Global Change Division, Office of Air and Radiation, U.S. Environmental Protection Agency, November 1991, pages 1, 4, and 6.

[16] *Ibid,* page 37.

**Figure G-1**

**SCHEMATIC OF COST AND BENEFIT ANALYSES OF TITLE VI**




* Several ozone-depleting substances are also greenhouse gases with high radiative forcing potential relative to carbon dioxide. We do not assess the impact of global warming in this analysis.

estimates of tropospheric ozone.[17] Using UV-b radiation, tropospheric ozone, and global warming data as inputs, the EPA estimates human health and environmental effects.[18] Lastly, the EPA monetizes the benefits of improved human and environmental health where possible.[19] In this analysis, our assessment of benefits is slightly different from that of the previous RIAs because we attribute benefits to effects of reduced stratospheric ozone and not to global warming. We present the benefits estimate as a net present value, rather than an annualized value, because annualization incorrectly imputes benefits of later phaseouts to earlier years. For example, annualization of the benefits of phasing out HCFC-22 by 2020 attributes benefits to years prior to 2030, when neither the costs nor the benefits of that phaseout have yet occurred. Consequently, an annualized estimate will overstate benefits at the beginning of the time span and understate them later.

### Emissions Modeling

The methodology for predicting global use of ozone-depleting chemicals and the resulting emissions is similar to the methodology used in *Regulatory Impact Analysis: Compliance with Section 604 of the Clean Air Act for the Phaseout of Ozone-Depleting Chemicals* (1992). The main difference in the baseline scenario, which assumes no Title VI controls, involves methyl bromide. In this analysis we assume the following as a baseline: 1) in 1990 facilities worldwide produce 63 million kilograms of methyl bromide and facilities in the U.S. produce 29.1 million kilograms; 2) methyl bromide production grows at 5.5 percent annually until 2025 and zero percent thereafter; 3) 50 percent of methyl bromide production generates emissions; 4) humans generate about 25 percent of total methyl bromide emissions[20]; and 5) bromine is 40 times as effective as chlorine at destroying ozone. Also, based on NASA's new data regarding the extent of ozone depletion in the Northern Hemisphere, the model assumes that the weighted average ozone depletion was 3.38 percent in 1989 relative to 1979 . [21]

The control scenario used in this prospective analysis is based on the "CAA phaseout scenario" established in the 1992 RIA and the "President's schedule" outlined in *Addendum to the 1992 Phaseout Regulatory Impact Analysis: Accelerating the Phaseout of CFC's, Halons, Methyl Chloroform, Carbon Tetrachloride, and HCFC's* (1993). The phaseout schedule, presented in Table G-2, summarizes the emission reductions incorporated in this study's control scenario. The emissions model forecasts global use and emissions of CFCs, MCFs, carbon tetrachloride, HCFCs, and halons under the control scenario in two major steps.[22] For sections

[17] The relationships between stratospheric ozone depletion and global warming and between stratospheric ozone depletion and tropospheric ozone are incompletely understood at this time. The *Regulatory Impact Analysis: Compliance with Section 604 of the Clean Air Act for the Phaseout of Ozone Depleting Chemicals* (1992) and the *Addendum to the 1992 Phaseout Regulatory Impact Analysis: Accelerating the Phaseout of CFCs, Halons, Methyl Chloroform, Carbon Tetrachloride, and HCFCs* (1993) base their tropospheric ozone estimates on the "Effects of Increased UV Radiation on Urban Ozone" (Whitten and Gery, 1986).

[18] Note that the *Regulatory Impact Analysis: Compliance with Section 604 of the Clean Air Act for the Phaseout of Ozone Depleting Chemicals* (1992) and the *Addendum to the 1992 Phaseout Regulatory Impact Analysis: Accelerating the Phaseout of CFCs, Halons, Methyl Chloroform, Carbon Tetrachloride, and HCFCs* (1993) do not provide data on the models used to estimate UV-b radiation.

[19] The benefits estimates used in the RIAs' benefit/cost comparison sections do not reflect economic impacts (e.g., profit increases that occur if alternative technologies are more efficient than ODS-using technologies).

[20] Sources of methyl bromide include anthropogenic and natural sources. Natural sources include the ocean, plants, and soil.

[21] Stolarski, Watson, Testimony to the Senate Commerce, Science, and Transportation Subcommittee on Science, Technology, and Space, April 16, 1991; ICF, Incorporated, *Addendum to the 1992 Phaseout Regulatory Impact Analysis: Accelerating the Phaseout of CFCs, Halons, Methyl Chloroform, Carbon Tetrachloride, and HCFCs*, Prepared for the Stratospheric Protection Division, Office of Air and Radiation, U.S. Environmental Protection Agency, September 10, 1993, page 9.

[22] See ICF, Incorporated, *Addendum to the 1992 Phaseout Regulatory Impact Analysis: Accelerating the Phaseout of CFCs, Halons, Methyl Chloroform, Carbon Tetrachloride, and HCFCs*, Prepared for

reflecting the accelerated reduction and phaseout schedule, the model first applies the chemical demand growth rates developed for the *Regulatory Impact Analysis: Protection of Stratospheric Ozone* (1988), with the assumption that all nations will comply with the Copenhagen Amendments. For the other sections (i.e., section 611, section 609, and the ecological components of sections 604 and 606), the model assumes that all nations comply with the Montreal Protocol and the 1990 London Agreements.[23] Second, the model applies these growth rates to the release rates for the chemicals.[24] This calculation generates the total emissions estimate for each chemical from 1985 to 2165.[25]

## Stratospheric Ozone Depletion Modeling and Global Warming

Using the total emissions estimates of ODSs, the atmospheric lifetimes of the chemicals, and conversion factors, the EPA calculates stratospheric chlorine and bromine concentrations. Lifetimes indicate the length of time that the chlorine and bromine associated with a specific chemical will likely remain in the atmosphere. Conversion factors relate the emissions to stratospheric ozone changes.[26] To translate changes in stratospheric chlorine and bromine concentrations to changes in total column ozone, the EPA modifies Connell's parameterized version of a one-dimensional atmospheric chemistry model.[27] The EPA calibrates this model to incorporate the effects of atmospheric processes (e.g., heterogeneous chemical reactions) by applying an adjustment factor to the stratospheric ozone content; this calibration ensures that the model's global results are consistent with historical ozone trends for northern hemisphere middle and high latitudes.[28] The EPA's model assumes that increases in stratospheric chlorine are the primary causes of the observed ozone change and that the annual average change in UV-b predicted by the modeling framework equals the annual average UV-b change inferred from observed ozone trends and radiation models.

In conjunction with the calibrated version of Connell's parameterized model, a second model incorporates the ability of CFCs, halons, MCF, carbon tetrachloride, and HCFCs to act as greenhouse gases (substances that contribute to the warming of the earth's atmosphere by absorbing infrared radiation emitted from the earth's surface).[29] This second model, adapted from the

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the Stratospheric Protection Division, Office of Air and Radiation, U.S. Environmental Protection Agency, September 10, 1993, page 9.

[23] Ibid, ICF (1993a), 9; Ibid ICF (1992), 3-6 and 3-12.

[24] Ibid, ICF (1993a), 1; Ibid ICF (1992), 3-12.

[25] ICF Incorporated's *Addendum to the 1992 Phaseout Regulatory Impact Analysis: Accelerating the Phaseout of CFCs, Halons, Methyl Chloroform, Carbon Tetrachloride, and HCFCs* (1993a) does not present emissions estimates.

[26] The sources of the ozone depleting potential estimates include Fisher *et al.* (1990a) and the 1987 Montreal Protocol. (ICF Incorporated, *Regulatory Impact Analysis: Compliance with Section 604 of the Clean Air Act for the Phaseout of Ozone-Depleting Chemicals*, Prepared for the Global Change Division, Office of Air and Radiation, U.S. Environmental Protection Agency, 1992, page 3-16.)

[27] Connell, Peter, "A Parameterized Numerical Fit to Total Column Ozone Changes Calculated by the LLNL 1-D Model of the Troposphere and Stratosphere," Lawrence Livermore National Laboratory, Livermore, CA, 1986. (For a description of the stratospheric ozone model, see U.S. Environmental Protection Agency, *Assessing the Risks of Trace Gases that Can Modify the Stratosphere*, 1987; U.S. Environmental Protection Agency, *Future Concentrations of Stratospheric Chlorine and Bromine*, EPA 400/1-88-005, August 1988; and U.S. Environmental Protection Agency, *Regulatory Impact Analysis: Protection of Stratospheric Ozone*, August 1, 1988.)

[28] Rodriguez, J.M., M.K.W. Ko, and N.D. Sze, " Antarctic Chlorine Chemistry: Possible Global Implications," *Geophysical Research Letters, 15*, 1988, pages 257-260.

[29] CFC substitutes may indirectly influence global warming by affecting the energy efficiency of CFC-using capital stock (e.g., insulating foam and refrigerators). As chemicals that are more or less energy efficient replace CFCs, total energy demand could diminish or increase, causing changes in the emissions of energy-related greenhouse gases. (ICF Incorporated, *Regulatory Impact Analysis: Compliance with Section 604 of the Clean Air Act for the Phaseout of Ozone-Depleting Chemicals*, Prepared for the Global

Goddard Institute of Space Sciences' climate model, uses estimates for emissions of controlled chemicals, substitute chemicals, and energy-related greenhouse gases to calculate changes in global temperature over time.[30] The model adjusts column ozone and temperature so that they are consistent with consensus ozone-depleting potential and global warming potential estimates.[31] The model also reflects 1) radiative and chemical feedback from water vapor, 2) ocean absorption, 3) atmospheric circulation effects, and 4) chemical interactions between substances.[32, 33]

Estimates from stratospheric ozone modeling may be under- or overestimates, depending on heterogeneous reactions in the aerosol layer, ozone depletion in the Arctic, the linearity of the atmospheric response, and other factors.[34] The EPA estimates that the accelerated reduction and phaseout schedules of section 604 and 606 will result in 7 percent less ozone depletion from baseline levels in 2005 and 47.01 percent less ozone depletion in 2075.[35]

## Physical Effects

For the physical effects that scientists have modeled with dose-response functions, we use data on UV-b radiation and tropospheric ozone to calculate benefits. We include benefits that scientists have identified but not yet quantified in a qualitative discussion. Below we present the benefits methodology for each section.

### Physical Effects: Sections 604 and 606

Table G-4 presents the quantified and unquantified physical effects estimates of sections 604 and 606, which generate about 98 percent of the benefits. The quantified benefits include the following: reduced incidences of mortality and morbidity associated with skin cancer (melanoma and nonmelanoma); reduced incidences of cataract morbidity and the associated pain and suffering; reduced crop damage associated with UV-b radiation and tropospheric ozone; and reduced polymer degradation from UV-b radiation.

Change Division, Office of Air and Radiation, U.S. Environmental Protection Agency, 1992, pages 3-17 and 3-18.)

[30] U.S. Environmental Protection Agency, *Regulatory Impact Analysis: Protection of Stratospheric Ozone*, August 1, 1988; ICF Incorporated, *Regulatory Impact Analysis: Compliance with Section 604 of the Clean Air Act for the Phaseout of Ozone-Depleting Chemicals*, Prepared for the Global Change Division, Office of Air and Radiation, U.S. Environmental Protection Agency, 1992, page 3-18.

[31] National Aeronautics and Space Administration Conference Publication 3023, *An Assessment Model for Atmospheric Composition*, 1988; ICF Incorporated, *Regulatory Impact Analysis: The National Recycling and Emission Reduction Program (Section 608 of the Clean Air Act Amendments of 1990)*, Prepared for the Stratospheric Protection Division, U.S. Environmental Protection Agency, 1993, page 5-2.

[32] Radiative forcing constants and lifetimes form the basis of the global warming potential estimates, calculated with an infinite time horizon (Lashof and Ahuja, 1990). Fisher *et al.* provide data on direct radiative forcing constants (Fisher *et al.*, 1990b).

[33] ICF Incorporated, *Regulatory Impact Analysis: The National Recycling and Emission Reduction Program (Section 608 of the Clean Air Act Amendments of 1990)*, Prepared for the Stratospheric Protection Division, U.S. Environmental Protection Agency, 1993, page 5-2; ICF, Incorporated, *Addendum to the 1992 Phaseout Regulatory Impact Analysis: Accelerating the Phaseout of CFCs, Halons, Methyl Chloroform, Carbon Tetrachloride, and HCFCs*, Prepared for the Stratospheric Protection Division, Office of Air and Radiation, U.S. Environmental Protection Agency, September 10, 1993, page 9.

[34] ICF Incorporated, *Regulatory Impact Analysis: Compliance with Section 604 of the Clean Air Act for the Phaseout of Ozone-Depleting Chemicals*, Prepared for the Global Change Division, Office of Air and Radiation, U.S. Environmental Protection Agency, 1992, page 5-8.

[35] ICF, Incorporated, *Addendum to the 1992 Phaseout Regulatory Impact Analysis: Accelerating the Phaseout of CFCs, Halons, Methyl Chloroform, Carbon Tetrachloride, and HCFCs*, Prepared for the Stratospheric Protection Division, Office of Air and Radiation, U.S. Environmental Protection Agency, September 10, 1993, page 14.

**Table G-4**
**Benefits of Section 604, 606, and 609**

| **Health Effects- Quantified** | **Estimate** | **Basis for Estimate** |
|---|---|---|
| • Melanoma and nonmelanoma skin cancer (fatal) | 6.3 million lives saved from skin cancer in the U.S. between 1990 and 2165 | Dose-response function based on UV exposure and demographics of exposed populations.[1] |
| • Melanoma and nonmelanoma skin cancer (non-fatal) | 299 million avoided cases of non-fatal skin cancers in the U.S. between 1990 and 2165 | Dose-response function based on UV exposure and demographics of exposed populations.[1] |
| • Cataracts | 27.5 million avoided cases in the U.S. between 1990 and 2165 | Dose-response function uses a multivariate logistic risk function based on demographic characteristics and medical history. [1] |
| **Ecological Effects- Quantified** | **Estimate** | **Basis for Estimate** |
| • American crop harvests | Avoided 7.5 percent decrease from UV-b radiation by 2075 | Dose-response sources: Teramura and Murali (1986), Rowe and Adams (1987) |
| • American crops | Avoided decrease from tropospheric ozone | Estimate of increase in tropospheric ozone: Whitten and Gery (1986). Dose-response source: Rowe and Adams (1987) |
| • Polymers | Avoided damage to materials from UV-b radiation | Source of UV-b/stabilizer relationship: Horst (1986) |
| **Health Effects- Unquantified** | | |
| Skin cancer: reduced pain and suffering | | |
| Reduced morbidity effects of increased UV. For example,<br>• reduced actinic keratosis (pre-cancerous lesions resulting from excessive sun exposure)<br>• reduced immune system suppression. | | |
| **Ecological Effects- Unquantified** | | |
| Ecological effects of UV. For example, benefits relating to the following:<br>• recreational fishing<br>• forests<br>• overall marine ecosystem<br>• avoided sea level rise, including avoided beach erosion, loss of coastal wetlands, salinity of estuaries and aquifers<br>• other crops<br>• other plant species<br>• fish harvests | | |
| Ecological benefits of reduced tropospheric ozone relating to the overall marine ecosystem, forests, man-made materials, crops, other plant species, and fish harvests | | |
| Benefits to people and the environment outside the U.S. | | |
| Effects, both ecological and human health, associated with global warming. | | |

Notes:
1) For more detail see EPA's *Regulatory Impact Analysis: Protection of Stratospheric Ozone* (1988).
2) Note that the ecological effects, unlike the health effects, do not reflect the accelerated reduction and phaseout schedule of section 606.
3) Benefits due to the section 606 methyl bromide phaseout are not included in the benefits total because the EPA provides neither annual incidence estimates nor a monetary value. The EPA does provide, however, a total estimate of 2,800 avoided skin cancer fatalities in the U.S.

Using the change in UV radiation exposure due to current and future ozone depletion, we estimate the number of additional cases of skin cancer (melanoma and non-melanoma) and cataracts. With the exception of non-melanoma mortality, which is estimated as a fixed percentage of non-melanoma incidence, we use dose-response functions to develop future incremental skin cancer estimates. We employ nearly identical approaches in developing the three dose-response functions for non-fatal non-melanoma (i.e., basal cell and squamous cell carcinoma), non-fatal melanoma, and fatal melanoma.

The first step uses results from studies that have identified key groups of wavelengths ("action spectra") within the UV spectrum that are associated with specific types of health effects (e.g., DNA damage).[36] Once the appropriate action spectrum for a health effect is determined, the next step involves estimating the amount of UV dose received at various latitudes across the U.S. in the years prior to ozone depletion. The third step involves obtaining nationwide skin cancer incidence and mortality data for each health effect.[37] These data are then combined with the estimated variation in UV doses across latitudes in a cross-sectional analysis of the relationship between skin cancer incidence or mortality and differences in UV exposure.[38] This statistical analysis uses an equation of the form: (fractional change in incidence) = (fractional change in UV dose + 1)$^b$ -1, where b (the biological amplification factor) equals the percent change in incidence associated with a one percent change in dose. The dose-response function for cataracts is developed similarly.[39]

The health benefits model uses these dose-response functions to project incremental cases of non-fatal non-melanoma, fatal and non-fatal melanoma, and cataracts that will occur due to future increases in UV exposure caused by stratospheric ozone depletion. In essence, future incremental health effects are estimated by multiplying the baseline level of each health effect by the percentage change in UV exposure for different latitudes in the U.S. times the appropriate dose-response factor. Because the baseline levels of all of these UV-related health effects tend to be higher for older people and for those with lighter skins, our method for projecting future incremental skin cancers and cataracts incorporates this and other relevant factors in its benefits estimates. Estimates of non-melanoma fatalities are not calculated from a dose-response function. Instead, the model assumes that the number of non-melanoma deaths will be a fixed percentage of the

[36] Setlow, R.B., "The Wavelengths of Sunlight Effective in Producing Skin Cancer: A Theoretical Analysis," Proceedings of the National Academy of Sciences, 71(9):3363-3366, 1974.

[37] Non-fatal basal and squamous cell non-melanoma incidence rates were obtained from Scotto, J., T. Fears, and Fraumeni, "Incidence of Nonmelanoma Skin cancer in the United States," U.S. Department of Health and Human Services, (NIH) 82-2433, Bethesda, MD, 1981. Non-fatal melanoma incidence rates were obtained from National Cancer Institute SEER Report, 1984. Fatal melanoma incidence rates were obtained from Pitcher, H.M., "Examination of the Empirical Relationship Between Melanoma Death Rates in the United States 1950-1979 and Satellite-Based Estimates of Exposure to Ultraviolet Radiation," U.S. EPA, Washington, DC, March 17, 1987, draft.

[38] Non-fatal Non-Melanoma: Scotto, J, and T. Fears, "Estimating Increases in Skin Cancer Morbidity Due to Increases in Ultraviolet Radiation Exposure," Cancer Investigation, 1(2), 119-126, 1983. Non-fatal Melanoma: Scotto, J, and T. Fears, "The Association of Solar Ultraviolet and Skin Melanoma Incidence Among Caucasians in the United States," Cancer Investigation, 5(4), 275-283, 1987. Melanoma mortality: Pitcher, H.M., and J.D. Longstreth, "Melanoma Mortality and Exposure to Ultraviolet Radiation: An Empirical Relationship, Environment International, vol. 17, 7-21, 1991.

[39] Cataract prevalence data were obtained from Leske and Sperduto, "The Epidemiology of Senile Cataracts: A Review," American Journal of Epidemiology, Vol. 118, No.2, 152-165, 1983. For information on the dose-response relationship, see Hiller, R., R. Sperduto, and F. Ederer, "Epidemiological Associations with Cataract in the 1971-1972 National Health and Nutrition Survey," American Journal of Epidemiology, Vol. 18, No. 2, 239-249, 1983.

total non-melanoma cases.[40] We estimate that from 1990 to 2165 sections 604 and 606 will result in 6.3 million avoided deaths from skin cancer, 27.5 million avoided cataract cases, and 299.0 million cases of non-fatal skin cancers (melanoma and non-melanoma).

Although the evidence linking UV-b and melanoma is controversial, studies suggest that exposure to sunlight is a major environmental risk factor for melanoma. However, uncertainty exists about three aspects of this relationship: the appropriate action spectrum (i.e., the relative contribution of different wavelengths of light to overall risk), the appropriate dose metric (acute, intermittent, or chronic), and the importance of age at exposure. Although UV-b was initially thought to be solely responsible for melanoma, studies by Setlow et al. (1993) and Ley (1997) have shown that UV-a as well as UV-b is a significant factor in the induction of melanoma. The uncertainty surrounding the dose-metric stems from the fact that chronic, cumulative, low-level exposures to sunlight are not associated with development of melanoma. Instead, melanoma risk is higher among those intermittently exposed to sunlight and that melanoma occurs most frequently on body parts that are intermittently exposed. Therefore, current thinking suggests that intermittent, intense bursts of UV exposure (i.e., sunburns) are an important factor in the development of melanoma. Epidemiological studies exploring this hypothesis have confirmed such an association, though the strength of these findings may be weakened by recall bias (Berwick 1998). Finally, melanoma may exhibit a significant latency period; studies such as Holman and Armstrong (1984) have found that severe early life exposures to UV are an important risk factor for melanoma in adults. However, the most recent study of this effect (Autier and Dore, 1998) found that childhood exposures are important only in addition to severe adult exposures.

The effect of the uncertainties in the first two aspects of the UV/melanoma relationship (action spectrum and dose metric) on the melanoma mortality estimates is difficult to determine based on current information. If melanoma mortality exhibits a latency period, our results may be overestimated, because the analysis did not specifically model a latency period.

To estimate crop damage, we apply earlier studies on the relationship between crops, UV-b radiation, and tropospheric ozone to the changes in UV-b radiation and tropospheric ozone predicted by the emissions models.[41] We estimate that the avoided increase in damage to American crop harvests from UV-b radiation by 2075 will equal about 7.5 percent. To calculate the benefits of avoided photodegradation of all UV-b sensitive polymers, we use the Horst *et al.* study (1986) on the relationship between UV-b radiation and the increase in polymer stabilizers needed to mitigate rigid PVC pipe damage.[42]

The unquantified effects of sections 604 and 606 include the following: avoided pain and suffering from skin cancer, ecological effects of UV-b radiation and tropospheric ozone, human health and environmental benefits outside the United States, and changes in pulmonary and respiratory

[40] Non-melanoma mortality estimates are based on the assumption that one percent of non-melanoma incidence results in mortality.

[41] Sources of dose-response relationship for crops and UV-b: Teramura and Murali (1986) and Rowe and Adams (1987). Source of dose-response relationship for crops and tropospheric ozone: Rowe and Adams (1987). Source of increased tropospheric ozone estimates: Whitten and Gery (1986). Our benefits analysis does not include assessing the effects of tropospheric ozone on forests. Although there are C-R functions available that would allow an assessment, we could not use them because of we do not have the necessary measure of tropospheric ozone changes.

[42] Although the *Regulatory Impact Analysis: Compliance with Section 604 of the Clean Air Act for the Phaseout of Ozone-Depleting Chemicals* provides monetized benefits estimates of reduced crop damage from tropospheric ozone and reduced polymer damage from UV-b, it does not provide a quantified estimate in non-monetary terms.

functions from increased tropospheric ozone.[43] The EPA also lists avoided actinic keratosis (pre-cancerous lesions from excessive sun exposure) as another unquantified benefit. Using the first National Health and Nutrition Examination (NHANES I), Engles *et al.* linked increased UV exposure to increased incidence of actinic keratosis.[44] This study, however, did not provide sufficient quantitative information relating the incidence of actinic keratosis to levels of UV radiation. In addition, several researchers provide data suggesting that avoided increases in infection intensity may constitute an unquantified benefit. For example, Perna *et al.* associated UV-b exposure with the reactivation of Herpes virus infections.[45] Other studies have linked UV exposures to reductions in the ability of animals to control infections with *Leishmania sp.* (Giannini and DeFabo*)*; malaria (Taylor and Eagles). The yeast candida (Denkins *et al.* and Chung *et al)*;.the bacterium *staphylococcus aureus* (Chung *et al*).[46] Valerie *et al.* also showed that UV irradiation of cells grown *in vitro* and exposed to sunlight for as little as 10 to 30 minutes can activate the human immunodeficiency virus type 1 (HIV-1).[47] Scientists, however, have not yet provided a quantitative relationship between the impact of UV-b-induced immunosuppression and human disease.[48]

## Physical Effects: Sections 608, 609, and 611

For sections 608, 609, and 611 we base the quantified benefits estimates on the methodology used for sections 604 and 606, but do not provide the quantified estimates cited in the RIAs. For section 608 we use the same emissions, stratospheric ozone, and UV-b radiation methodologies used for sections 604 and 606; the quantified benefits of section 608, however, comprise only benefits from reduced incidences of skin cancer morbidity and mortality. For section 609 the benefits estimate is simply a percentage of the benefits of section 604; in fact, we avoid double counting by omitting 609 benefits from the calculation of the total Title VI benefits estimate. For section 611 we calculate the benefits estimate with a benefit per kilogram ratio obtained from data in the *Regulatory Impact Analysis: Compliance with Section 604 of the Clean Air Act for the Phaseout of Ozone Depleting Chemicals* (1992). We apply this ratio to the emissions reduction caused by firms that accelerate the use of MCF substitutes to avoid labeling.

The unquantified benefits estimates of sections 608, 609, and 611 are the same as the unquantified benefits of sections 604 and 606, with one exception. The analysis of section 611 includes two additional benefits: an increase in available information regarding ozone-depleting substances and enhanced implementation and enforcement of EPA's refrigerant recycling program. Quantified

[43] ICF Incorporated's *Regulatory Impact Analysis: Compliance with Section 604 of the Clean Air Act for the Phaseout of Ozone-Depleting Chemicals* (1992) does not present the information on the studies linking tropospheric ozone with pulmonary and respiratory effects.

[44] Engles, A., M.L. Johnson, and S. Haynes,"Health Effects of Sunlight Exposure in the United States: Results from the First National Health and Nutrition Examination Survey, 1971-1974," *Archives of Dermatology*, Vol. 4, January 1988, pages 72-79.

[45] Perna, J.J., M.L. Mannix, J.E. Rooney, A.L. Notkins, and S.E. Straus, "Reactivation of Latent Herpes Simplex Virus Infections by Ultraviolet Light: A Human Model," *Journal of the American Academy of Dermatology*, 17, 1987, pages 473-478.

[46] Chung, H.T., D.C. Lee, S.Y. Im, and R.A. Daynes, "UVR-Exposed Animals Exhibit and Enhanced Susceptibility to Bacterial and Fungal Infections," *Journal of Investigative Dermatology,* Vol. 90, No. 4, April 1988, page 52; Denkins, Y., I.J., Fidler, and Kripke, M.L., "Exposure of Mice to UV-B Radiation Suppresses Delayed Hypersensitivity to *Candida albicans*," *Photobiology and Photochemistry*, 1989; Giannini, S.H., and E.C. DeFabo, "Abrogation of Skin Lesions in Cutaneous Leishmaniasis by Ultraviolet B Irradiation," *Leishmaniasis: The First Centenary (1885-1985) New Strategies for Control*, Heart, D.T. (ed.), NATO ASI Series A: Life Sciences , London, Plenum Pub., Cos.; Taylor, D.W. and D.A. Eagles, "Assessing the Effects of Ultraviolet Radiation on Malarial Immunity," Prepared for Sabotka and Company under EPA contract number 68-01-7288, subcontract number 132.914.

[47] Valerie, K., A. Delers, C. Bruck, C. Thiriart, H. Rosenberg, C. Debouck, and M. Rosenberg, "Activation of Human Immunodeficiency Virus Type 1 by DNA Damage in Human Cells," *Nature,* Vol. 333, May 5, 1988, pages 78-81.

[48] Ibid, ICF (1992), 6-26.

and unquantified benefits of 608 and 611 are summarized in Table G-5 and G-6 respectively.

**Table G-5**
**Benefits of Section 608**

| |
|---|
| **Quantified Health Effects** |
| Skin cancer: fatal and nonfatal |
| **Health Effects- Unquantified** |
| Skin cancer: reduced pain and suffering |
| Cataracts: reduced morbidity, pain and suffering |
| Reduced morbidity effects of increased UV. For example,<br>• reduced actinic keratosis (pre-cancerous lesions resulting from excessive sun exposure)<br>• reduced immune system suppression. |
| **Ecological Effects- Unquantified** |
| Ecological effects of UV. For example, benefits relating to the following:<br>• recreational fishing<br>• forests<br>• overall marine ecosystem<br>• avoided sea level rise - which, in turn, leads to:<br>decreased beach erosion<br>decreased loss of coastal wetlands<br>decreases in the salinity of estuaries and aquifers<br>• other crops<br>• other plant species |
| Other ecological benefits of reduced tropospheric ozone relating to<br>• the overall marine ecosystem<br>• forests<br>• man-made materials (e.g., degradation of elastomers, textile fibers and dyes, certain paints)<br>• other crops<br>• other plant species |
| Benefits to people and the environment outside the U.S. |
| Effects, both ecological and human health, associated with global warming. |

**Table G-6**
**Benefits of Section 611**

| |
|---|
| **Health Effects- Quantified** |
| Skin cancer: fatal and nonfatal |
| Cataracts: reduced morbidity, pain and suffering |
| **Ecological Effects- Quantified** |
| Crops: reduced damage associated with increased UV radiation |
| Crops: reduced damage associated with increased tropospheric ozone |
| Polymers: reduced degradation from UV-b radiation |
| **Health Effects- Unquantified** |
| Skin cancer: reduced pain and suffering |
| Reduced morbidity effects of increased UV. For example,<br>• reduced actinic keratosis (pre-cancerous lesions resulting from excessive sun exposure)<br>• reduced immune system suppression |
| **Ecological Effects- Unquantified** |
| Ecological effects of UV. For example, benefits relating to the following:<br>• recreational fishing<br>• forests<br>• overall marine ecosystem<br>• avoided sea level rise - which, in turn, leads to:<br>decreased beach erosion<br>decreased loss of coastal wetlands<br>decreases in the salinity of estuaries and aquifers<br>• crops in general<br>• other plant species<br>• fish harvests |
| Ecological benefits of reduced tropospheric ozone relating to<br>• the overall marine ecosystem<br>• forests<br>• man-made materials (e.g., degradation of elastomers, textile fibers and dyes, certain paints)<br>• crops in general<br>• other plant species<br>• fish harvests |
| Benefits to people and the environment outside the U.S. |
| Enhanced implementation and enforcement of EPA's refrigerant recycling program |
| Increase in available information regarding ozone-depleting substances (ODSs); consumers who wish to buy products that do not contain ODSs will be better able to express their preferences through their purchasing power. |
| Effects, both ecological and human health, associated with global warming. |

## Valuation

To calculate monetary values of the quantified benefits, we multiply the physical effects estimates by the appropriate physical effects value. For the health benefits, we use $15,000 for the avoided cost of cataracts, $15,000 for the avoided cost of melanoma skin cancer, and $5,000 for the avoided cost of nonmelanoma skin cancer.[49] This analysis employs a value of statistical life of $4.8 million (1990 dollars), which is the value used to calculate the criteria pollutant mortality benefits estimate presented in Chapter 6, Table 6-3. To calculate the monetary benefits of increased crop yields, the model multiplies the change in crop yields by crop values from the Department of Agriculture.[50] To calculate the monetary benefits related to fish, we apply $739 per ton (1990 dollars) to the increase in fish harvests.[51] We define the polymer benefits as the avoided loss in consumer surplus associated with increased polymer prices. We assume that the cost is proportional to the increase in price following the addition of stabilizers and that the price of polymer stabilizers will increase by 1.86 percent for each 25 percent increase in stabilizer.[52]

With a two percent discount rate, the benefits of sections 608, 609, and 611 are $671 million, $296 million, and $831 million (1990 dollars), respectively. We do not separate these values into their components. The total monetized health benefits for section 604 and 606 with a two percent discount rate are $4.2 trillion and the total monetized ecological benefits are $92.5 billion; thus, the total benefits of sections 604 and 606 are about $4.3 trillion. Table G-7 is a tabular summary of the monetary values of the benefits from sections 604 and 606, which generate about 98 percent of the monetized benefits.[53]

[49] Wasson, John and Steve Abseck, "Memorandum: Further Detail on the Costs and Benefits of Phasing Out Ozone Depleting Substances, EPA Contract No. 68-D4-0103, WA-205," Prepared for Jim DeMocker, U.S. Environmental Protection Agency, October 9, 1995.

[50] The RIAs do not provide the specific crop values used.

[51] The U.S. Department of Commerce provides the fish values. (Ibid, ICF (1992), 6-29.)

[52] Ibid, ICF (1992), 6-41.

[53] The dollar year was not available for some cost of illness estimates in Table G-7. Because these estimates come from a 1988 RIA, we are thus underestimating the monetized benefits of the health effects associated with these unadjusted values.

**Table G-7**
**Sections 604 and 606: Valuation of Total Benefits from 1990 to 2165, With a Two Percent Discount Rate**

| Quantified Effects | Valuation (1990 dollars) | Source Data |
|---|---|---|
| **Health Effects** | | |
| Mortalities from skin cancer (melanoma and nonmelanoma) in the U.S. (1990-2165) | $3,900 billion | Value of statistical life: $4.8 million (1990 dollars). (See Appendix H for a description of source data.) |
| Cataract cases in the U.S. (1990-2165) | $72 billion | Avoided cost of cataracts: $15,000 (dollar year not provided) Costs include increased medical costs, increased work loss, increased costs for chores, other indirect social and economic costs, and willingness to pay to avoid cataracts. Data from literature review, contacts with health providers, and cataract patient survey. (Source: Rowe *et al.* 1987) |
| Nonfatal skin cancer cases (melanoma and nonmelanoma) in the U.S. (1990-2165) | $220 billion | Cost of melanoma skin cancer: $15,000 per case (dollar year not provided); costs of nonmelanoma skin cancer: $5,000 per case (dollar year not provided). Estimates include increased medical costs and decreased productivity but do not include costs of caregiving and chores performed by others. Data from Skin Cancer Focus Group. (See ICF's August 1988 RIA for details.) |
| **Total Health Benefits** | **$4,200 billion** | |
| **Ecological Effects** | | |
| • Decrease in American crop harvests from UV-b radiation by 2075 | $49 billion | Crop values from Department of Agriculture. |
| • Decrease in American crops from tropospheric ozone by 2075 | $28 billion | Crop values from Department of Agriculture. |
| • Damage to polymers from UV-b radiation by 2075 | $6 billion | Costs are proportional to the increase in polymer prices following the addition of stabilizers. Price increase of 1.86% expected for a 25% increase in stabilizer. |
| **Total Environmental Benefits** | **$84 billion** | |
| **Total Benefits** | **$4,300 billion** | |

Note : 1) The RIAs do not provide specific crop values

## Adjustments to Estimates From Existing Analyses

To ensure consistency with assumptions in other portions of the current study, we adjust certain parameters used in existing regulatory impact assessments of Title VI provisions. We vary several parameters and compare the resulting net present values of Title VI's costs and benefits. The Title VI analysis generates net present values, rather than annualized values, because annualization incorrectly imputes benefits of later phaseouts to earlier years. For example, annualization of the benefits of phasing out HCFC-22 by 2020 attributes benefits to years prior to 2030, when neither the costs nor the benefits of that phaseout have yet occurred. Consequently, the annualized estimate overstates benefits at the beginning of the time span and understates them later.

Table G-8 describes the values we use for the following parameters: discount rate and value of statistical life. We are able to adjust key parameters in the benefits analyses of sections 604, 606, and 609 and the cost analyses of sections 604 and 606. We cannot adjust parameters for other sections, however, because we lack annual cost and benefit data from these sections.[54] Moreover, for section 604 and the accelerated phaseout schedule of section 606, we are unable to modify the parameters for the analysis of ecological benefits. Nevertheless, the benefits from sections 604 and 606 constitute the majority of Title VI benefits (approximately 98 percent at a two percent discount rate) and only about one percent of the benefits of these sections result from ecological benefits. In addition, sections 604 and 606 account for about 97 percent of the costs (at a two percent discount rate).

### *Discount Rate*

Because the benefits occur over several hundred years, the chosen discount rate can have an especially large effect on the benefits estimate. In this analysis we use a five percent discount rate for our primary estimate. This is consistent with the retrospective analysis of the Clean Air Act and the other analyses conducted for the present study.[55] We also preform sensitivity tests using discount rates of three percent and seven percent. Finally, for consistency with cost and benefit estimates that we cannot adjust, we calculate aggregate benefits and costs using a discount rate of two percent.

[54] We calculate the benefits of section 609 as a percentage of the benefits of section 604, so we do not need annual data for section 609.

[55] U.S. Environmental Protection Agency, *The Benefits and Costs of the Clean Air Act, 1970 to 1990,* October 1997.

**Table G-8**
**Adjustments to Key Parameters of Existing Analyses That Support the 812 Title VI Estimates**

| Parameter | Assumption for Section 812 Prospective | Adjustments to Title VI Analyses | | | |
|---|---|---|---|---|---|
| | | Sections 604 and 606 | Section 608 | Section 609 | Section 611 |
| Discount Rate (Used for Costs and Benefits) | Central Case: 5% Sensitivity tests: 3% and 7% | Used Section 812 parameters, plus 2%. | 2% (No adjustment possible.) | 2% (No adjustment possible for costs.) | 2% (No adjustment possible.) |
| Value of Statistical Life (Benefits Only) | Distribution of values from $0.6 million to $13.5 million with expected value of $4.8 million | Used Section 812 parameters. | $3 to $12 million | Used Section 812 parameters. | $3 to $12 million |

### *Value of Statistical Life*

The value of statistical life (VSL) is essential for measuring the monetized benefits associated with a reduced number of skin cancer mortality cases. We use a $4.8 million central estimate of VSL, based on analysis described in Appendix H. To reflect the uncertainty of the VSL estimates, we employ a Monte Carlo approach using a Weibull distribution of VSL estimates as an input. This distribution is the same as that used in the analysis of criteria pollutants.[56]

[56] The Weibull distribution has the following parameters: a location of $0.0, a scale of $5.32 million, and a shape of 1.509588.

## Cost and Benefit Results With Adjusted Parameters

Both cost and benefit estimates are sensitive to the discount rate. As mentioned earlier, the discount rate has a particularly significant effect on the benefits estimate because the benefits occur over several hundred years (1990 to 2165). These benefits result from actions taken to reduce ozone-depleting chemical emissions from 1990 to 2075, the time period over which costa are incurred. In this section we first present the net present value of the costs and benefits using the central discount rate of five percent. We then discuss the results of the sensitivity tests using discount rates of three and seven percent. Lastly, we show the results using a two percent discount rate, which is consistent with the discount rates used in existing RIAs and which allows us to compare the costs and benefits of all the major sections of Title VI, including provisions where discount rate adjustments are not possible.

The adjusted primary benefit estimate (using a five percent discount rate) for Title VI is $530 billion and the cost estimate is $30 billion. The benefits range from $240 billion with a seven percent discount rate and $1,900 billion with a three percent discount rate. The costs range from $20

billion to $40 billion, with the same respective discount rates. The benefits of Title VI greatly exceed the costs for all discount rate assumptions; in fact, the benefits are about 20 times greater than the costs at a five percent discount rate.[57] (See Table G-9) Even the seven percent discount rate sensitivity test yields total benefits that are 12 times greater than the costs.

### Five Percent Discount Rate

With a five percent discount rate, the expected human health benefits from sections 604 and 606 are approximately $400 billion. Table G-10 shows the results of the statistical simulation modeling analysis; the 5th and 95th percentile values are $100 billion to $900 billion, respectively. The annual human health benefits from sections 604 and 606, calculated with a five percent discount rate, steadily increase until about 2045; they then decrease until 2165, the last year in the analysis. (See Figure G-2.) About 93 percent of the benefits occur from 2015 to 2165.

The costs of sections 604 and 606 of Title VI are approximately $26 billion; these sections generate approximately 97 percent of the costs. The human health benefits for sections 604 and 606 are almost 17 times greater than the costs of these sections.

[57] Note that we do not include the costs of the methyl bromide phaseout of section 606 because existing RIAs do not provide benefits estimates for this phaseout. The costs, calculated with a three percent discount rate, are about $1.7 billion.

**Table G-9**
**Costs and Benefits of Sections 604 and 606[1]**

| Discount Rate | Benefits (Trillions) 1990 Dollars | Costs (Trillions) 1990 Dollars | Benefits/Cost Ratio |
|---|---|---|---|
| 2% | $4.24 | $0.06 | 76 |
| 3% | $1.81 | $0.04 | 44 |
| 5% | $0.44 | $0.03 | 17 |
| 7% | $0.14 | $0.02 | 8 |

Notes:

1. The cost and benefits estimates associated with a two percent discount rate are the estimates for sections 604, 606, 608, 609, and 611. For the other discount rates the estimates represent the costs and the human health benefits for sections 604 and 606. These two sections generate the majority of the Title VI costs and benefits (approximately 98 percent of the benefits and 97 percent of the costs in the two percent discount rate calculations).
2. We do not include the costs of the methyl bromide phaseout of section 606 because existing RIAs do not provide benefit estimates for this phaseout.
3. In general, the costs occur from 1990 to 2075, while the benefits occur from 1990 to 2165. (Tables G-11 and G-12 provide the specific time frame for each section of Title VI.)

## *Three Percent and Seven Percent Sensitivity Tests*

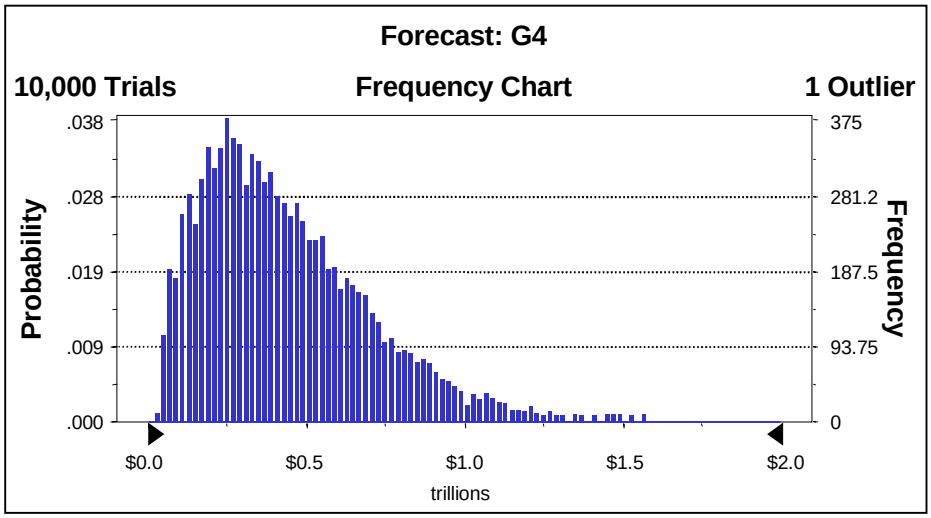
The expected human health benefits from sections 604 and 606 are approximately $1,800 billion at a three percent discount rate and $100 billion at a seven percent discount rate. With a three percent discount rate, the range of expected human health benefits is $100 billion to $7,800 billion, with 90 percent of these expected benefits between $400 billion and $4,000 billion. By contrast, at a seven percent discount rate the range of expected human health benefits is $14 billion to $700 billion, with 90 percent of the expected human health benefits between $33 billion and $300 billion.

The annual benefits from sections 604 and 606, calculated with a three percent discount rate, steadily increase until about 2062; they then decrease till 2165, the last year in the analysis. About 99 percent of the benefits occur from 2015 to 2165. At a seven percent discount rate the annual benefits from sections 604 and 606 steadily increase until about 2038 and then decrease till 2165. About 92 percent of the benefits calculated with a seven percent discount rate occur from 2015 to 2165.

The costs of sections 604 and 606 of Title VI are approximately $41 billion at a three percent discount rate and approximately $18 billion at a seven percent discount rate; these sections account for approximately 97 percent of the total costs. The costs for sections 604 and 606 are about 44 times smaller than the human health benefits for these sections at a three percent discount rate and about 8 times smaller at a seven percent discount rate.

**Table G-10**
**Human Health Benefits for Sections 604 and 606**

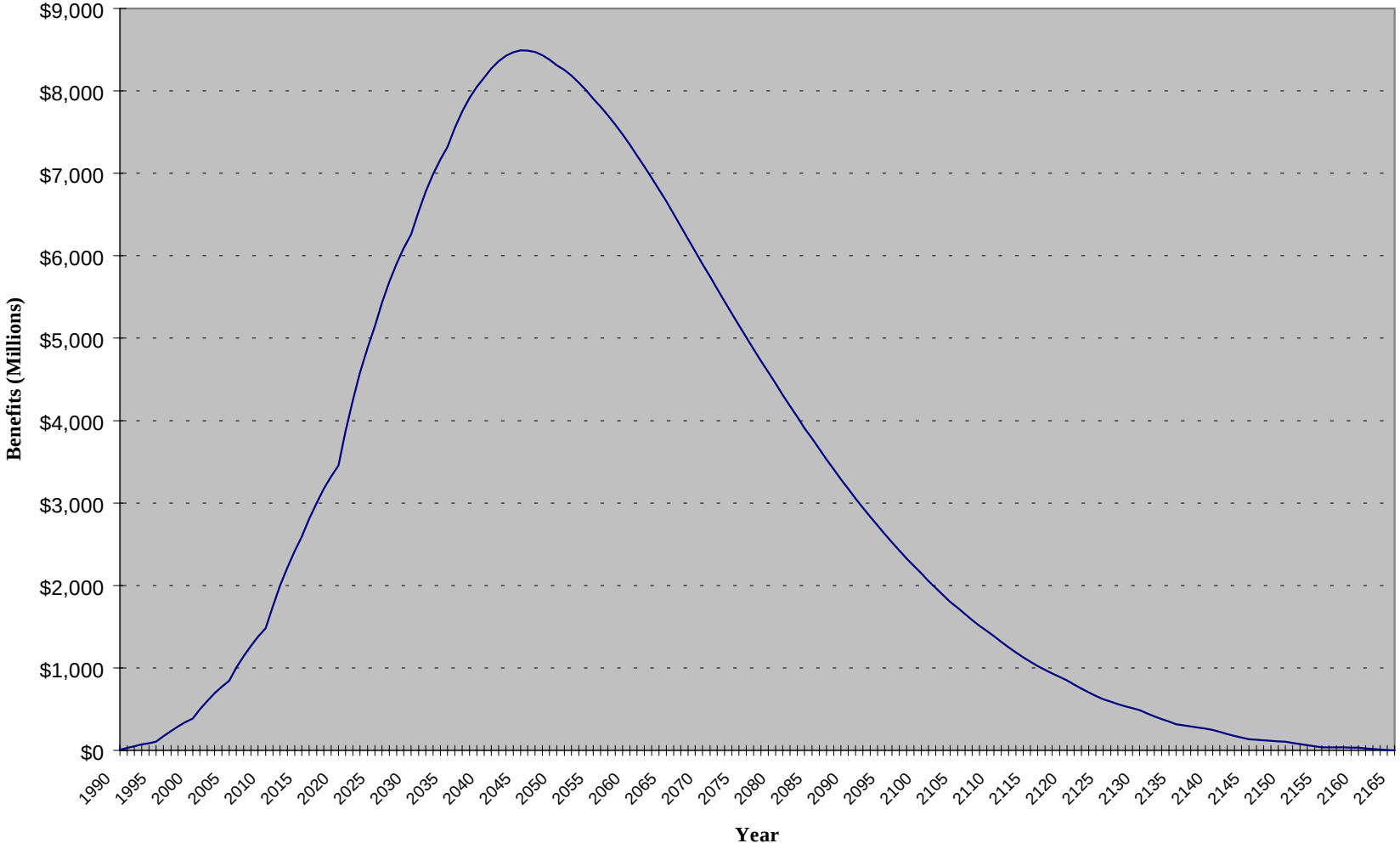
| | **Billions of 1990 dollars** |
|---|---|
| **Range:** | $40 to $2,600 |
| **Mean:** | $400 |
| **Median:** | $400 |
| **Standard Deviation:** | $300 |
| **Percentiles:** | |
| **5%** | $100 |
| **50%** | $400 |
| **95%** | $900 |




| | |
|---|---|
| Percent of Benefits from 1990 to 2014 | 3.64% |
| Percent of Benefits from 2015 to 2165 | 93.36% |

Note: Estimates calculated with a five percent discount rate.

**Figure G-2**
**Annual Human Health Benefits From Sections 604 and 606 (Discounted at 5%)**



### Two Percent Discount Rate

Using benefits estimates from RIAs for sections 608 and 611 and re-calculating the benefits of sections 604, 606, and 609 with a two percent discount rate and a VSL of $4.8 million yields a *total* benefits estimate of $4.3 trillion (1990 dollars) for Title VI.[58] (See Table G-11). Of this estimate, $4.2 trillion (98 percent) results from the human health benefits of sections 604 and 606. The range of expected benefits from human health improvements is $0.3 trillion to $20.8 trillion, with 90 percent of the expected benefits between $0.9 trillion and $9.4 trillion. The annual human health benefits from sections 604 and 606, calculated with a two percent discount rate, steadily increase from 1990 until about 2077; they then decrease till 2165. About 99 percent of the benefits occur from 2015 to 2165.

To estimate the costs of Title VI with a two percent discount rate, we use the estimates from the RIAs that analyze sections 608, 609, and 611 and we re-calculate the costs of sections 604 and 606 using the two percent discount rate.[59] Total present value Title VI costs with a two percent discount rate are approximately $57 billion. The cost estimate is about 76 times smaller than the comparable benefits estimate. Table G-12 lists the cost components.

### Undiscounted Benefits

The annual undiscounted benefits from sections 604 and 606 steadily increase until about 2110; they then decrease in steps until 2165. The steps appear to be related to the application of 10-year cohort survival rates for persons born after 2075. See Figure G-3 for a graphical depiction of the annual benefits.

[58] The RIAs for sections 608 and 611 present a lower bound benefits estimate that incorporates a $3 million VSL and an upper bound benefits estimate that incorporates a $12 million VSL. To obtain a point estimate, we weight the lower bound by 80 percent and the upper bound by 20 percent. (The benefits of section 608 do not include the HCFC phaseout benefits because the phaseout is too complex to model.) In addition, note that the benefits of section 609 are a subset of the benefits of sections 604 and 606, so we do not add them separately to the benefits of the other sections to obtain the total benefits.

[59] The estimate for section 611 is an average of the lower bound, which assumes that only companies using ODSs as solvents will label products, and an upper bound, which assumes that 10 times as many companies use solvent-cleaned products and will need to label their products. Also, the costs for section 608 do not include the costs of the HCFC phaseout.

**Table G-11**

**Summary of Benefits of Title VI with a Two Percent Discount Rate and $4.8 Million VSL**

| | Section | Benefits (Millions) 1990 Dollars | Notes | Years During Which Benefits Accrue |
|---|---|---|---|---|
| **604 & 606** | **Class I Phaseout** | **$4,338,000** | | |
| | - Reduced Mortality, Cataracts, and Non-Fatal Cancers | $4,243,000 | | 1990 to 2165 |
| | - Methyl Bromide Reductions | | No information currently available. | 1994 to 2160 |
| | - Ecological Benefits | $84,000 | | 1989 to 2075 |
| **605** | **Class II Phaseout** | | No information currently available. | |
| **608** | **National Recycling and Emission Reduction Program** | **$670** | 1) Only health effects are monetized.<br>2) The RIA for section 608 does not include benefits of HCFC phaseout because this phaseout is too complex and predicting baseline innovation is too difficult.<br>3) The benefits estimate listed is the weighted average of the benefits calculated with $3 million and $12 million for the VSL. (The $3 million estimate has a weight of 0.8 and the $12 million estimate has a weight of 0.2)<br>4) Benefits reflect the accelerated phaseout schedule. | 1994 to 2165 |
| **609** | **Servicing of Motor Vehicle Air Conditioners** | **$300** | The benefits of section 609 are a subset of the benefits of sections 604 and 606; we calculate section 609 benefits as 0.00682% of the benefits of 604 and 606 combined. (This percentage is the total benefits of 609 in 1989 dollars divided by the total 604 benefits in 1989 dollars.) | 1991 to 2075 |
| **611** | **Labeling** | **$830** | 1) The benefits do not reflect the accelerated phase-out schedule.<br>2) The benefits estimate listed is the weighted average of the benefits calculated with $3 million and $12 million for the value of a statistical life. (The $3 million estimate has a weight of 0.8 and the $12 estimate has a weight of 0.2) | 1989 to 2075 |
| | **TOTAL** | **$4,339,000** | | |

Notes: 1) All benefits expressed in 1990 dollars using the implicit GDP deflator from the *1998 Economic Report of the President.*
2) The benefits of section 611 and the ecological benefits of section 604 do not reflect the accelerated reduction and phaseout schedule of section 606.

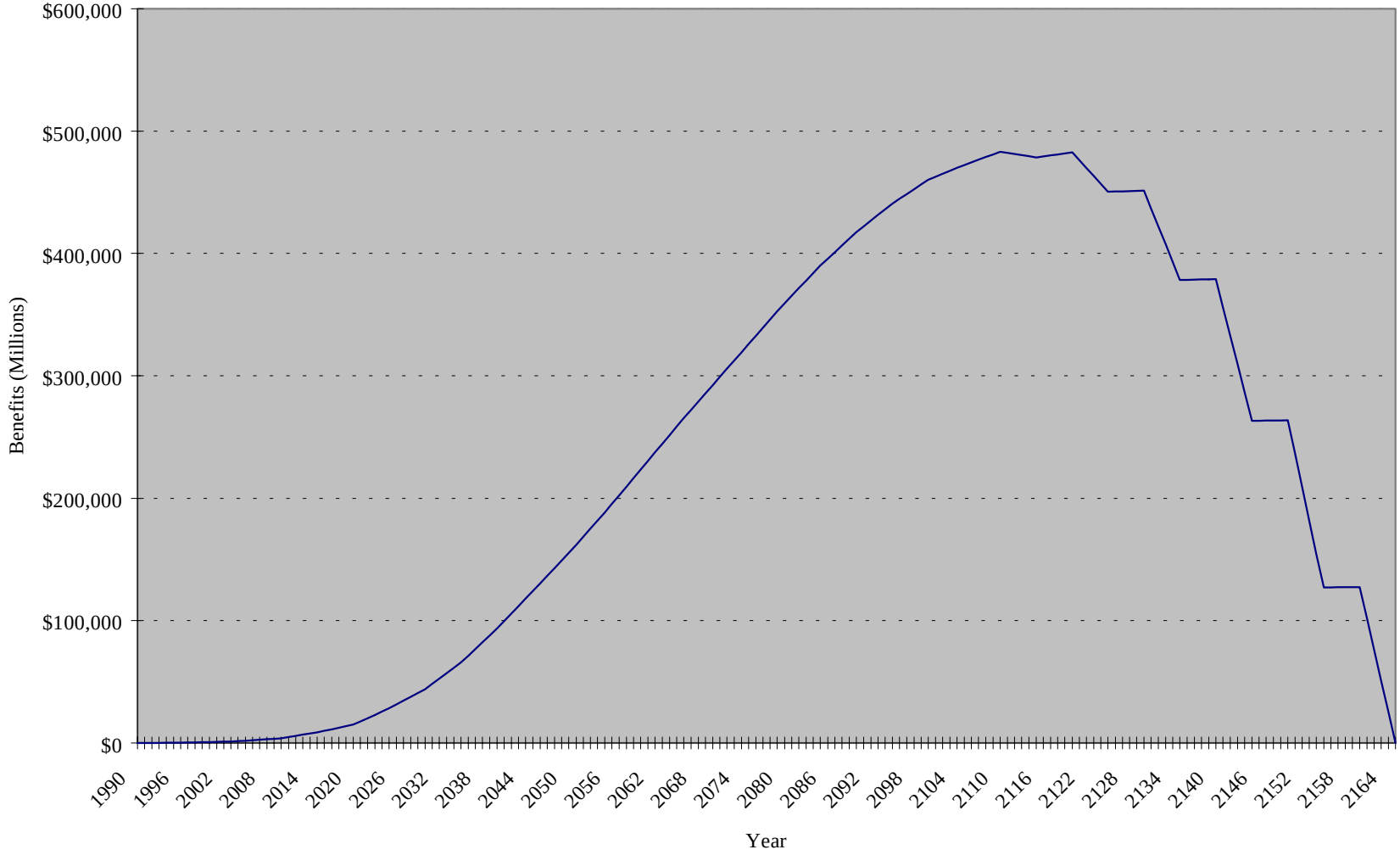
**Table G-12**

**Summary of Costs for Title VI by Section with a Two Percent Discount Rate**

| | Section | Cost Estimate (Millions, 1990 Dollars) | Notes | Years During Which Costs Accrue |
|---|---|---|---|---|
| **604 & 606** | **Class I Phaseout** | **$56,000** | Does not include cost of methyl bromide reductions. | 1990 to 2075 |
| **605** | **Class II Phaseout** | | No information currently available. | |
| **608** | **National Recycling and Emission Reduction Program** | **$1,200** | 1) We convert costs from 1991 to 1990 dollars using the GDP deflator from the *1998 Economic Report of the President*.<br>2) The RIA for Sect. 608 does not include costs of HCFC phaseout.<br>3) At a 4% discount rate the costs are $1,074.38 million, and at a 7% discount rate the costs are $853.91. | 1994 to 2015 |
| **609** | **Servicing of Motor Vehicle Air Conditioners** | **$14** | 1) We convert from 1991 to 1990 dollars using the GDP deflator from the *1998 Economic Report of the President*.<br>2) To avoid counting the same costs for both section 604 and 609, we include only the operator training and equipment certification costs of section 609 here. | 1992 to 2008 |
| **611** | **Labeling** | **$250** | 1) The costs are probably in 1990 dollars, but this is unclear.<br>2) Most costs are one-time costs.<br>3) The cost estimate is an average of the lower bound, which assumes that only firms using ODSs as solvents will label products, and an upper bound, which assumes that 10 times as many firms will need to label their products because they incorporate solvent-cleaned products. | 1994 to 2000 |
| | **TOTAL** | **$57,000** | | |

Notes: 1) The costs listed above for sections 604 & 606 do not include methyl bromide costs, which equal $1.7 billion with a 3% discount rate, because the RIA did not present the corresponding benefits.
2) The costs of sections 609 and 611 do not reflect the accelerated reduction and phaseout schedules of section 606.

**Figure G-3**
**ANNUAL UNDISCOUNTED HUMAN HEALTH BENEFITS OF SECTIONS 604 AND 606**



## Limitations And Uncertainties

An analysis of programs that protect stratospheric ozone necessarily considers impacts over hundreds of years, introducing a wide range of uncertainty in the estimates of costs and benefits. There are clearly limitations and uncertainties in predicting the advancement of both medical treatment and ODS substitution technologies over time, modeling averting behavior of individuals in response to changes in UV-b radiation levels, anticipating the results of new information that might alter the modeling of stratospheric ozone depletion and formation, and forecasting economic parameters such as the growth in GDP and the valuation of health risk reduction.

We have not attempted to characterize the impact of all the uncertainties and limitations inherent in this type of analysis. For more detail, we refer the reader to the source material in EPA's RIAs for Title VI provisions and the descriptions of the cost and benefit modeling approaches found there. As part of this analysis, however, we conduct selected quantitative sensitivity tests and literature reviews to characterize several major uncertainties in the cost and benefits analyses presented here. The discussion that follows includes characterization of the following: issues in long-term discounting; limitations in the cost modeling; and limitations in the benefits modeling, including the modeling of averting behavior.

### *Long-term Discounting*

As demonstrated above, the discount rate can have an important effect on the estimation of costs and benefits that accrue over a long period of time. Long-term discounting may present special problems that are worth exploring in some detail through sensitivity tests of alternative discount rate assumptions. For example, some economic literature suggests that accounting for intergenerational transfers in a manner different from intragenerational transfers may be appropriate.[60] One possible rationale for treating long-term, intergenerational transfers differently is that an individual's rate of time preference (which presumably applies only for his or her lifetime, or intragenerationally) may differ from his or her bequest motive for future generations. At least one empirical study suggests that individuals may implicitly apply lower discount rates for programs where the benefits accrue later in time.[61] In addition, people may attribute the same level of importance to all events that occur in the *far-distant* future, regardless of the relative position of these events in time. According to Weitzman, analysts should apply the lowest possible nonnegative rate to events in the far-distant future.[62]

Although some of the arguments for using an alternative discounting procedure for long-term benefits and costs are persuasive, implementation of an alternative procedure is not straightforward. There appears to be little guidance in the existing economic literature on the key issues of what discount rate to use for long-term versus short-term discounting as well as when to alter the discount rate. Recently drafted EPA guidance on the conduct of economic analyses, however, suggests that longer-term discount rates might be

[60] Arrow, K.J., W.R. Cline, K.G. Maler, M. Munasinghe, R. Squitieri, and J.E. Stiglitz, "Intertemporal Equity, Discounting, and Economic Efficiency," *Climate Change 1995: Economic and Social Dimensions of Climate Change*. Edited by J.P. Bruce, H. Lee, and E.F. Haites, Cambridge: Cambridge University Press, 1996; Lind, Robert C., "Intergenerational Equity, Discounting, and the Role of Cost-Benefit Analysis in Evaluating Global Climate Policy," *Integrative Assessment of Mitigation, Impacts, and Adaptation to Climate,* Edited by N. Nakicenovic, *et al.,* Laxenburg, Austria: International Institute of Applied Systems Analysis, 1994; Schelling, Thomas C., "Intergenerational Discounting," *Energy Policy*. 23(4/5), 1995, pages 395-401; Solow, Robert, "An Almost Practical Step Toward Sustainability," Paper presented at the Fortieth Anniversary of Resources for the Future, October 8, 1992, in Washington, D.C.

[61] Cropper, Maureen L., Sema K. Aydede, and Paul R. Portney, "Discounting Human Lives." *American Journal of Agricultural Economics*, December 1991: 1410-1414.

[62] Weitzman, Martin L., "Why the Far-Distant Future Should Be Discounted at its Lowest Possible Rate," *Journal of Environmental Economics and Management*, Volume 36, 201-208 (1998).

approximated through Ramsey Rule discounting, using effective annual discount rates of from 0.5 to 3.0 percent.[63] Throughout our presentation of Title VI cost and benefit results, we use a five percent discount rate for our primary estimate. We also calculate alternative estimates using three and seven percent discount rates. These discount rates maintain consistency with other analyses of the prospective.

## Costs

Major uncertainties in the cost estimates result where it is difficult to predict the pace and nature of innovation in key industries. To the extent the models used do not quantify transition costs in the long term, the uncertainty in the cost estimates increases. In addition, predicting the responses of manufacturers to the different sections of Title VI is difficult. Table G-13 lists the primary causes of uncertainty in the cost estimates.

[63] We use the discount rate of 0.5 percent as a low nonnegative discount rate for events in the far-distant future. (Frank Arnold *et al.*'s *Draft Final Report: Discounting in Environmental Policy Evaluation* supports long-term discount rates from 0.5 percent to 3.0 percent.)

**Table G-13**
**Major Limitations of Existing Cost Analyses for Title VI**

| Limitations | Effects on Cost Estimates |
|---|---|
| **Sections 604*, 606, and 609** | |
| The assumption of little or no technical innovation in ODS-related industries over the time span of the analysis may be inaccurate. | Inclusion of innovation may decrease the cost estimate. |
| The model does not quantify transition costs, such as temporary layoffs, administrative costs, and the costs of unknown environmental hazards created by the use of alternatives to CFCs. | Inclusion of transition costs may increase the cost estimate. |
| **Section 608** | |
| The assumptions of the capital and operating costs of recovery devices and the time necessary to perform recycling operations are largely hypothetical. | A better understanding of the capital and operating costs, as well as the time necessary for recycling, may either increase or decrease cost estimates. |
| The extent of compliance with recycling rates mandated by the Venting Prohibition is very uncertain.[1] Therefore, the baseline assumptions regarding the percentages of CFCs that are actually recycled are also very uncertain. | A better ability to forecast recycling rates may either decrease or increase cost estimates. |
| The baseline recycling value assumes no innovation in recycling technologies through 2017, which may be inaccurate. | Including innovation in recycling technologies may decrease the cost estimates. |
| **Section 611** | |
| Manufacturers' responses to the labeling requirement may include labeling, reformulating products, ceasing production, or petitioning for an exemption to the labeling requirement. Predicting the frequency of these responses is difficult. | A better ability to forecast manufacturers' responses may either decrease or increase cost estimates. |

Note: *We do not include the costs of the methyl bromide phaseout in the total cost estimate because the RIAs do not provide the benefits of this phaseout. The cost estimates for the methyl bromide phaseout are uncertain, because the model assumes that the demand for output manufactured with methyl bromide is perfectly inelastic and that the methyl bromide production industry is perfectly competitive. While these assumptions may be unrealistic, they allowed the analysis to focus on consumer impacts and ignore effects on output markets.

[1] The Venting Prohibition is essentially a recovery and recycling requirement. For more detail see ICF (1993).

## Benefits

Several factors contribute to uncertainty in the benefit estimates. (See Table G-14 for a list of major limitations.) For example, scientists have an incomplete understanding of the processes that govern ozone depletion and affect exposure to UV-b radiation. In addition, the dose-response coefficients relating UV-b exposure to melanoma skin cancer and cataracts are difficult to estimate. Scientists have not yet developed quantified dose-response relationships for some benefits, such as reduced damage to the immune system from UV-b radiation. As a result, the benefit estimates may either overestimate or underestimate the true benefits of Title VI provisions.

Data limitations also impede attempts to monetize certain benefits. For example, there are well established concentration-response functions that would allow us to measure the effects of tropospheric ozone on forests. We are, however, unable to use the CR functions because we do not have the necessary measured changes in ozone. As a result, we are also unable to monetize these benefits.

Another difficulty involves the long term nature of the study. Predicting invention, research and development, producer and consumer responses to price changes, and technological change for the next century and a half is highly speculative. Predicting major natural events that influence the effects of stratospheric ozone depletion is also difficult.[64] Our inability to forecast with accuracy may cause the benefit estimate to be too high or too low.

Lastly, the quantitative analysis of Title VI does not account for potential increases in averting behavior (e.g., people's efforts to protect themselves from UV-b radiation). Murdoch and Thayer (1990) estimate that the cost-of-illness estimates for nonmelanoma skin cancer cases between 2000 and 2050 may be almost twice the estimated cost of averting behavior (application of sunscreen).[65] To estimate benefits, the Title VI analysis relies on epidemiological studies, which incorporate averting behavior as currently practiced. Omission of future increases in averting behavior may nonetheless overstate the benefits of reduced emissions of ozone-depleting chemicals.[66] The analysis may understate the benefits, however, if individuals alter their behaviors in ways that could increase exposure or risk (e.g., sunbathing more frequently and/or for longer periods).[67]

[64] For example, volcanic eruptions increase dust levels, which may affect risks from stratospheric ozone depletion.

[65] Murdoch, James C. and Mark A. Thayer, "The Benefits of Reducing the Incidence of Nonmelanoma Skin Cancers: A Defensive Expenditures Approach," *Journal of Environmental Economics and Management*, 1990, pages 107-119.

[66] Although Dr. Marianne Berwick, an epidemiologist at Memorial Sloan-Kettering Cancer Center in New York, issued a study indicating that sunscreens are ineffective in preventing melanoma, many dermatologists contest this assertion ("Studies Doubt Sunscreens Stop a Cancer," *The New York Times*, February 2, 1998, page 19; Berwick , Marianne, *Sunscreens and Skin Cancer: The Epidemiological Evidence*, February 17, 1998; Boyd, Christopher, "Sunscreen Research Burns Up Skin Specialists: Doctors Fear Controversial Report Will Confuse Public," *Orlando Sentinel*, March 1, 1998, page A4).

[67] Autier *et al.*, 1999.

**Table G-14**
**Major Limitations of Existing Benefits Analyses for Title VI**

| Limitations | Effects on Benefits Estimates |
|---|---|
| Scientists have an incomplete understanding of<br>• the chemical and physical processes that cause ozone depletion,<br>• the relationship between ozone depletion and exposure to ultraviolet radiation (UV-b), and<br>• the dose-response coefficients relating UV-b exposure to melanoma skin cancer and cataracts. | A better understanding may either increase or decrease the benefits estimate. |
| Scientists have not yet developed quantified dose-response relationships for some benefits, such as reduced damage to the immune system from UV-b radiation. | Additional dose-response relationships will increase the benefits estimate. |
| The long term nature of the studies introduces a significant degree of uncertainty. For example, predicting innovation, research and development, producer and consumer responses to price changes, technological change, and major natural events for the next 100 to 150 years is difficult. | A better ability to forecast future events may either decrease or increase benefits estimates. |
| Although truncation of benefits and cost streams is necessary for the analysis, it does influence the size of the benefit and cost estimates. | The current method of truncating benefit and costs streams results in a greater underestimation of benefits than costs. |
| The RIAs do not account for averting behavior (i.e., people's efforts to protect themselves from UV-b radiation) or behavior increasing exposure or risk (e.g., increased sunbathing). | Inclusion of averting behavior may decrease the benefits estimate, while inclusion of enhancing behavior may increase the benefits estimate. |
| Not all RIAs for Title VI include comprehensively monetized benefits, due, in part, to key data gaps (e.g., accepted concentration response functions for ozone effects on forests). | More comprehensive monetization will increase the benefits estimate. |
| **Section 608** | |
| The extent of compliance with recycling rates mandated by the Venting Prohibition is very uncertain. Therefore, the baseline assumptions regarding the percentages of CFCs that are actually recycled are also very uncertain. | A better ability to forecast recycling rates may either decrease or increase benefits estimates. |
| The baseline recycling value assumes no innovation in recycling technologies through 2017, which may be inaccurate. | Including innovation in recycling technologies may increase the benefits estimate. |
| **Section 611** | |
| Manufacturers' and consumers' responses to labeling rules are difficult to predict. | An enhanced ability to forecast their responses may either increase or decrease the benefits estimate. |
| Benefits attributed to labeling regulations may actually result from other circumstances as well. | Consequently, the benefits resulting from this rule may be less than the estimate included in the RIA. |
| Although some sectors may reduce the use of MCF as a result of the labeling rule, other sectors may increase their use of this substance. | A better ability to predict people's actions may decrease the benefits estimate. |

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# Valuation of Human Health and Welfare Effects of Criteria Pollutants

Appendix H

This appendix describes the derivations of the economic valuations for health and welfare endpoints considered in the benefits analysis. It includes three primary sections. First, we introduce the method for monetizing improvements in health and welfare. Second, we summarize dollar estimates used to value benefits and outline the derivation of each estimate. 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. Economic valuations are characterized in terms of a central (point) estimate as well as a probability distribution which reflects the uncertainty around the central estimate. Third, we present the results of the economic benefits analysis. All dollar values are in 1990 dollars. This third section concludes with an exploration of the uncertainties in valuing the benefits attributable to the CAAA.

## Methods Used to Value Health And Welfare Effects

The general approach to benefits analysis involves a three-step process— (i) identification of potential physical effects (i.e., individual health and welfare endpoints); (ii) quantification of significant endpoints; and (iii) monetization of benefits. The first two steps, identification and quantification of physical effects, are described in Appendix D, Human Health and Welfare Effects of Criteria Pollutants. The third step is detailed in this appendix. Monetization of benefits attributed to the CAAA involves applying dollar estimates obtained from economic literature to individual health and welfare endpoints relevant for the 812 prospective analysis. As context to understanding the methodology for transferring estimated values of physical effects, this section provides a brief discussion of the theoretical economic foundation of, and general approach to, valuing the benefits of improved air quality.

Economists equate the dollar value of a benefit to the level of well-being an individual enjoys from the provision or consumption of a particular good or composite good (i.e., bundle or mix of goods). A fundamental assumption in economic theory is that individuals can trade between different consumption levels of these goods, services, or money, and maintain the same level of welfare. Typically, this willingness to trade-off between goods is measured as willingness to pay (WTP) or willingness to accept compensation (WTA). These measures are essentially dollar equivalents to changes in the level of consumption of a good or service so that the individual maintains the same level of well-being. In other words, the individual is indifferent between his or her current bundle of goods and the alternative bundle of goods.

While WTP and WTA represent an individual's own assessment of the dollar value of better health, they are not necessarily equivalent measures.[1] WTP, in the case of health, is the largest amount of money a person would pay to obtain an improvement (or avoid a decline) in health. When faced with two

[1] The measures differ for several reasons. For example the measures have different points of reference from which to evaluate changes in welfare. WTP's reference point is the level of utility without the improvement. WTA's reference point is the level of utility with the improvement. Moreover, the measures have different upper bound constraints. WTP measures what a person would pay to obtain better health and is bound by the person's wealth and income. WTA, on the other hand, measures what a person must be paid to forego better health. WTA does not have an upper bound, but it must be at least as large as WTP. Economists, however, do not expect significant differences between WTP and WTA when the dollar amounts are small relative to the individual's wealth and income.

options, to either (1) pay a certain dollar amount to enjoy the health improvement or (2) abstain from paying the dollar amount and not experience the health improvement, the individual feels either choice provides the same degree of well-being. Alternatively, willingness to accept compensation (WTA) is the smallest amount of money a person would voluntarily accept as compensation to forego an improvement, or endure a decline, in health. The individual feels that to accept the payment and not experience the health improvement or refuse the compensation and experience improved health will provide the same degree of well-being. In practice, WTP is generally used to value benefits because it is often easier to measure and quantify.[2] In this report, we refer to all valuation estimates as WTP values, even though the underlying economic valuation literature is based on studies which elicited expressions of WTP and/or WTA.[3]

In the context of cost-benefit analysis, WTP is useful for estimating the monetary value of non-market, public goods. A major characteristic of public goods is that they are nonrival (i.e., one person's consumption of the good does not reduce the amount available to others). In the case of health-related improvements due to environmental quality, the benefits are also nonexclusive. Benefits are not (and to some extent, cannot be) regulated. As a result, the benefits are actually reductions in the probabilities or risk of enduring certain health problems. In theory, the total social value associated with the decrease in risk is

$$\sum_{i=1}^{N} (\textit{number of units of risk reduction})_i * (\textit{WTP per unit risk reduction}) \quad (1)$$

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. The units are in terms of cases reduced per unit of time (usually one year).

Using mortality risk as an example, suppose that a given reduction in PM concentrations results in lowering the risk of death by 1/10,000 per year. Then for every 10,000 individuals, one less death would be expected if ambient PM concentrations are reduced. If an individual's 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.

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 nonmarket 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 a respiratory illness) is further complicated by several factors, such as wealth, income, age, pre-existing health impairments, or other personal characteristics. There are many policy contexts where distinguishing among WTP estimates based on categorical differences (e.g., distinguishing between WTP of a low-income group and a high-income group) is controversial. Given the consideration of these influencing factors and the limitations on information available for developing WTP estimates, EPA sought to develop the most appropriate and accurate estimates possible. Derivations of the dollar value estimates for this study are discussed below.

[2]It is worth noting that the appropriateness of either WTP or WTA also depends on property rights. In the case of a policy aimed at reducing existing pollution levels, a WTP measure implicitly assumes that the property rights rest with the polluting firm. Alternatively, WTA measures implicitly assume that the property rights rest with the public. (Carson and Mitchell, 1993.)

[3]In some cases (e.g., hospital admissions), neither WTA nor WTP estimates are available. In those cases, cost of illness (COI) estimates are applied in lieu of WTP values. COI estimates understate the true welfare change since important value components (e.g., pain and suffering associated with the health effect) are not reflected in the out-of-pocket costs for the hospital stay.

## Valuation of Specific Health Endpoints

Since the Section 812 CAA retrospective analysis (U.S. EPA 1997), there have been significant advances made in economic valuation methodologies for both mortality and morbidity effects. Much of the literature presents emerging new approaches for characterizing the effects of potentially important determinants of WTP, such as age, income, risk perception, and current health status. Despite this progress, many of the more recent studies test techniques that are in the development stage and use data from work reviewed and incorporated in the Section 812 retrospective analysis. This section reviews the sources and methodology used to derive WTP estimates for premature mortality and a variety of morbidity effects valued in the present study. In addition, there are brief discussions of more recent advances relevant to particular endpoints.

### *Valuation of Premature Mortality Avoided*

The economic benefits associated with premature mortality were the largest category of monetized benefits in the Section 812 CAA retrospective analysis (U.S. EPA 1997).[4] In addition, EPA identified valuation of mortality benefits as the largest contributor to the range of uncertainty in monetized benefits. Because of the uncertainty in estimates of the value of premature mortality avoidance, it is important to adequately characterize and understand the various types of economic approaches available for mortality valuation. Such an assessment also requires an understanding of how alternative valuation approaches reflect that some individuals may be more susceptible to air pollution-induced mortality.

The health science literature on air pollution indicates that several human characteristics affect the degree to which mortality risk affects an individual. For example, some age groups are more susceptible to air pollution than others (e.g., the elderly and children). Health status prior to exposure also affects susceptibility – at risk individuals include those who have suffered strokes or are suffering from cardiovascular disease and angina (Rowlatt, et al. 1998).

To reflect the full range of knowledge of air pollution-induced mortality, an ideal estimate of mortality risk reduction benefits would be an *ex ante* willingness to pay (WTP) to improve one's own chances of survival plus WTP to improve other individuals' survival rates.[5] The measure would take into account the specific nature of the risk reduction commodity that is provided to individuals, as well as the context in which risk is reduced. To measure this value, it is important to assess how reductions in air pollution reduce the risk of dying from the time that reductions take effect onward, and how individuals value these changes. Each individual's survival curve, or the probability of surviving beyond a given age, should shift as a result of an environmental quality improvement. That is, changing the current probability of survival for an individual also shifts future probabilities of that individual's survival. This probability shift will differ across individuals because survival curves are dependent on such characteristics as age, health state, and the current age to which the individual is likely to survive. For example, Figure H-1 illustrates how a risk reduction may change a survival curve for a given population. In this figure, the solid line shows a survival curve for white males, from California 1980 life tables (adapted from Selvin, 1996), up to age 80. The dashed line shows that the probability of survival beyond a given age increases with a reduction in mortality risk.

[4] As noted in the methods section, 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."

[5] For a more detailed discussion of altruistic values related to the value of life, see Jones-Lee (1992).

While the change in a survival curve represents a cumulative effect of a change in risk over time, the annual change in risk of death represents a static effect of a risk reduction. As discussed in Appendix D in greater detail, the instantaneous risk of death at a specific age is often used to illustrate the effects of changes in risk. The annual risk of death is related to the probability of survival in that it represents the rate at which the survival probability changes at any given age, divided by the probability of surviving beyond that age. Figure H-2 shows how a constant risk reduction reduces annual risk of death across various age cohorts. The baseline risk of death increases with each cohort (solid line). As a result, the reduction in risk (in this hypothetical example a constant 25 percent reduction) lowers each cohorts' risk level at a different rate. The elderly experience a greater reduction in risk than younger cohorts as can be seen by the increasing difference between the solid and dashed line. It is important to note that this example shows the effect of a uniform risk reduction, and air pollution controls may have risk reduction effects that vary across age cohorts.

An alternative way to view the age-dependent effect of risk reduction is to consider changes in the cumulative effect of risk as measured by changes in remaining life expectancy. Remaining life expectancy is measured as the average number of additional years expected to be lived by those individuals alive at a given age, and derives from the area under the survival curve at any given age. The age-dependent effects of a hypothetical change in risk are portrayed in Figure H-3. Consider the effect of risk reduction on two cohorts, aged 10 years apart. When each cohort was at age 40 both had the same life expectancy shown in Figure H-3 as point A′. Given a risk reduction in the future that occurs when one cohort is at age 60 and the other at age 70, the life expectancy of the 60 year old increases by the amount A′B′, and the life

**Figure H-1**
**Hypothetical Survival Curve Shift**

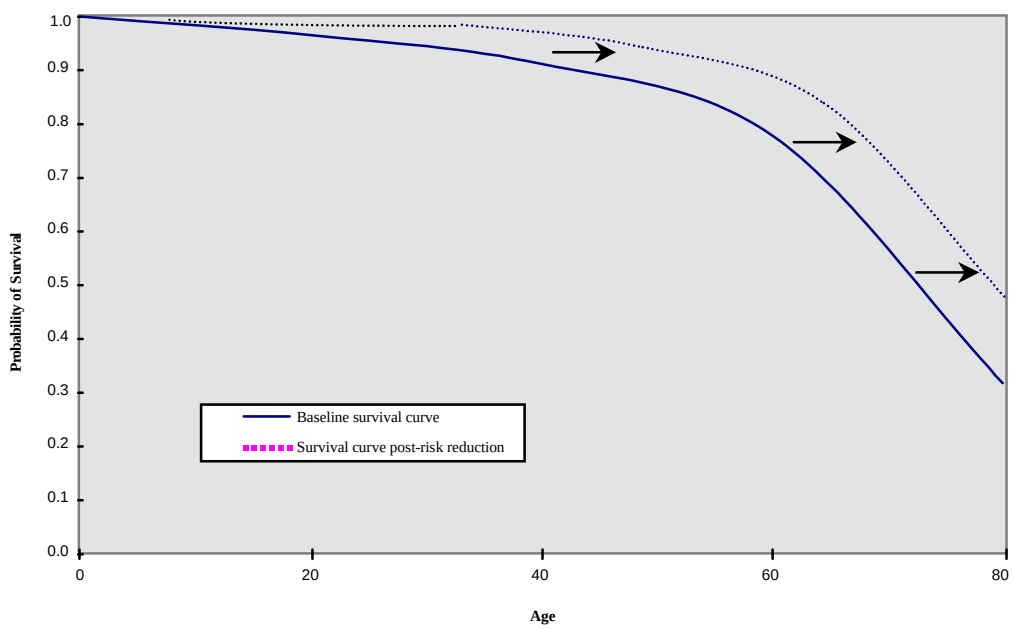


**Figure H-2**
**Change in 1990 Annual Risk of Death by 25 Percent**

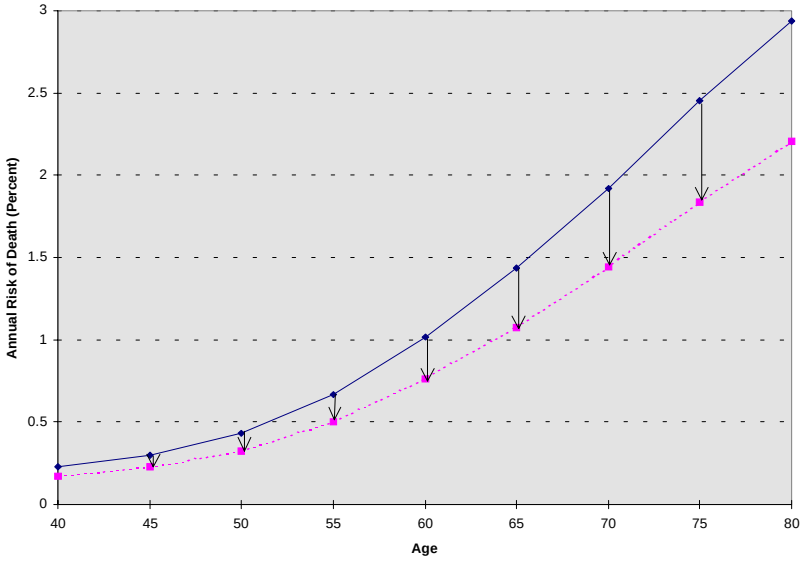
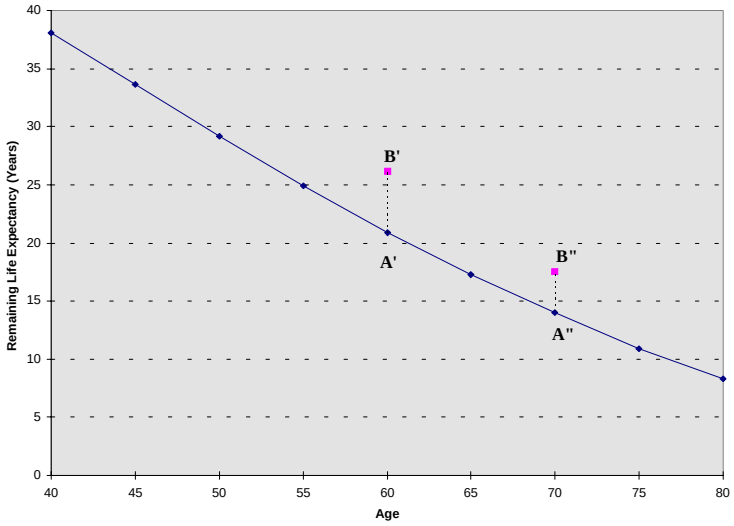



**Figure H-3**
**Increase in 1990 Remaining Life Expectancy**



expectancy of the 70 year old increases by the amount A′′B′′. The change in life expectancy is greater for the younger cohort than the older cohort because these measures represent a cumulative accrual of increased life expectancy (i.e., the younger cohort will benefit from the lower risk environment for more years).

Because the risk reduction results in various changes in risk levels, individual values for risk reduction are likely to vary as well. Some individuals having a greater change in risk, and hence life expectancy, may have different values for the change than those individuals experiencing a smaller change in risk. Note that future generations may hold values for health as well. Cropper and Sussman (1990) develop theoretical models formalizing these concepts when investigating how an individual's values for reduction of a future risk to oneself and to future generations should be discounted to the present.

While these theoretical models reflect the types of values necessary to estimate the impact of the CAAA, they are difficult to implement. First, they require an estimate of individuals' survival curves. In order to develop these survival probabilities, it is necessary to characterize the dose/response relationship for the regulated pollutants and know how this information varies with age and health states over time. Second, it is necessary to estimate values for risk reductions, considering the key dimensions in which risk and valuation of risk reduction may vary (e.g., with age and health state).

### *Mortality Valuation Methodologies*

This section summarizes alternative approaches to mortality risk valuation, and outlines the approach used to measure the economic value of these types of benefits for air pollution reductions associated with the CAAA. The first part provides background on the methods that individuals have developed to estimate the value of risk reduction benefits, including commonly-applied approaches to valuation as well as approaches that are beginning to be established in the risk valuation literature. The second part discusses the appropriateness of using these methodologies for assessing the economic value of mortality benefits associated with air pollution reduction. The Agency has concluded that recent advances in the literature show promise in incorporating several of the factors that are likely to influence value, but problems with the methodological approaches and lack of data needed to reliably to appropriately estimate values with the newer models leads us to adopt a value of statistical life approach for the primary estimate of air pollution-related mortality benefits.

### Commonly Applied Approaches

The preferred approach researchers have taken to estimate values for avoiding premature mortality is based on individual WTP for risk reduction. Although some cost-benefit analyses have based values on avoided lost earnings (i.e., the human capital approach), the WTP approach is preferred because it more closely conforms to economic theory.[6] The common WTP measures of the value of life-saving programs include the value of statistical life (VSL) and the value of a statistical life year (VSLY). Newer approaches to estimate values incorporate changes in life expectancy, risk of dying, life-days per person, and age-specific preferences. This section describes these approaches and discusses issues that arise in their application to estimate the value of mortality risk reduction benefits.

The most commonly applied approaches for mortality valuation are the value of statistical life and value of statistical life year. Both of these approaches

[6] In a recent article by Ireland and Gilbert (1998), the authors evaluate value of life estimates used in tort recovery cases. The article discusses the concept that for an individual there can be finite utility (or determined value) to life and at the same time no monetary equivalent. The authors do, however, build on this argument to demonstrate that existing value of life estimates are in fact lower bounds to the true value. By "lower bound," the authors refer to a value representative of a specific individual, not of a statistical life. In citing a reasonable value of life range, they use a range similar to that of the 812 retrospective analysis, although the authors do not cite the source of this range. Ireland and Gilbert write, "A decedent has lost something of immense value, for which estimates in the $4-$6 million range is clearly a low market value estimate".

directly address the value of premature death and health impairment. The VSL method measures the value of a given reduction in risk and an individual's WTP to reduce that risk, relying on wage and occupational risk tradeoff data or the results of contingent valuation surveys. Individual WTP amounts for small reductions in mortality risk are "standardized" to reflect reduction of population risk of one statistical life saved. The result of applying this method is not the value of an identifiable life, but instead the value of reducing fatal risks in a population (Viscusi 1992).

Viscusi (1992) summarizes the value of life literature, including almost forty studies providing VSL estimates relevant for policy application. For the section 812 retrospective analysis, EPA identified 26 studies from that review that reflect the application of the most sound and defensible methodological elements (see Table H-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 on estimates of the additional compensation demanded in the labor market for riskier jobs. Using a Weibull distribution to describe the distribution of the mean mortality risk valuation estimates from these studies, the mean estimate of the distribution is $4.8 million with a standard deviation of $3.2 million (1990$).

Since EPA's retrospective analysis, Desvousges *et al.* (1998) has conducted a meta-analysis of twenty-nine mortality studies presented in Viscusi (1993) and Fisher, Chestnut, and Violette (1989).[7] Desvousges *et al.'s* meta-analysis yields $3.3. million (1990 dollars) as a value of statistical life, with a 90 percent confidence interval between $0.4 and $6.3 million.[8] Their estimate, $3.3 million, falls well within the range generated by EPA's uncertainty analysis of VSL estimates. The selection of studies accounts for much of the difference between their analysis and EPA's. The Desvousges *et al.* analysis includes thirteen studies that EPA did not use and EPA includes ten studies omitted by Desvousges *et al.*

[7] In addition to the Viscusi (1993) study, the 812 retrospective examined two other studies, Miller et al. (1990) and the Fisher, Chestnut, and Violette (1989). We opted to not use the Miller et al. study given our concerns regarding the appropriateness of the selection of studies for valuing reductions in environment-related mortality risk and concerns about the adjustments made to the underlying data. The Fisher, Chestnut, and Violette (1989) study was not used because the data was not as current or comprehensive as the data in the Viscusi study.

[8] Desvousges *et al.* do not adjust the value of statistical life to account for age differences. They do note that a single estimate for the value of statistical life may not be a good representation of the differences between willingness-to-pay of the elderly and young, healthy workers. They state that Moore and Viscusi (1988) demonstrate that willingness-to-pay is higher for people with more life years to lose while Desvousges et al. (1996) and Johnson et al. (1998) indicate that willingness-to-pay is lower for people with limited abilities to engage in activities and care for themselves.

**Table H-1**
**Summary of Mortality Valuation Estimates**

| 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 |
| Garen (1988) | Labor Market | 13.5 |

SOURCE: Viscusi, 1992 and EPA analysis.

When applying VSL estimates to estimate mortality benefits, it is important to determine the differences between the nature of air pollution risk and risks faced by persons whose risk-dollar tradeoff decisions have been addressed in the literature. First, several studies indicate that the value people place on mortality risk reduction may depend on the nature of the risk (e.g., Fisher et al. 1989; Beggs 1984). Current VSL estimates do not account for a number of the important factors that affect risk perception. For example, premature mortality risks from air pollution are experienced on an involuntary basis and are generally uncompensated, while job-related risks are assumed by individuals who presumably have some choice as to occupation and are compensated for taking a riskier job. Second, the demographics of the population at risk from air pollution, particularly in

terms of age, income, and health state, may differ from the demographics of individuals surveyed in the literature. For a more detailed discussion of how these factors can affect the economic valuation of premature mortality, and specifically estimates derived from the VSL approach, see the discussion, "Benefits Transfer and VSL," presented in the section titled, "Uncertainties in the Valuation Estimates."

The VSLY method values life-years that would be lost if an individual were to die prematurely. Most commonly, VSLY estimates are an annualized equivalent of VSL estimates (Moore and Viscusi 1988, French and Mauskopf 1992). A VSLY estimate may imply a stream of constant values per year. The annualized VSLY estimate depends on three factors: the underlying VSL estimate; a discount rate; and the number of remaining life years implied by the underlying VSL estimate.

We develop an estimate of the value of a statistical life-year lost (VSLY) based on an approach suggested by Moore and Viscusi (1988). 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 also suggest that the typical respondent in a mortality risk study may have a life expectancy of an additional 35 years. Using the 35-year life expectancy and VSL estimate of $4.8 million, their approach yields an estimate of $137,000 per life-year lost or saved. In the prospective analysis, we also assume that an individual discounts future additional years. This implies that the value of each life-year lost must be greater than the non-discounted value. Assuming a five percent discount rate and adopting the above outlined approach, the implied value of each life year lost used in the prospective analysis is $293,000 (in 1990 dollars).

Critics note several disadvantages to using this type of VSLY method, most notably that the value of avoiding premature death depends on more than just lifespan. With the VSLY approach, the benefit attributed to avoiding a premature death depends directly on how premature it is – resulting in smaller values for older people, who have shorter life expectancies, and larger values for younger people.

While this approach attempts to derive age-adjusted values of expected life remaining using VSL estimates, it does not address potential differences in the value of a statistical life due to differences in the average age of the affected population or the average age at which an effect is experienced. Studies have shown that simple progressive declines in value as estimated with the VSLY method may be an oversimplification; in many cases, values for health peak several times throughout a lifetime (e.g., after having children, after retirement). In addition, in many cases, data restrictions limit researchers' ability to estimate VSLY because it is difficult to obtain estimates of age-specific risks and the number of life-years lost.

## Life Quality Adjustments

Another way to make adjustments to account for heterogeneity in value of life estimates is an approach that incorporates health status by applying a VSLY estimate (generated from the VSL literature) to an estimate of quality-adjusted life years (QALY). The resulting value estimates measure improvements in health based on individuals' attitudes toward symptoms or different levels of pain or physical impairment (Tolley et al. 1994). This approach utilizes survey techniques to rate different health conditions and adjust the number of life years lost to represent lost quality-adjusted life years. As a result, this approach aims to develop a value for a single QALY that is the same regardless of individual characteristics. In other words, the approach tries to standardize the measure of mortality risk reduction that emerges from a health effects analysis, making valuation more straightforward.

The Life Quality Adjustment approach may implicitly incorporate morbidity impacts to assess values for various causes of death, and is often used in health economics to assess the cost effectiveness of medical spending programs, to value morbidity avoidance, and to value mortality avoidance. Using a QALY rating system, health quality ranges from 0 to

1, where 1 may represent full health, 0 death, and some number in between (e.g., 0.8) an impaired condition. If an individual lives with a health quality index of 0.8, then the implied value of avoiding a year with this condition and having full health in its place would be 0.2 × VSLY. By the same token, the value of gaining an additional life year in this condition is 80 percent of the value of gaining a year in full health (i.e., 0.8 × VSLY) and represents an annual value for mortality risk avoidance for a person with the condition.

Tolley, et al. (1994) estimate values for a variety of health conditions using numerous techniques, including, in some cases, valuation of quality-adjusted life years. For example, when estimating values for acute and chronic symptoms using QALYs, the authors calculate low, medium and high value estimates based on a range of VSL estimates. Specifically, the authors use the following three VSLY estimates (1991$) for QALY valuation:

- Low Estimate = $70,000 VSLY: Derived from Miller, Calhoun and Arthur (1990) – VSL of $1.95 million, two percent discount rate.

- Medium Estimate = $120,000 VSLY: Derived from Miller, Calhoun and Arthur (1990) – VSL of $1.95 million, six percent discount rate.

- High Estimate = $175,000 VSLY: Derived from Moore and Viscusi (1988) – VSL of $6.0 million, 0 percent discount rate.

The authors multiply the VSLY estimate by the estimate of QALYs to calculate a value for each symptom. It is not clear from the analysis discussion which symptom values represent the application of this approach.

Cutler and Richardson (1998) apply a VSL estimate to an estimate of QALYs to measure the value of health improvements between 1970 and 1990 for ten health conditions. To do this, the authors use an VSLY estimate of $100,000, derived as the intermediate value of results reported in studies by Viscusi (1993) and Tolley et al. (1994). In addition, the authors estimate QALYs using information on disease prevalence in the US from 1970 to 1990, weighted by a factor that represents how quality of life for a given condition has changed over time (e.g., more buildings have ramps and elevators for individuals who have mobility problems, thus raising quality of life over time).

Murray and Lopez (1996) modify the above theoretical approach by deriving an estimate of disability-adjusted life years (DALYs). DALY estimates consider the years of life lost and years lived with disability, adjusted for the severity of the disability. The approach to estimate DALYs is similar to that used to estimate QALYs in that both incorporate judgments about the value of time spent in different health states. However, DALY and QALY estimation methods differ in that the methods to estimate DALYs are elicited from preferences for particular value choices using a specific standardized set of value choices.

The Life Quality Adjustment approach scales WTP values (VSL estimates) using a measure of life years that reflect heterogeneity in quality of health (QALYs). In many cases, the applied VSLY estimates do not reflect consistent use of VSL estimates or discount rates. In addition, in each of these valuation analyses health economists have constructed a scale or index that ranks health outcomes in terms of how adverse individuals believe them to be. Often, the extreme points on the scale are "perfect health" and "immediate death," but some applications allow for health outcomes that might be viewed as worse than death. These ranking methods do not yield estimates of WTP, and therefore are not linked to utility theory. It is not clear that the ranking of health outcomes obtained by these indices would match the ranking obtained by knowing individuals' WTP for various health effects. As discussed by Johansson (1995), these scales or indices rely on much more restrictive assumptions about the nature of individual preferences than are normally made in WTP studies.

## Longevity

Several recent efforts estimate values for an identifiable life by estimating the WTP for own life extension. Johannesson and Johansson (1996, 1997) estimate the WTP to increase one's life expectancy by one additional year (i.e., extending men's life expectancy from age 75 to 76, and women's from age 80 to 81, conditional on reaching age 75 or 80). Johannesson, Johansson, and Lofgren (1997) estimate the value of an immediate small reduction in mortality risk (a "blip" or one year of fatal risk prevention).

While this methodology represents a utility-theory based value, the value estimate for a single year of longevity does not exactly correspond to what is needed for an assessment of air pollution benefits. Johannesson and Johansson (1996, 1997) estimate a value for a single year of life extension near the end of one's lifetime – values at this age are likely to be low because of a low expectation of quality of life at this advanced age. It is likely that mortality values will vary within an individual's lifetime and with probability of survival. In addition, mortality associated with pollutant exposure will likely yield a longevity loss greater than one year (e.g., mortality associated with particulate matter yields an average longevity loss of approximately 14 life years among those who are afflicted). Moreover, because of the hypothetical nature of the contingent valuation method, it is unclear whether respondents accept the scenarios presented and whether enough context was provided to understand the risk and the budget implications of the scenario and the response.

## Cost Effectiveness

Garber and Phelps (1997) present a methodology for valuing a discounted life year that is determined by income and risk aversion in a life-cycle model. To calculate the optimal cost effectiveness cut-off for medical intervention, the authors assume values of a utility function, health production function, income, discount rate, and baseline mortality to derive a value equivalent to WTP for a discounted life year. In this model, utility is a function of income (less medical expenditures), and future income is a function of survival and medical expenditures. As a result, the authors use mortality rates to calculate expected income. Changes in these mortality rates result in changes in survival probabilities, and hence income. The model estimates an individual's willingness to trade income from one period to another; the discounted change in income is equivalent to WTP for a change in risk.

Although this methodology is based on a life-cycle model using survival probabilities, it is simplistic in its assumptions and is based on assumed preferences, rather than on revealed preferences or those stated by an individual. In effect, the model estimates values based largely on one empirical input: individual income. For example, the VSL for a 40 year-old cannot exceed $250,000 because that amount exceeds the discounted expected income. The largest value of discounted life-year obtained by the authors is approximately $37,000.

### *Valuation Strategy Chosen for this Analysis*

To estimate the economic value of mortality benefits associated with air pollution reductions, economic theorists prefer estimates that reflect *ex ante* values of reducing the risk of mortality across the population (i.e., for individuals having different health states and other characteristics such as income level and risk perception). This requires an estimate of an individual WTP for a reduction in an involuntary risk that will change individuals' survival probabilities for a lifetime. Developing a valuation estimate based on this theoretically ideal approach, however, is currently subject to significant data and methodological problems. Moreover, many of the valuation methods that are frequently presented as an alternative to the VSL approach rely on VSL estimates and calculate values that depend on lifespan data, which may be difficult to measure given the current health data limitations. Consequently, EPA's current interpretation of the state-of-the-art in premature mortality valuation leads to adoption of the VSL approach for development of the primary benefit estimate.

As discussed above, several different approaches for estimating a mortality-related value have been developed. Each, however, has either methodological inconsistencies with the preferred utility-based approach, or does not provide a value estimate for a commodity comparable to that provided by reduced air pollution. We summarize the potential problems of these alternatives below and in Table H-2:

- **Life Quality Adjustment:** This approach relies on VSL estimates applied to survey estimates of life-years (i.e., QALYs or DALYs) for the economic valuation. Currently, no generally accepted estimate or range of estimates of VSLY have been established, instead these values derive from various VSL studies and reflect numerous discount rates. In addition, the life years estimates require data sets that can account for the health states or utilities specific to a wide variety of health effects associated with air pollution. In many cases, these estimates are not available or are based on health professionals' perceptions of various health outcomes, and not necessarily based in economic utility theory.

- **Longevity:** The longevity valuation approach of Johannesson and Johansson (1996 and 1997) provides an estimate of the value for an identifiable one-year life extension. While the contingent valuation approach used may be consistent with utility theory, the commodity valued does not represent the commodity gained through improvement of ambient air quality.

- **Cost Effectiveness:** While the approach taken by Garber and Phelps relies on survival probabilities throughout an individual's lifetime, the methodology is based on a utility function that makes specific assumptions about individual preferences to measure WTP rather than eliciting value from either a revealed or stated preference approach. Moreover, this approach measures a WTP that is constrained by income. Where individual risks are small (perhaps one in ten thousand) relative to certain loss of life, individual WTP may also be small relative to income.

**Exhibit H-2**
**Summary of Alternative Methods for Assessing the Value of Reduced Mortality Risk**

| Method | Description | Strengths | Weaknesses | References |
|---|---|---|---|---|
| Value of Statistical Life (VSL) - hedonic wage studies | Uses wage and risk data to estimate WTP to avoid risk in the workplace | - Revealed preference<br>- Well-established approach: more than 60 primary studies | - Workplace risk context; working-age subjects and voluntary risk<br>- VSL may imply ex post risk | Summaries by Viscusi (1992) and others; many primary studies |
| VSL - contingent valuation studies | Uses survey responses to estimate WTP to avoid risks | - Flexible approach; some studies use environmental risk context<br>- Good data on WTP by respondent | - Risk information not well-understood by subjects; questions may be unfamiliar<br>- VSL may imply ex post risk | Summaries by Viscusi (1992) and others |
| VSL - consumer market studies | Uses consumer expense and risk data (e.g., smoke detectors) to estimate WTP to avoid risks | - Revealed preference<br>- Flexible approach | - Major difficulties estimating both risk and expense variables<br>- VSL may imply ex post risk | Summaries by Viscusi (1992) and others |
| Value of Statistical Life Year (VSLY) | Annual equivalent of VSL estimates | - Provides financially accurate adjustment for age at death | - Adjustment may not reflect how individuals consider life-years; assumes they have equal value for all remaining life-years | Viscusi and Moore (1988); French and Mauskopf (1992) |
| Quality Adjusted Life Year (QALY) | Applies quality adjustment to life-extension data, uses cost-effectiveness data to value | - Widely used in public health literature that assess different private medical interventions | - Lack of data on health state indices and life quality adjustments that are applicable to an air pollution context | Tolley (1994); Cutler and Richardson (1998) |
| WTP for change in survival curve | Reflects WTP for change in risk, potentially incorporates age-specific nature of risk reduction | - Theoretically preferred approach that most accurately reflects nature of risk reductions from air pollution control | - Almost no current literature<br>- Lack of available data due to the severe methodological difficulties in presenting complex risk data to subjects and eliciting reliable values | Cropper and Sussman (1990) |
| WTP for change in longevity | Uses stated preference approach to generate WTP for longevity or longer life expectancy | - Life expectancy is familiar term to most individuals | - Life expectancy is a simplifed term that does not incorporate age-specific risk information<br>-Methodological and data problems in attempting to adapt to air pollution context | Johannesson and Johansson (1997); Health Canada (1998) |
| Cost-Effectiveness | Develops a standard of comparison to measure the efficiency of various treatments in achieving a given health outcome | - Widely used in public health contexts | - Public health context may be for private goods (i.e., treatment)<br>- Dollar values do not necessarily reflect patient preferences | Garber and Phelps (1997) |

Note: WTP = willingness to pay

## Valuation of Hospital Admissions Avoided

The valuation of this benefits category reflects the value of reduced incidences of hospital admissions due to respiratory or cardiovascular conditions. We measure avoided hospital admissions as opposed to the number of avoided cases of respiratory or cardiovascular conditions, because of the availability of C-R relationships for the hospital admissions endpoint. Hospital admissions reflect a class of health effects linked to air pollution which are acute in nature but more severe than the symptom-day measures discussed below.

As described in Chapter 5, our approach to estimating the number of incidences for this category involves reliance on several concentration-response (C-R) functions. Each concentration-response function provides an alternative definition of either respiratory effects or cardiovascular effects, and defines alternative relationships between a single health affect and different pollutants. For the valuation of these incidences, the current literature provides well-developed and detailed cost estimates of hospitalization by health effect or illness. Using illness-specific estimates of avoided medical costs and avoided costs of lost work-time, developed by Elixhauser (1993), we construct cost of illness (COI) estimates that are specific to the suite of health effects defined by each C-R function. For example, we use twelve distinct C-R functions to quantify the expected change in respiratory admissions.[9] Consequently in this analysis, we develop twelve separate COI estimates, each reflecting the unique composition of health effects considered in the individual studies.

Because each epidemiology study defines a health effect by a group of ICD codes, we construct COI estimates for each study by aggregating estimates that are specific to an ICD code. These estimates use the following information reported by Elixhauser (1993): average hospital costs, average length of stay, and baseline incidences.[10] We use this ICD code information to develop valuation estimates that have two components, hospital charges and lost earnings due to the hospital stay. Our estimate of lost earnings due to time spent in the hospital is based on valuing the average length of hospital stay at a daily rate of $83. This daily rate is the median weekly wage divided by five work days and is based on U.S. Department of Commerce figures (1992). After developing values for each relevant ICD code (i.e., hospital costs plus lost earnings), we weight these values based on their prevalence in the baseline. The final COI estimate, specific to each study, is the sum of the weighted value of ICD code-specific estimates.

We use a Monte Carlo approach to combines the valuation and physical effects modeling to generate a benefits estimate for hospital admissions. This approach also allows us to account for the variability in costs due to alternative definitions of respiratory and cardiovascular conditions that result in a hospital admission. The Monte Carlo process for integrating the C-R function and its COI value involves first randomly selecting an estimated change in incidences from the suite of applicable C-R functions. For example, we use five epidemiology studies for the endpoint hospital admissions due to cardiovascular effects, and develop COI estimates specific to each study. The Monte Carlo modeling then selects the COI estimate specifically developed for that C-R function. These values are multiplied to generate a single benefits estimate for reduced hospital admissions. This process is repeated so that the value from each iteration is collected to generate a distribution that characterizes the range and probability of possible benefits estimates. The primary benefit estimates of avoided cardiovascular-related hospital admissions reflect the central value of this distribution.

The use of COI estimates suggests we are likely to significantly underestimate the WTP to avoid hospital

[9]For more detailed discussion of the various health effects considered by each C-R function and methodology for estimating the number of avoided hospital admissions, see Appendix D.

[10]Potential illnesses associated with respiratory and cardiovascular admissions were identified by ICD-9 code.

admission. The valuation of any given health effect, such as hospitalization, should reflect the value of avoiding associated pain and suffering and lost leisure time, in addition to medical costs and lost work time. While the probability distributions in this analysis characterize a range of potential costs associated with hospitalization, they do not account for the omission of factors from the COI estimates, such as pain and suffering. Consequently, the valuations for these endpoints most likely understate the true social values for avoiding hospital admissions due to respiratory or cardiovascular conditions.

### Valuation of Chronic Bronchitis Avoided

In this analysis, chronic bronchitis is one of the two monetized morbidity endpoints whose effects 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 therefore incorporates the present discounted value of a potentially long stream of costs (e.g., medical expenditures and lost earnings) and reduced health-state utility.[11]

Two studies, Viscusi *et al.* (1991) and Krupnick and Cropper (1992) provide estimates of WTP to avoid a case of chronic bronchitis. While alternative estimates exist, many are derived from these two primary studies.[12] The study by Viscusi *et al.* uses a sample that is larger and more representative of the general population, while the Krupnick and Cropper study solicits values only from individuals who have a relative with the disease. As a result, the valuation of chronic bronchitis is based on the distribution of WTP responses from Viscusi *et al.* (1991).

Both the Viscusi *et al.* and the Krupnick and Cropper studies estimate the WTP to avoid a severe case of chronic bronchitis (CB). The incidence of pollution-related chronic bronchitis, however, is based on three studies which consider only new incidences of the illness and the resulting severity is unknown.[13] In response to the uncertainty regarding how the severity of a new case may progress, the prospective analysis adjusts Viscusi *et al.*'s WTP estimates downward. This adjustment reflects the decrease in severity of a case of pollution-related CB relative to the case in the Viscusi study and the elasticity of WTP with respect to severity. The elasticity of WTP to avoid CB is a marginal value and not unit elastic (i.e., not equal to one). Consequently, WTP adjustments are made in one percent increments. At each step, the WTP specific to a given CB severity level *(sev)*, is adjusted to derive the WTP to avoid a case with a one percent lower level of severity by calculating ( 0.99**sev*).[14] In this analysis, we derive an estimate of WTP for a case of chronic bronchitis that represents a 50 percent reduction in the severity described in the Viscusi study. The iterative procedure continues until the severity is half of the of the Viscusi value.

With the downward adjustment to Viscusi *et al.*'s WTP estimate, calculating the WTP to avoid a case of

[11] 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 in this analysis.

[12] For examples of alternative estimates see Desvousges *et al.* (1998) and Tolley *et al.* (1994). Both studies present estimates of avoiding one year of chronic bronchitis that are based on adjusting values from either Viscusi *et al.* (1991) or Krupnick and Cropper (1992).

[13] The three studies are Abbey *et al.* (1993), Abbey *et al.* (1995) and Schwartz (1993). For more discussion of estimating the number of avoided cases of chronic bronchitis see Appendix D, Human Health Effect of Criteria Pollutants. Incidences are predicted separately for each year during the period 1990-2010. It is important that only new cases of chronic bronchitis are considered in this analysis because WTP estimates reflect lifetime expenditures and lower utility associated with the illness. If the total prevalence of chronic bronchitis, rather than the incidence of only new chronic bronchitis were predicted each year, valuation estimates reflecting lifetime losses 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.

[14] Note that the elasticity changes at each iteration because the elasticity of WTP with respect to severity is a function of severity.

pollution-related chronic bronchitis has three components, each introducing some uncertainty. The components are (1) WTP to avoid a case of severe CB, (2) the severity level of an average pollution-related case of CB relative to that of the severe case, and (3) the elasticity of WTP with respect to severity. Based on assumptions about the distributions of each component's value, a distribution of WTP to avoid a pollution-related case of CB is derived by Monte Carlo methods. Each of the three underlying distributions is described briefly below.

The distribution of WTP to avoid a severe case of CB is based on the distribution of WTP responses in the Viscusi study. Viscusi *et al.* derived an implicit WTP to avoid a statistical case of chronic bronchitis from respondents' WTP for a specified reduction in risk. The mean response implied a WTP of about $1 million (1990 dollars); the median response implied a WTP of about $530,000 (1990 dollars).[15] Viscusi *et al.* report the mean and median of their distribution of WTP responses and the decile points. The distribution of reliable WTP responses from the Viscusi study can therefore be approximated by a discrete distribution, assigning equal probability to each of the first nine decile points (or one-ninth probability to each decile). This method omits five percent of the responses from each end of the distribution (i.e., the extreme tails which are considered unreliable). Our present study uses this trimmed distribution of Viscusi *et al.*'s WTP responses, for which the mean is $720,000 (1990 dollars), as the distribution of WTPs to avoid a severe case of CB.

The distribution of the severity level of an average case of pollution-related CB is based on the severity levels used in Krupnick and Cropper's study, which estimates the relationship between severity level and the natural log of WTP. The distribution is triangular with a mean of 6.5 and endpoints at 1.0 and 12, although the most severe case of CB in that study is assigned a severity level of 13.[16]

The elasticity of WTP to avoid a case of CB with respect to the severity of the case equals a constant times the severity level. This constant, estimated in Krupnick and Cropper's study of the relationship between severity and the natural log of WTP, is normally distributed with mean of 0.18 and standard deviation of 0.0669.

Using distributions of the three WTP components described above, the Monte Carlo analysis generates a distribution with a mean of $260,000 for WTP to avoid a pollution-related case of CB. Consistent with economic theory, the COI estimates generated by Cropper and Krupnick (1990) are lower than the mean WTP estimate (i.e., COI does not reflects the desire to avoid pain and suffering).[17] These COI estimates are approximately $86,000 for a 30 year old, $84,000 for a 40 year old, $76,000 for a 50 year old, and $43,000 for a 60 year old (in 1990 dollars). The prospective's WTP estimate is 3 to 6 times greater than the full COI estimate for 30 year olds and 60 year olds, respectively.

## Valuation of Chronic Asthma Avoided

Chronic asthma is the other morbidity endpoint that is valued as a health condition lasting throughout an individual's lifetime. The number of new cases of chronic asthma is based on a study by McDonnell *et al.* (1999), and specifically examines the effects of ozone as a potential cause of the illness among adult males (i.e., ages 27 and older). Similar to the valuation of chronic bronchitis, WTP to avoid chronic asthma

[15] 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.

[16] The Krupnick and Cropper study bases its most severe case of CB (i.e., severity level equal to 13) on that used in the Viscusi study.

[17] 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, Cropper and Desvousges calculate several estimates of the present value of the stream of medical expenditures and lost earnings associated with an average case of chronic bronchitis.

is presented as the net present value of what would potentially be a stream of costs and lower well-being incurred over a lifetime.

Estimates of WTP to avoid asthma are provided in two studies, one by Blumenschein and Johannesson (1998) and one by O'Conor and Blomquist (1997). Both studies use the contingent valuation method to solicit annual WTP estimates from individuals who have been diagnosed as asthmatics. Each study, however, applies a different valuation approach. Blumenschein and Johannesson solicit WTP values by asking dichotomous choice and open-ended bidding game questions. They report an average monthly WTP of $162 which amounts to an annual value of approximately $1,900 (1990 dollars). Alternatively, O'Conor and Blomquist apply a risk-risk tradeoff approach similar to that used in the chronic bronchitis studies. They calculate $1,200 (1990 dollars) as the average annual WTP to avoid asthma.

To maintain consistency between the health effects modeling and the valuation, the WTP estimates were adjusted to account for two factors. As mentioned earlier, valuation of chronic morbidity endpoints should approximate the costs and lowered health-state utility that are incurred over an individual's lifetime. We assume that the health condition does not affect the average life expectancy of an individual (i.e., does not cause premature mortality). Recognizing that the average life expectancy will vary with different age groups and that each age group does not represent an equal portion of the population, the present discounted stream of WTP is calculated for seven different age cohorts (between the ages 27 and 85). In turn, the net present value for each age group is weighted by that age category's representative share of the total population. This calculation was performed for the mean WTP estimates presented in the two studies. The central estimate of WTP to avoid a case of chronic asthma among adult males, approximately $25,000, is the average of the present discounted value from the two studies. The analysis characterizes the uncertainty around this estimate by applying upper and lower values based on the present discounted value derived from each study, $19,000 derived from O'Conor and Blomquist study and $29,000 from the Blumenschein and Johannesson study.

## Valuation of Other Morbidity Endpoints Avoided

The valuation of a specific short-term morbidity endpoint is generally solicited by representing the illness as a cluster of acute symptoms. For each symptom, the WTP is calculated. These values, in turn, are aggregated to arrive at the WTP to avoid a specific short term condition. For example, the endpoint lower respiratory symptoms (LRS) is represented by two or more of the following symptoms: runny or stuffy nose; coughing; and eye irritation. The WTP to avoid one day of LRS is the sum of values associated with these symptoms. The primary advantage of this approach is that is provides some flexibility in constructing estimates to represent a variety of health effects.

At the time of the Section 812 retrospective analysis there were only a small number of available studies on which to base estimates (two or three studies, for some endpoints; only one study for others). Since the retrospective analysis, much of the literature suggests there are developing approaches that may eventually lead to the refinement of estimates and the overcoming of some limitations to the current approach to constructing values. For example there is extensive progress in developing valuation techniques that reflect an individual's current health state and more accurately account for a symptoms's attributes (i.e., duration and severity).

There are several aspects of the short-term morbidity valuation estimates worth noting. First, estimates of WTP may be understated for at least two reasons. 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. In addition, 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. The second point

worth noting is that the total benefit attributed to the reduction of particular pollutant's concentration is determined largely by the benefit associated with its corresponding reduction in mortality risk. This is largely due to the dollar value associated with mortality which is significantly greater than any other valuation estimate. More detailed explanations for valuation of specific morbidity endpoints are given in Table H-3. The table summarizes the sources and derivation of the economic values used in the analysis.

## Valuation of Welfare Effects

Economic valuations for welfare effects quantified in the analysis (i.e., visibility and worker productivity) are documented in Table H-3.[18] Worker productivity, unlike the avoidance of work loss days or restricted activity days, reflects productivity benefits due to improvements in work conditions (i.e., reduced ambient ozone) rather than health improvements (i.e., reduced risk of hospitalization). It is measured in terms of the reduction in daily income of the average worker engaged in strenuous outdoor labor and estimated at $1 per ten percent increase in ozone concentration. (Crocker and Horst, 1981). We discuss the derivation of the visibility valuation further below.

### *Visibility Valuation*

Since the late 1970s, a number of contingent valuation (CV) studies of visibility changes have been published in the economics literature. These studies often classify visibility benefits as either residential or recreational. CV studies of residential visibility generally survey individuals in urban and suburban settings. The valuation is also applicable to households in rural areas. Residential values relate to the impact of visibility changes on an individual's daily life (e.g., at home, at work, and while engaged in routine recreational activities). Benefits of recreational visibility relate to the impact of visibility changes manifested at parks and wilderness areas that are expected to be experienced by its visitors. Recreational visibility benefits may, however, reflect the value an individual places on visibility improvements regardless of whether or not the person plans to visit the park.[19]

The reported estimates, expressed as household willingness to pay (WTP) for a hypothesized improvement in visibility, have a wide range of values. For examples, studies of visibility values from western cities have reported somewhat lower values than those from eastern cities. This difference raises the question of how visibility benefits should be evaluated with respect to location (e.g., eastern U.S. versus western U.S.), commodity definition (e.g., changes in recreational areas versus residential areas), and units of measurement (e.g., visual range, light extinction, and deciview). While the differing values reported in the literature may appear to imply that visibility is valued differently in the eastern and western U.S., other evidence suggests that eastern and western visibility are not fundamentally different commodities. For example, NAPAP data indicates 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). These

[18] In valuing welfare effects, the retrospective analysis included the benefits of reduced household soiling. This valuation was based on 1972 data that projected expenditure patterns from 1972 to 1985 (Manuel *et al.*, 1982). While this study was appropriate for the twenty year time period of the retrospective (1970 to 1990), it is of questionable applicability for the current study. Since the original study, there have been alternative estimates of benefits due to reduced soiling. These estimates, however, continue to be based on the original study and its underlying data (e.g., Desvousges et al., 1998). Consequently, these valuation coefficients do not reflect more recent information on air pollution composition and potentially significant changes in patterns of household expenditure and allocation. Progress in the valuation of this category's benefits is further limited by the challenges of developing dose-response functions that accurately assess the level and rate of materials damage and soiling. Recent literature does suggest there is progress in refining approaches, although it has not quite advanced to the level necessary for credible quantification or monetization of benefits associated with reduced materials damage and soiling.

[19] This type of valuation is typically labeled "existence value." For more discussion see Chestnut and Rowe, 1990.

results suggest that the valuation of marginal visibility changes is dependent on baseline conditions and proximity to the commodity being valued (e.g., improved visibility in a region with an abundance of National Parks such as the Pacific Northwest). Returning to the NAPAP example, the similarity in values may reflect the similarities between baseline visibility in eastern and western coastal zones (i.e., coastal areas typically have higher humidity, while areas of the west tend to have lower humidity and hence a greater baseline visibility).

For the purposes of this report, we interpret recreational settings applicable for this category of effects to include National Parks throughout the nation. Other recreational settings may also be applicable, for example National Forests, state parks, or even hiking trails or roadside areas with scenic vistas. In those cases, a lack of suitable economic valuation literature to identify these other areas and/or a lack of visitation data prevents us from generating estimates for those recreational vista areas. Moreover, we develop estimates of recreational visibility changes that account for the tendency of individuals to value visibility changes based on proximity to the National Park.

We estimate visibility benefits based on a derived visibility valuation function. In both cases, residential and recreational visibility, the valuation function takes the following form:

$$HHWTP = B * ln(VR1/VR2)$$

where:

HHWTP = annual WTP per household for visibility changes

VR1 = the starting annual average visual range

VR2 = the annual average visual range after the change in air quality

B = the estimated visibility coefficient.

The form of this valuation function is designed to reflect the way individuals perceive and express value for changes in visibility. In other words, the expressed WTP for visibility changes varies with the percentage change in visual range, a measure that is closely related to, though not exactly analogous to, the Deci View index used in Chapter 4.

We develop estimates of the visibility coefficients for residential and recreational visibility from two studies.[20] We use figures reported in Chestnut and Dennis (1997) for the valuation of residential visibility. This study publishes estimates of visibility benefits for the Eastern U.S that are based on original research conducted in two Eastern cities (Atlanta and Chicago) by McClelland et al. (1990). We use a central B coefficient for residential visibility of $141, as reported in Chestnut and Dennis (1997). For the valuation of recreational visibility benefits, we use a study by Chestnut and Rowe (1990). This study reports WTP estimates of recreational visibility in three park regions, the Western, Southwestern, and Eastern U.S. For recreational visibility, the coefficients vary based on the study region and whether the household is within or outside of the National Park region of concern. "In-region" coefficients are higher than those for "out-of-region" households. The "in-region" estimates for California, the Southwest, and Southeast are $105, $137, and $65, respectively; the corresponding "out-of-region" estimates are $73, $110, and $40, respectively.

Our valuation of visibility changes is largely based on unpublished, but peer-reviewed work. For example, we use the secondary analysis of Chestnut and Dennis (1997) to value residential visibility benefits. This article is published in the *Journal of Air and Waste Management Association*, but relies on the unpublished results reported by McClelland et al. (1990). The source of our recreational visibility estimates, Chestnut and Rowe (1990), is also unpublished. Both studies were originally developed as part of the National Acid Precipitation Assessment Program (NAPAP) and, therefore, have been subject to peer-review as part of that program. Moreover, these two studies are frequently cited and

[20] The unit of measure for the visibility coefficients is dollars. However, these coefficients are scaled by the small incremental changes in visibility to generate our WTP estimates.

recommended for use in published analyses of visibility valuation.[21]

Concerns about the method used in the McClelland et al. study, however, suggest their results may not incorporate two potentially important adjustments. First, their study does not account for the "warm glow" effect, in which respondents may provide higher willingness to pay estimates simply because they favor "good causes" such as environmental improvement. Second, while the study accounts for non-response bias, it may not employ the best available methods. The effect of both these factors is to suggest an overestimate of WTP. As a result, we exclude residential visibility estimates from the overall primary benefits estimate.

[21]For example see Desvousges et al. (1998).

**Table H-3**
**Unit Values Used for Economic Valuation of 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. = 3,240,000 | Central Estimate: Value is the mean of value-of-statistical-life estimates from 26 studies (5 contingent valuation and 21 labor market studies).<br><br>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 Estimate: 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).<br><br>Uncertainty: Assuming the discount rate is five percent, and assuming an expected 35 years remaining to the average 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. Because the VSL is expressed as a Weibull distribution, as indicated above, the value of a statistical life-year is also expressed as a Weibull distribution, with mean equal to 0.061 multiplied by the mean of the original Weibull distribution (0.061 x $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). |

| 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 Estimate: 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 (as described in Viscusi et al., 1991) to avoid a severe case of CB for the difference in severity and taking into account the elasticity of WTP with respect to severity of CB. The mean of the resulting distribution is $260,000.<br><br>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 distributed with mean = 0.18 and standard deviation = 0.0669 (from Krupnick and Cropper, 1992). See text for further explanation. |
| **Chronic Asthma** | $25,000 | Triangular distribution, centered at $25,000 on the interval [$19,000, $30,000] | Central Estimate: Based on results reported in two studies (Blumenschein and Johannesson, 1998 and O'Conor and Blumquist, 1997). Assumes a 5% discount rate and reflects adjustments for age distribution among adults (ages 27 and older) and projected life years remaining.<br><br>Uncertainty: Reflects the range in central estimate values reported in the two studies. |

| Health or Welfare Endpoint | Estimated Value Per Incidence (1990$) | | Derivation of Estimates |
|---|---|---|---|
| | Central Estimate | Uncertainty Distribution | |
| **Hospital Admissions** | | | |
| 1. All Respiratory<br><br>- ICD codes: 460-519 | variable—function of the analysis | See Derivation of Estimates | Central Estimate: Central estimate is the result of the analysis. The analysis uses 12 distinct C-R functions. A COI estimate is constructed for each. The COI estimates are based on ICD-9 code level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total respiratory illnesses) reported in Elixhauser (1993).<br><br>Uncertainty: Probability distribution is a result of the analysis and reflects: (1) uncertainty range of C-R function outcome; and (2) variation in study-specific COI estimates. |
| 2. All Cardiovascular<br>- ICD codes: 390-429 | variable—function of the analysis | See Derivation of Estimates | Central Estimate: Central estimate is the result of the analysis. The analysis uses five distinct C-R functions. A COI estimate is constructed for each. The COI estimates are based on ICD-9 code level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total respiratory illnesses) reported in Elixhauser (1993).<br><br>Uncertainty: Probability distribution is a result of the analysis and reflects: (1) uncertainty range of C-R function outcome; and (2) variation in study-specific COI estimates. |
| 3. Emergency room visits for asthma | $194 | Triangular distribution, centered at $194 on the interval [$144, $269] | Central Estimate: COI estimate based on data reported by Smith et al. (1997).<br><br>Uncertainty: Based on reported 95% confidence intervals for annual estimates of the number and costs of ER visits. |

| Health or Welfare Endpoint | Estimated Value Per Incidence (1990$) | | Derivation of Estimates |
|---|---|---|---|
| | Central Estimate | Uncertainty Distribution | |
| **Respiratory Ailments Not Requiring Hospitalization** | | | |
| 1. Upper Resp. Symptoms (URS)<br><br>(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 Estimate: Combinations of the 3 symptoms for which WTP estimates are available that closely match those listed by Pope et al. result in 7 different "symptom clusters," each describing a "type" of URS. A dollar value was derived for each type of URS, using IEc mid-range estimates of WTP to avoid each symptom in the cluster and assuming additivity of WTPs. The dollar value for URS is the average of the dollar values for the 7 different types of URS.<br><br>Uncertainty: Assumed to be a continuous uniform distribution across the range of values described by the 7 URS types. |
| 2. Lower Resp. Symptoms (LRS)<br><br>(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 Estimate: 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.<br><br>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 Estimate: Average of low and high values recommended by IEC for use in section 812 analysis (Neumann et al., 1994).<br><br>Uncertainty: Continuous distribution between low and high values (Neumann et al., 1994) assigns equal likelihood of occurrence of any value within the range. |

| Health or Welfare Endpoint | Estimated Value Per Incidence (1990$) | | Derivation of Estimates |
|---|---|---|---|
| | Central Estimate | Uncertainty Distribution | |
| 4. Acute Respiratory Symptoms and Illnesses<br><br>- Presence of any of 19 acute respiratory symptoms<br><br>- Any Resp. Symptom<br><br>- Respiratory Illness | $18 | 1. URS, probability = 40%<br>LRS, probability = 40%<br>URS+LRS, prob. = 20%<br><br>2. If URS, use URS $ dist.<br>If LRS, use LRS $ dist.<br>If URS+LRS, randomly select one value each from URS and LRS $ distributions; sum the two | Central Estimate: 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.<br><br>Uncertainty: Based on variability assumed for central estimate, and URS and LRS uncertainty distributions presented previously. |
| 5. Asthma Attack | $32 | Continuous uniform distribution over the interval [$12, $54] | Central Estimate: 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.<br><br>Uncertainty: Based on the range of values estimated for each of the four severity definitions. |
| 6. Moderate or worse asthma | $32 | Continuous uniform distribution over the interval [12, 54] | Central Estimate: Reflects the mean WTP to avoid a "bad asthma day" as reported by Rowe and Chestnut (1986).<br><br>Uncertainty: Taken to be a continuous uniform distribution across the range of values obtained from the study. |
| 7. Shortness of breath, chest tightness or wheeze | $5.30 | Continuous uniform distribution over the interval [$0, $10.60] | Central Estimate: 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).<br><br>Uncertainty: Taken to be a continuous uniform distribution across the range of values obtained from the two studies. |

| Health or Welfare Endpoint | Estimated Value Per Incidence (1990$) | | Derivation of Estimates |
|---|---|---|---|
| | Central Estimate | Uncertainty Distribution | |
| **Restricted Activity and Work Loss Days** | | | |
| 1. WLDs | $83 | none available | Central Estimate: Median weekly wage for 1990 divided by 5 (U.S. Department of Commerce, 1992)<br><br>Uncertainty: Insufficient information to derive an uncertainty estimate. |
| 2. MRADs | $38 | Triangular distribution centered at $38 on the interval [$16, $61] | Central Estimate: 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).<br><br>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. |
| **Welfare Effects** | | | |
| 1. Visibility<br><br>Residential Visibility<br>"in-region"<br>"out-of-region" | Valuation function:<br><br>HHWTP= B * ln(VR1/VR2)<br><br>where:<br>HHWTP = annual WTP per household<br>B = estimated visibility coefficient<br>VR1 = starting annual average visual range<br>VR2 = the annual average visual range after the change in air quality | | Central Estimate: Estimated WTP for valuation of visibility changes depend upon two factors: (i) visibility coefficient, B, and (ii) incremental change in visual range. Visibility coefficients applied in the primary analysis vary by category of visibility change and region. Recreational visibility valuation is based on Chestnut and Rowe (1990). For "in region" recreational visibility, the coefficients are $105, $137, $65, for California, the Southwest, and the Southeast, respectively. For "out-of-region" recreational visibility, the coefficients are $73, $110, $40, for California, the Southwest, and the Southeast, respectively. |
| 2. Worker Productivity | Change in daily wages: $1 per worker per 10% change in $O_3$ | none available | Central Estimate: Based on elasticity of income with respect to O3 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). |

Note: All WTP estimates converted to 1990 dollars using the Consumer Price Index (CPI); COI estimates converted using the CPI-Medical.

## Results of Valuation of Health and Welfare Effects

We estimate total human health and welfare benefits by combining the economic valuations described in this Appendix with the health and welfare effects results presented in Appendix D for projection years 2000 and 2010. The valuation results reflect the annual estimates of benefits for the 48 contiguous States, or "all U.S. population," which provides a more accurate depiction of the *trend* of economic benefits over the 20-year study period [22] For our Primary Central estimate we attribute to Titles I through V of the CAAA total annual human health benefits of $68 billion in 2000 and $118 billion in 2010.

As noted in Appendix D, we also include alternative estimates for some health and welfare impacts, which form the basis of several alternative benefit estimates. For each of the health effects estimates, we quantify statistical uncertainty. The range of estimated health and welfare effects, along with the uncertain economic unit valuations, are combined to estimate a range of possible results. We use the Monte Carlo method presented in Chapter 8 to combine the health and economic information. Both tables show 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. We summarize our primary estimates of 2000 and 2010 monetized benefits in Table H-3 and Table H-5, respectively. The tables provide our Primary Central estimate, in addition to our Primary Low estimate, 5th percentile values, and our Primary High estimate, 95th percentile estimates, for each benefit category.

We also apply the Monte Carlo method when generating aggregate monetized benefit results. The Monte Carlo method used in the analysis assumes that each health and welfare endpoint is independent of the others. We adopt this approach in response to the very low probability that the aggregate benefits will equal the sum of the fifth percentile benefits from each of the ten endpoints. Consequently, the upper and lower fifth percentiles of the estimated benefits from the individual endpoints does not equal the estimated totals for the Primary High and Primary Low estimates.

There are two additional aspects of our results that warrant discussion. The first is the valuation of premature mortality due to PM exposure. The second is our strategy to avoid double-counting when aggregating health benefits. As discussed in Chapter 5, premature mortality is estimated based on PM exposure. Our primary estimates reflect a lag between PM exposure and the timing of premature mortality. While this lag does not alter the number of estimated incidences, it does alter the monetization of benefits. Because we value the "event" rather than the present change in risk, the value of avoided future premature mortality should be discounted. Therefore, the type of lag structure employed plays a direct role in the valuation of this endpoint.

The primary analysis reflects a five-year lag structure. Under this scenario, 50 percent of the estimated cases of avoided mortality occur within the first two years. The remaining 50 percent are then distributed across the next three years. Our valuation of avoided premature mortality applies a five percent discount rate to the lagged estimates over the periods 2000 to 2005 and 2010 to 2015. We discount over the period between the initial PM exposure change (either 2000 or 2010) and timing of the projected incidences.

[22] In Appendix D, we present physical effect estimates for affected population in the contiguous 48 States and for affected populations within 50 kilometers of a monitor. We present those results as a sensitivity test that characterizes the possible magnitude of human health effects. For the purpose of assessing the total benefit of the CAAA, the results affecting populations in 48 states provide a better characterization of the total direct benefits than do the "monitored area only" results. The results of only monitored areas does not account for the benefits of air quality improvements affecting approximately 25 percent of the population. The "all U.S. population" results, however, 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.

Many of the monetized health benefit categories include overlapping health endpoints, creating the potential for double-counting. In an effort to avoid overstating the benefits, we do not aggregate all of the quantified health effects. For example, "asthma attacks" and "moderate to worse asthma", are all considered components of the endpoint, "Any of 19 Respiratory Symptoms". Consequently, we present the results but do not include them in our reported total benefits figures. In other cases, there are endpoints included in our aggregation of benefits that appear to have overlapping health effects. For those benefit categories that describe similar health effects, it is important to keep in mind that estimated incidences are based on unique portions of the population.

**Table H-4**
**Primary Estimates of Health and Welfare Benefits Due to Criteria Pollutants in 2000**

| | Monetary Benefits (in millions 1990$) | | |
|---|---|---|---|
| **Benefits Category** | **5th %ile** | **Mean** | **95th %ile** |
| **Mortality** | | | |
| Ages 30+ | $ 8,600 | $ 63,000 | $ 150,000 |
| **Chronic Illness** | | | |
| Chronic Bronchitis | $ 220 | $ 3,600 | $ 11,000 |
| Chronic Asthma | 29 | 140 | 240 |
| **Hospitalization** | | | |
| All Respiratory | $ 46 | $ 78 | $ 120 |
| Total Cardiovascular | 53 | 200 | 430 |
| Asthma-Related ER Visits | 0.1 | 0.6 | 1.8 |
| **Minor Illness** | | | |
| Acute Bronchitis | $ 0 | $ 1.3 | $ 3.3 |
| URS | 2.8 | 12 | 26 |
| LRS | 1.4 | 3.9 | 7.2 |
| Respiratory Illness | 0.4 | 2.5 | 6.1 |
| Mod/Worse Asthma[1] | 1.2 | 8.5 | 19 |
| Asthma Attacks[1] | 13 | 35 | 66 |
| Chest tightness, Shortness of Breath, or Wheeze | 0 | 0.5 | 2.4 |
| Shortness of Breath | 0 | 0.3 | 0.7 |
| Work Loss Days | 180 | 210 | 240 |
| MRAD/Any-of-19 | 420 | 760 | 1,100 |
| **Welfare** | | | |
| Decreased Worker Productivity | $ 460 | $ 460 | $ 460 |
| Visibility Recreational | 1,700 | 2,000 | 2,300 |
| Agriculture | 46 | 450 | 860 |
| **Total Benefits[2]** | | **$ 71,000** | |

Note:
[1] Moderate to worse asthma and asthma attacks are endpoints included in the definition of MRAD/Any of 19 respiratory effects. Although valuation estimates are presented for these categories, the values are not included in total benefits to avoid the potential for double-counting.
[2] Summing 5th and 95th percentile values would yield a misleading estimate of the 5th and 95th percentile estimate of total health benefits. For example, the likelihood that the 5th percentile estimates for each endpoint would simultaneously be drawn from a Monte Carlo procedure is much less than 5 percent. As a result, we present only the total mean.

**Table H-5**
**Primary Estimates of Health and Welfare Benefits Due to Criteria Pollutants in 2010**

| | Monetary Benefits (in millions 1990$) | | |
|---|---|---|---|
| **Benefits Category** | **5th %ile** | **Mean** | **95th %ile** |
| **Mortality** | | | |
| Ages 30+ | $ 14,000 | $ 100,000 | $ 250,000 |
| **Chronic Illness** | | | |
| Chronic Bronchitis | $ 360 | $ 5,600 | $ 18,000 |
| Chronic Asthma | 40 | 180 | 300 |
| **Hospitalization** | | | |
| All Respiratory | $ 76 | $ 130 | $ 200 |
| Total Cardiovascular | 93 | 390 | 960 |
| Asthma-Related ER Visits | 0.1 | 1 | 2.8 |
| **Minor Illness** | | | |
| Acute Bronchitis | $ 0 | $ 2.1 | $ 5.2 |
| URS | 4.2 | 19 | 39 |
| LRS | 2.2 | 6.2 | 12 |
| Respiratory Illness | 0.9 | 6.3 | 15 |
| Mod/Worse Asthma[1] | 1.9 | 13 | 29 |
| Asthma Attacks[1] | 20 | 55 | 100 |
| Chest tightness, Shortness of Breath, or Wheeze | 0 | 0.6 | 3.1 |
| Shortness of Breath | 0 | 0.5 | 1.2 |
| Work Loss Days | 300 | 340 | 380 |
| MRAD/Any-of-19 | 680 | 1,200 | 1,800 |
| **Welfare** | | | |
| Decreased Worker Productivity | $ 710 | $ 710 | $710 |
| Visibility Recreational | 2,500 | 2,900 | 3,300 |
| Agriculture | 7.1 | 550 | 1,100 |
| **Total Benefits[2]** | | **$ 110,000** | |

Note:
[1] Moderate to worse asthma, asthma attacks, and shortness of breath are endpoints included in the definition of MRAD/Any of 19 respiratory effects. Although valuation estimates are presented for these categories, the values are not included in total benefits to avoid the potential for double-counting.
[2] Summing 5th and 95th percentile values would yield a misleading estimate of the 5th and 95th percentile estimate of total health benefits. For example, the likelihood that the 5th percentile estimates for each endpoint would simultaneously be drawn from a Monte Carlo procedure is much less than 5 percent. As a result, we present only the total mean.

## Uncertainties in the Valuation Estimates

The uncertainty ranges for the results on the present value of the aggregate measured monetary benefits reported in Table H-4 and Table H-5 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;
- Uncertainty about the economic value of each quantified health and welfare effect.

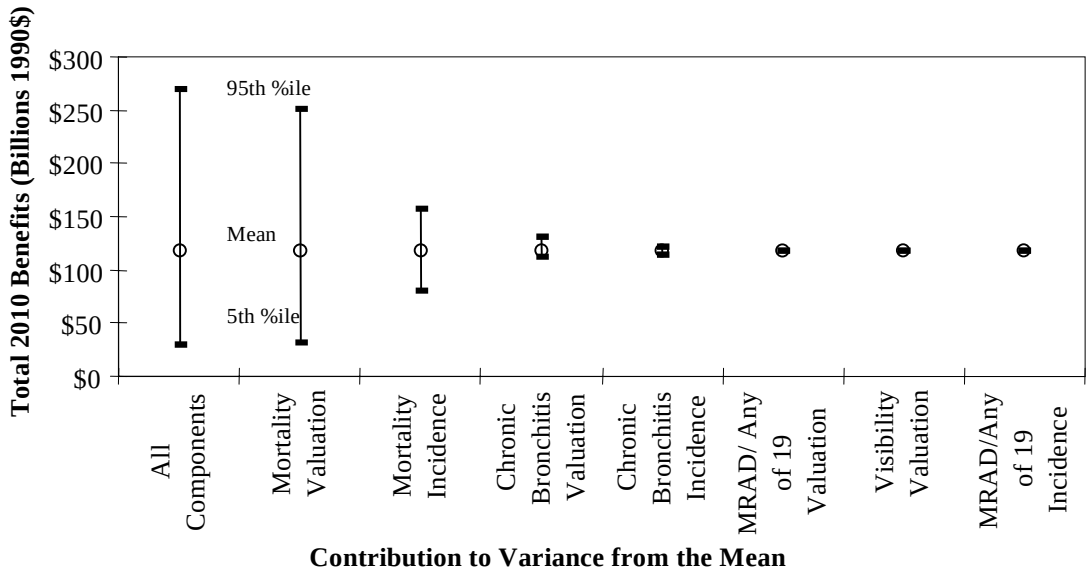
These aggregate uncertainty results incorporate many decisions about analytical procedures and specific assumptions discussed in the Appendices to this report.

In order to provide a more complete understanding of the economic benefit results, we conduct sensitivity analyses which examine several additional important aspects of the main analysis. We begin with an analysis of 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. We then follow with an examination of several issues involving the estimated economic benefits of mortality. In the third section, we provide some insight into the potential effects of income growth on the valuation of health effects.

### *Relative Importance of Different Components of Uncertainty*

The estimated uncertainty ranges in our primary results tables, Table H-4 and Table H-5, reflect the measured uncertainty associated with both avoided incidence and economic valuation. A better understanding of the relative influence of individual

**Figure H-4**
**Analysis of Contribution of Key Parameters to Quantified Uncertainty**



variables on the overall uncertainty in the analysis can be gained by isolating the individual effects of important variables on the range of estimated total 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 quantified uncertainty. The sensitivity analysis then isolates how this single source of variability contributes to the variation in the primary estimates of total benefits. The results are summarized in Figure H-4. The nine individual uncertainty factors that contribute the most to the overall uncertainty are shown in Figure H-4, 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.

## *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. We begin with a discussion of the uncertainties and possible biases related to the "benefits transfer" approach employed to develop our VSL estimate. We then discuss an alternative method for the valuation of reduced premature mortality, value of statistical life year (VSLY). We conclude this section with a sensitivity test that compare the benefit estimates using a VSL approach and a VSLY approach. Given the lag structure employed in estimating reduced premature mortality, we also provide alternative calculations for the valuation of this benefits category using two additional discount rates, three and seven percent.

## Benefits Tranfer and VSL

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 risks being valued are similar, and (2) the extent to which the subjects in the studies are similar to the population affected by changes in pollution concentrations. 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.

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.

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. Several studies indicate that the value people place on mortality risk reduction may depend on the nature of the risk (e.g., Fisher et al. 1989; Beggs 1984). 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.

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.

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. 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.

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 be willing to pay more to reduce risk, all else equal, than individuals with lower incomes or wealth. This does not imply that individuals with higher incomes are willing to pay proportionally higher values. While many analyses assume income elasticity of willingness to pay is unit elastic (i.e., ten percent higher income level implies a ten percent higher willingness to pay to reduce risk changes), empirical evidence suggests that income elasticity is substantially less than one.

The effects of income changes on WTP estimates can influence benefit estimates in two different ways: (i) as longitudinal changes that reflect estimates of income change in the affected population over time, and (ii) as cross-sectional changes based on differences in income between study populations and the attracted populations. Empirical evidence of the effect of income on WTP gathered to date is based on studies examining cross-sectional data. Income elasticity adjustments to better account for changes over time, therefore, will necessarily be based on potentially inappropriate data.[23]

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.

## VSLY

An alternative valuation of avoided premature mortality is to use the VSLY. This approach uses life-years lost as the unit of measure, rather than estimating a single value of a statistical life lost (applicable to all ages). With statistical life-years lost as the unit of measure, the valuation 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).

We estimate the value of a statistical life-year assuming 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. This values assumes a five percent discount rate and that the undiscounted value of a life-year is the same no matter when it occurs in an individual's life.

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

[23]For more information on the potential impact of income elasticity on the valuation of health benefits, see the following section, "Sensitivity Test for Impact of Income Changes Over Time."

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-10 coefficients (and, from these, age-specific relative risks) were derived from the few age-specific PM-10 or TSP 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 approach developed for this analysis assumes that exposure to elevated levels of PM increases the probability of dying at a specific age. Increasing the probability of dying at each age lowers the life expectancy for each age cohort. The average number of life-years lost will depend on the distribution of ages in the population in a location. In addition, this analysis incorporates the five-year PM mortality lag structure described in Chapter 5 and Appendix D. It distributes the mortality for each cohort across a five-year period (25 percent in each of the first two years, 16.7 percent in each of the remaining years) and adjusts the loss of life expectancy accordingly. That is, when applying the lag assumption within a given cohort, individuals who die later are expected to lose fewer life years than those who die earlier. Further, this analysis applies a five percent discount rate when calculating the present discounted value of the avoided losses of life expectancy in each cohort over the five-year lag period.

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. Rowlatt, et al. (1998) indicate that at risk individuals include those who have suffered strokes or are suffering from cardiovascular disease and angina. The Criteria Document for Particulate Matter (U.S. EPA, 1996), however, identifies only two studies which attempt to evaluate the disproportionality in premature mortality among people who are elderly and/or sickly. 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.

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 was 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.

## Sensitivity Test of Benefits Due to Reduced Premature Mortality Valuation

Examining the sensitivity of the total benefits of reduced premature mortality to alternative valuation techniques does provide some illumination to the potential impacts of alternative approaches. This section presents alternative results to our primary estimate of mortality valuation using the life-years lost approach, and also examine the effects of alternative discount rates.

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 approach developed for this analysis assumes that exposure to

elevated levels of PM increases the probability of dying at a specific age. Increasing the probability of dying at each age lowers the life expectancy for each age cohort. The average number of life-years lost will depend on the distribution of ages in the population in a location. In addition, this analysis incorporates the five-year PM mortality lag structure described in Chapter 5 and Appendix D. It distributes the mortality for each cohort across a five-year period (25 percent in each of the first two years, 16.7 percent in each of the remaining years) and adjusts the loss of life expectancy accordingly. That is, when applying the lag assumption within a given cohort, individuals who die later are expected to lose less live expectancy than those who die earlier. Further, this analysis applies a five percent discount rate when summing the value of the avoided losses of life expectancy in each cohort over the five-year lag period.

The alternative central estimates for avoided PM-related premature mortality using a five percent discount rate are $33 billion in 2000 and $53 billion in 2010. The VSLY approach results in estimates that are almost 50 percent lower than our primary estimates of benefits due to avoided pre-mature mortality. The sensitivity analysis, however, indicates that the pattern of monetized mortality benefits with each valuation procedure is essentially invariant to the discount rate. We summarize these results in Table H-6.

We emphasize that the results of the VSLY approach to valuing avoided mortality benefits represent a crude estimate of the value of changes in age-specific life expectancy. These results should be interpreted cautiously, due to the several significant assumptions required to generate a monetized estimate of life years lost from the relative risks reported in the Pope et al., 1995 study and the available economic literature. These assumptions include, but are not limited to: extrapolation of the age distribution of the U.S. population in future years; assumptions about the age-specificity of the relative risk reported by Pope et al., 1995; assumptions about the life expectancy of different age groups; assumption of a particular lag structure; assumptions about the age-specificity of the lag period (if any); derivation of VSLY estimates from VSL estimates; assumptions about the variation in VSLY with age; and selection of an appropriate rate at which to discount the lagged estimates of life years lost. Changes in any of these assumptions could significantly affect the VSLY benefit estimate. For example, if we were to assume no lag period for PM-related mortality effects instead of the five-year lag structure, VSLY benefit estimates would increase from $53 billion to $61 billion.

**Table H-6**
**Sensitivity Analysis of Alternative Discount Rates on the Valuation of Reduced Premature Mortality**

| Benefit Category & Discount Rate | 2000 (in millions, 1990$) | | | 2010 (in millions, 1990$) | | |
|---|---|---|---|---|---|---|
| | 5th %ile | Central | 95th %ile | 5th %ile | Central | 95th %ile |
| **VSL Approach** | | | | | | |
| 3% Discount Rate | $ 8,900 | $ 65,000 | $ 150,000 | $ 14,000 | $ 100,000 | $ 250,000 |
| 5% Discount Rate | 8,600 | 63,000 | 150,000 | 14,000 | 100,000 | 250,000 |
| 7% Discount Rate | 8,300 | 61,000 | 150,000 | 14,000 | 97,000 | 240,000 |
| **VSLY Approach** | | | | | | |
| 3% Discount Rate | $ 4,600 | $ 30,000 | $ 68,000 | $ 7,400 | $ 48,000 | $ 110,000 |
| 5% Discount Rate | 5,000 | 33,000 | 74,000 | 8,100 | 53,000 | 120,000 |
| 7% Discount Rate | 5,400 | 35,000 | 80,000 | 8,800 | 57,000 | 130,000 |

Note: The discount rate affects the benefits estimates of VSL and VSLY approach differently. With the VSL approach, higher discount rates lead to lower estimates because of the lag structure. With the VSLY approach, the higher discount rates lead to higher estimates because of its affect on the annualized values.

### *Sensitivity Test for Impact of Income Changes Over Time*

As an illustrative calculation, we adjust willingness-to-pay (WTP) measures to reflect the expected increase in real income over the full period of the analysis, 1990 to 2010. Our procedure results in an upward adjustment to more accurately reflect the valuation of improved health as income increases over time. In this section, we describe the procedure we use and the results of our illustrative calculation.

## Background and Methodology

Economists use income elasticity to evaluate how private and public goods are valued based on the interaction between income changes and demand. A negative relationship between income and demand for a good implies that the good is an inferior good. An individual demands less of a good as income rises. A positive relationship between income and the demand for a good implies that the good is normal (i.e., income elasticity is greater than zero). As income rises an individual demands more of a good. Depending on the relative responsiveness of demand to income changes, normal goods are characterized as a necessity or a luxury. When income elasticity is between 0 and +1, the good is considered a necessity (i.e., demand is not significantly responsive to income). In contrast, when income elasticity exceeds +1, the good is considered a luxury (i.e., the relative increase in the good's demand exceeds the increase in income).

The determination of a public good as inferior or normal based on income elasticity is complicated by its nonrival nature. In the case of a private good, varying the level of consumption is measured as a marginal change and implies that an individual will adjust his or her consumption level of other good(s). Consequently, income elasticity of demand estimates a change in quantity consumed, and not necessarily a change in utility (or the individual's well-being). With public goods, the conceptual logic is different. Income elasticity of WTP for public goods measures changes in consumer surplus. For example, one person enjoying the benefits of cleaner air does not reduce the probability of another person enjoying the same benefits. There are no apparent mechanisms for regulating who specifically will enjoy the benefits. In other words, there is no direct relationship between an individual's WTP and level of consumption.[24] The consumption level of public goods is exogenous to the individual's budget constraint. At the same time, WTP for a public good is not exogenous. An individual, therefore, must consider how his or her WTP affects the allocation of income among private and public goods.[25]

Flores and Carson (1997) provide examples of how income elasticity can change depending on how the good is defined (i.e., private or public). Given the divergence between private and public goods, they conclude that income elasticity of WTP and income elasticity of demand are related. The relationship does not imply that knowledge of income elasticity of demand is sufficient to estimate income elasticity of WTP given that the income elasticity of WTP depends on factors that cannot be observed.

In addition to the theoretical issues associated with WTP for public goods, there are important empirical issues. We are interested in how WTP changes with respect to increases in U.S. median income. Measuring changes due to growth in median income reflect shifts in overall preferences and utility (or in the case of public goods, social welfare). This type of analysis requires time series data. Unfortunately, there are very few relevant studies that use this approach to estimate income elasticity.[26] Consequently, we must rely on income elasticities estimated from cross-sectional data. The estimates

[24]The nonrival nature of public goods implies that the marginal social cost of consuming an additional unit of benefit is zero.

[25]CV studies solicit WTP estimates that are subject to the respondent's current budget constraint. The budget share factor requires that the income elasticities (for all consumed goods) sum to one. This generally implies that income elasticity of any single good is substantially less than one.

[26]Available studies using time series data estimate income elasticity of public health care expenditures by analyzing changes in government spending relative to gross domestic product (GDP). These studies are not particularly applicable to the valuation methodology used in the present study.

reflect differences in willingness to pay for improved health among various income levels. They are measures of an individual's preferences and expected utility given the person's current state (i.e., in the present).

There are several issues associated with the application of cross-sectional results to estimate longitudinal changes (i.e., changes over time). Most important is the potential for misinterpretation of our recommended application of income elasticity adjustment. Although we outline an approach that uses income elasticities derived from cross-sectional data, the adjustment is solely a proxy for how preferences and utility may change as projected overall average income (i.e., real GDP per capita) increases from 1990 to 2010. Application of these income elasticity estimates does not imply a strategy for adjusting benefits valuation by level of household income in any given year.

## Derivation of Elasticity Estimates

Based on our review of the available income elasticity literature, we conducted sensitivity analyses that characterize how the valuation of human health benefits may increase with a rise in real U.S. income. Given the range of different methodological approaches and limited available research, we calculate a range of illustrative values. Table H-7 summarizes the income elasticities we used to conduct the sensitivity analysis.

**Table H-7**
**Elasticity Values for Conducting Sensitivity Analysis**

| Health Endpoint | Lower Estimate | Central Estimate | Upper Estimate |
|---|---|---|---|
| Minor Health Effect | 0.04 | 0.14 | 0.30 |
| Severe and Chronic Health Effects | 0.25 | 0.45 | 0.60 |
| Premature Mortality | 0.08 | 0.40 | 1.00 |

Note: Sources for the derivation of these values can be found in Industrial Economics 1999.

Reported income elasticities suggest that the severity of the morbidity endpoint is a primary determinant of the strength of the relationship between changes in income and the willingness to pay. Without accounting for severity, there is a fairly wide range of values for income elasticity, 0.04 to 0.60. Estimates are more closely clustered if we account for the seriousness of the health effect. For the purposes of a sensitivity analysis, we use two different ranges based on whether morbidity endpoints are minor or severe. With respect to minor health effects, we use lower and upper values of 04 and 0.30, respectively. The central estimate is 0.14. For conducting a sensitivity test of the income elasticity effect on WTP to avoid severe health effects, we use a lower and upper estiamtes of 0.25 and 0.60, with 0.45 as the central estimate. The lower and upper estimates reflect the lowest and highest estimates derived from our literature review. The central estimate is the midpoint of the averages from each study.

With respect to VSL, estimates of income elasticity range from 0.08 to 1.10. We use lower and upper estimates that reflect the full range of values. The central estimate, 0.40, represents the midpoint between the average low value and the average high value of the studies we reviewed.

## *Illustrative Calculations — Morbidity Benefits Estimates*

Table H-8 provides a simplified example of how application of the elasticity ranges we derive could affect benefits estimates. For illustrative purposes, we use the WTP to avoid an asthma attack to represent a minor health effect and WTP to avoid a case of chronic bronchitis to represent a severe health effect. By the year 2010, the effect of income growth on WTP for a minor health effect can increase between one and eight percent, with the central estimate indicating three percent growth. The WTP to avoid a severe health effect grows faster with 2010 estimates, ranging between seven and sixteen percent and with the central estimate increasing by thirteen percent.

**Table H-8**
**Illustrative Adjustment to Estimates of WTP to Avoid Morbidity**

| | | | | WTP Estimate (1990 Dollars)[1] | | |
|---|---|---|---|---|---|---|
| **Year** | **US Population (in millions)** | **Real GDP (in millions)** | **Income** | **Lower Estimate** | **Central Estimate** | **Upper Estimate** |
| **Minor Health Effect- Asthma** | | | | **$E_y$=0.04** | **$E_y$=0.14** | **$E_y$=0.30** |
| 1990 | 249,440 | 5,744 | 23,026 | $32 | $32 | $32 |
| 2000 | 274,634 | 7,123 | 25,936 | $32.20 | $32.50 | $33.20 |
| 2010 | 297,716 | 8,959 | 30,092 | $32.30 | $33.20 | $34.70 |
| **Severe Health Effect- Chronic Bronchitis** | | | | **$E_y$=0.25** | **$E_y$=0.45** | **$E_y$=0.60** |
| 1990 | 249,440 | 5,744 | 23,026 | $260,000 | $260,000 | $260,000 |
| 2000 | 274,634 | 7,123 | 25,936 | $267,850 | $274,300 | $279,240 |
| 2010 | 297,716 | 8,959 | 30,092 | $277,990 | $293,280 | $305,290 |

Note:
[1] WTP estimates are reported in undiscounted 1990 dollars and represent value per case.

## *Illustrative Calculations — VSL Estimate*

We characterize the potential effect of income elasticity on the VSL estimate in Table H-9. An income elasticity of 0.08 demonstrates the effect of a slight adjustment to the VSL estimates as median income gradually rises. As shown in the figure, between 1990 and 2010, the VSL estimates increase by approximately two percent. The central estimate, 0.40, demonstrates that by 2010, a thirty percent increase in median income would result in VSL increasing by approximately eleven percent. The upper bound value demonstrates the effect of assuming one as the value of income elasticity. In this twenty year period of the prospective analysis, the VSL estimate would increase from $4.8 to $6.3 million if income elasticity equals one.

**Table H-9**
**Illustrative Adjustment to Estimates of The Value of Statistical Life**

| | | | | Value of Life Estimate (in thousands)[1] | | |
|---|---|---|---|---|---|---|
| Year | US Population (in millions) | Real GDP (in millions) | Income | Lower Estimate $E_y$=0.08 | Central Estimate $E_y$=0.40 | Upper Estimate $E_y$=1.0 |
| 1990 | 249,440 | 5,744 | 23,026 | $4,800 | $4,800 | $4,800 |
| 2000 | 274,634 | 7,123 | 25,936 | $4,848 | $5,036 | $5,410 |
| 2010 | 297,716 | 8,959 | 30,092 | $4,905 | $5,345 | $6,271 |

Note:
[1] Value of life estimates reported in undiscounted 1990 dollars.

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# Implications for Future Research

Appendix I

Throughout this report we have attempted to accurately characterize and, where possible, quantify the major sources of uncertainties that affect our primary estimate of the costs and benefits of the CAAA. In many cases, these uncertainties are the result of gaps in data or methods that might be addressed through additional research. In this Appendix, we provide a summary of important areas for new research which, if carried out, have the potential to increase accuracy and reduce uncertainty in future assessments.

## Overview

The uncertainties in the primary estimates and the controversies which persist regarding model choices and valuation paradigms highlight the need for a variety of new and continued research efforts. Based on the findings of this study, the highest priority research needs are:

- Improved emissions inventories and inventory management systems
- Improved tools for assessing the full range of social costs associated with regulation, including the tax-interaction effect
- A more geographically comprehensive air quality monitoring network, particularly for fine particles and hazardous air pollutants
- Development of integrated air quality modeling tools based on an open, consistent model architecture
- Increased basic and targeted research on the health effects of air pollution, especially particulate matter
- Continued efforts to assess the cancer and noncancer health effects of air toxics exposure
- Development of tools and data to assess the significance of wetland, aquatic, and terrestrial ecosystem changes associated with air pollution
- Continued development of economic valuation methods and data, particularly valuation of changes in risks of premature mortality associated with air pollution

We discuss each of these research needs in greater detail in the sections that follow.

## Emissions Modeling

Our analysis of emissions suggests several areas of research that could improve emissions data and modeling tools. The overall importance of ambient particulate matter estimates to the results of this analysis makes improved modeling of particulate matter and precursor emissions a high priority. Ambient monitoring of particulate composition, for example, indicates that particulate matter of crustal origin (e.g., from agricultural tilling, construction, and wind erosion) may be over-represented in our emissions inventories. As we discuss in the report, one possible explanation for this apparent inconsistency may be the extent to which these emissions are transported beyond their point of emission. Some preliminary evidence suggests that the mobility of a large fraction of these particles may be relatively limited, but further research is needed to confirm this hypothesis.

Comparison of emissions inventories with monitoring data also suggests that our inventory may

underestimate the organic and elemental carbon fraction of directly emitted particulates, particularly in urban areas. In this case, it is more difficult to assess the potential sources of underestimation. One hypothesis is that current emissions estimation tools may underestimate organic particulate emissions from mobile sources. Continued research into emissions rates for mobile sources could yield increased accuracy for particulate emissions. Additional tailpipe emissions studies may be needed, and emissions estimation techniques need to be developed to better reflect the results of those studies. In general, continued research to better reconcile monitoring data on the composition of ambient particulate matter on the one hand, with emissions estimates for primary and secondary sources of particulate matter on the other hand, should help in improving our ability to predict changes in fine PM concentrations.

One other emissions uncertainty that could be reduced by additional research involves volatile organic compound (VOC) emissions. Estimates of VOC emissions tend to be highly variable -- in the summer months especially, they can be closely linked to variations in temperature. As ambient ozone modeling becomes more sophisticated, however, better temporal and spatial resolution of VOC emissions inventories may be needed to take advantage of the increased capabilities of air quality models to process more highly resolved data.

In a broader sense, our current inability to quantitatively characterize and carry through the analysis the impact of key uncertainties in emissions estimation may give the misleading impression that these uncertainties are less important than other quantifiable sources of uncertainty. For example, the statistical simulation modeling analysis we present here reflects only quantifiable sources of uncertainty in the concentration-response and economic valuation steps of the analysis. Uncertainties in emissions estimates, however, may be among the most important in the entire analysis. Emissions estimates are a critical first step in our approach, so errors in this step can magnify as we work through the subsequent steps of the analysis. One way to enhance the quantification of emissions estimation uncertainties in future assessments, and to reduce any potential errors of inconsistency with the subsequent air quality modeling steps, is to develop a tool that both integrates emissions and air quality analyses and provides a means to more cost-effectively perform multiple scenario analyses. The Models-3 development effort, described below, may provide a modeling platform that is more amenable to sensitivity testing of alternative emissions results.

## Cost Estimation

The first prospective analysis relies on direct expenditure estimates to characterize the costs of the CAAA. As we state in the report, this approach probably does not represent a large source of error in our estimate of social costs, though there is some evidence it may provide conservative estimates. The direct cost approach does not provide information on other potential categories of impact that may be of interest, however, including total employment, employment by sector, capital accumulation patterns, and the pace of technological change. Additional cost-effective tools are needed to better estimate the secondary impacts of direct cost estimates for broad, programmatic assessments such as the section 812 series.

One potentially important area where research may enhance our ability to conduct broader assessments is development of computable general equilibrium (CGE) models that can be implemented in a resource-effective manner. The potential for introducing additional error when using such a forecasting tool, however, demands the model be capable of processing many scenarios of important economic inputs (e.g., alternative interest rate scenarios) to better bracket the range of future outcomes relevant to CAAA implementation.

A well-designed CGE model may also enhance our capability to estimate the effects of the tax-interaction effect, both on the cost and the benefit side. Additional empirical work will also be needed to confirm that the magnitude of the effect estimated in

the current literature, which is largely confined to the electric utility sector, is applicable for other economic sectors where the competitive dynamics and capital-intensity of production may differ from those in the electric utility industry.

## Air Quality Modeling

Our current limited ability to disaggregate the overall benefits of the CAAA is largely attributable to the complexity of the relationships between changes in precursor emissions and the ambient concentration outcomes. For example, nitrogen oxides are precursors of both fine particulate matter and ozone, and their presence in the atmosphere also affects the conversion of sulfur dioxide to fine particles. In addition, while low levels of nitrogen oxides can contribute to elevated ozone concentrations, very high levels of nitrogen oxides, in the right combination with VOCs and certain meteorological conditions, can suppress ozone concentrations. These complex inter-relationships among pollutants affected by the CAAA, coupled with the national scope of the analysis conducted here, demand the use of an air quality modeling technique that accurately reflects the complexities of atmospheric chemistry. Estimating the incremental impact of various combinations of emissions changes would require the repeated exercise of the model for each alternative set of emissions scenarios of interest.

The models we chose for this analysis, while they represent the current state of the art in modeling atmospheric chemistry, are difficult and expensive to run for a wide range of scenarios. To improve our ability to disaggregate the benefits of the CAAA, we need a fully integrated air quality modeling and emissions input system that accounts for the full range of pollutant interactions and relevant atmospheric chemistry. The current Models-3 effort holds promise in this area, but must be adequately funded to achieve these goals. Pursuit of a fully integrated modeling system also holds promise for generating more accurate ambient particulate matter estimates. The current best modeling systems for this purpose provide estimates based solely on changes in the concentrations of sulfate- and nitrate-derived particles, with limited abilities to assess changes associated with organic precursors of fine particles. Gaining a good understanding of organic particle formation may also be an important goal in better characterizing the full range of impacts of efforts to control air toxics under Title III. In addition, a more cost-effective air quality modeling tool may also enhance our ability to conduct comparative analyses and explore the sensitivity of air quality modeling and emissions estimation outcomes to alternative assumptions and modeling paradigms.

Improvements in exposure analysis might also be made with additional research into techniques for extrapolating the results of monitor-based analyses to unmonitored areas. In particular, we suggest further exploration and development of methods that base extrapolation on the causes of ambient air quality (e.g., local land use, emissions characterizations, meteorology, and terrain), rather than the outcomes of air quality modeling (e.g., simple extrapolation of air quality concentrations). In the course of developing this analysis, we began development of such an approach, termed the "homology mapping" technique. Continued development of this tool could improve the accuracy of our estimates in future analyses for those areas that are distant from monitors.

## Human Health Effects Estimation

The results of our analysis clearly highlight the importance of the link between premature mortality and air pollution. The wide range of current research on this link, including the several short-term and long-term cohort studies, provides a strong basis for establishing that particulate matter contributes to premature mortality among the exposed population. The existing studies, however, are limited by the availability and resolution of air quality monitoring data, data on the characteristics of exposed populations, and, in the case of the long-term studies, extensive time-series of these data. The continued enhancement of our air quality monitoring network,

particularly for fine particles, is critical to the better understanding of the relationships between fine particles and human health effects. Developing long-time series of fine particle data will take time, however. In the meantime, it is important to continue efforts to better isolate the separate and joint impacts of ambient pollutants on premature mortality, including better resolution of the incremental impacts of ozone, carbon monoxide, nitrogen oxides, sulfates, and particles in the ultrafine and fine fraction, as well as the coarse particles within $PM_{10}$.

In addition, the sensitivity analyses presented in Appendix D show that a resolution of competing alternative hypotheses about the presence and potential time period of a lag in the incidence of premature mortality following exposure may be important. Although in our judgment assuming a distributed five-year lag period may be warranted, there is no scientific basis for either the assumption of a lag or for determining the appropriate time period. We believe it will continue to be important to evaluate the existing evidence and develop new studies to clarify the extent to which the premature mortality outcomes reflected in the existing epidemiological literature ought to reflect a lag period between exposure and the mortality effect.

For premature mortality and for other health effects, our analysis is based on the premise that the available literature provides broadly applicable characterizations of the relationships between exposure to air pollutants and the incidence of health effects. We use the results of available studies on a national basis, although in many cases the underlying literature may be based on analysis of the concentration-response relationship in a particular region. It is possible, however, that region-specific factors may play a role in the results of these studies. For example, the composition of air pollutants such as particulate matter varies by region, and it is possible that other, perhaps unobservable factors may have a synergistic or mitigating effect on the incremental incidence of health effects. The literature on air pollution's influence on health is not yet broad enough for us to implement a regional approach to health effects estimation. As the literature base develops, however, a regional approach may be an option for future assessments. In the meantime, it is important to continue to develop a broader base of regional estimates of the effects of multiple pollutants on key health outcomes, including mortality, chronic bronchitis, and hospital admissions, to better reflect the impact of potentially important regional differences in the composition of particulate matter and other human health stressors. Expanding the current literature base may also provide a better means for evaluating the effects of air pollution on sub-populations of individuals, such as children and the infirm, that may be of increasing importance in the Federal government regulatory effort.

## Evaluation of the Effects of Air Toxics

In order to develop a meaningful estimate of the benefits of air toxics controls for future 812 Prospective analyses, we must address existing knowledge gaps and other methodological barriers that prevent more realistic analyses of the benefits of air toxics control. We have already begun developing a detailed research plan for improving the assessment of air toxics in future prospective analyses. For example, EPA has agreed to sponsor workshops that bring together experts in toxicology, exposure and risk assessment, and economics with the goal of establishing a framework for air toxics benefit estimation. In establishing such a framework, we will need to address issues and research needs related to estimating both air toxics exposure and the hazard posed by individual air toxics.

Exposure-related research needs include both the development of a database of air toxics measurements and the extent to which individuals, on average, would be exposed to the measured concentrations. To address the first issue, we plan to explore the potential for compiling a database of air toxics data from established state air toxics monitoring networks. We also plan to explore design options for the "super-site" monitoring programs that will permit them to be exploited to better understand exposure to air toxics

linked to key health effects categories, and to improve the performance of ambient concentration modeling efforts.

More generally, there is a need to continue to pursue research aimed at the following goals: (a) improving methods to estimate current levels and future changes in acute and chronic ambient exposure conditions nationwide; (b) evaluating the full distribution of concentration-response relationships linking exposure and health outcomes, with the goal of providing a better estimate of the central tendency of the relationships to support primary benefits estimation; and (c) tailoring economic valuation methods for the broad array of potential serious health effects such as renal damage, reproductive effects and fatal and non-fatal cancers, including accurate characterization of the impact on valuation of latency periods for these effects, where applicable.

## Ecological Effects Estimation

The research needs for future analysis of the CAAA's ecological benefits can be viewed from two perspectives. The first is the valuation of additional first order, acute ecological effects that change the level of service flows society receives from ecosystems, and the second is assessing and valuing the broader changes to the structure and function of ecosystems. Our analysis reflects the state of the current data and methods in this area by characterizing and quantifying ecosystem service flows affected by air pollutants, but many gaps remain. Pursuing a strategy of enlarging the array of quantified service flows would entail further development of economic models and collection of data. However, notably absent in this report is quantitative treatment of the changes of ecosystem structure and function that do not measurably affect the provision of service flows to society, such as nutrient cycling, species composition, and the resistance and resilience of ecosystems to disturbance. Because many ecological benefits of the CAAA fall into this category, future research could adopt a strategy of developing analytic tools to assist in the valuation of these impacts.

Our current analysis suggests several ways we can enhance the comprehensiveness of coverage for potentially important service flows. For example, while we can develop estimates of the changes in mercury emissions attributable to CAAA provisions, and there is an extensive literature on the effects of mercury in ecological systems, there are great uncertainties in estimating the fate and transport of incremental increases in airborne mercury emissions. The persistence of this element, the potentially long recovery times for ecological systems contaminated with mercury, and the potentially global scale of mercury transport suggest that overcoming this barrier will be challenging; however, existing tools may provide a good starting point for bounding analyses. Similar issues are present in assessing the ecological effects of air toxics. Some toxics are persistent in natural systems, can be attributed to multiple airborne and other sources, and have been accumulating in the environment for many years. Analyses aimed at characterizing effects at the watershed level, however, may be more successful in capturing many of the complexities of source-receptor relationships and receptor sensitivities than the national analyses we have traditionally pursued.

Analyses of nitrogen deposition in the current report are based on a displaced cost approach. The uncertainties and potential circularity of this approach limit its applicability to a subset of the aquatic systems susceptible to eutrophication. A more widely applicable and therefore more promising approach for future analyses will be an avoided damages analysis. To complete such an approach, however, further research is needed to better explain the dose-response relationships between increased nitrogen loads, thresholds of nitrogen loading that lead to eutrophication, and the ecological mechanisms that lead to the loss of service flows such as recreational and commercial fishing. Some recent analyses attempt to bridge this gap through the development of reduced form relationships between nitrogen loads and service flow disruption, but these types of approaches also have only limited applicability unless they can be shown to hold for long periods of time and across a wider range of marine environments, climate conditions, and species types.

In some cases acute impacts to ecosystems, such as the disappearance of game species from particular ecosystems or foliar damage to trees, attract the attention of the public and policy makers. Such acute impacts generally have effects that are observable and alter the provision of ecosystem services in a measurable way. Less often noticed are the ecosystem-level ecological impacts that change ecosystem structure and function but do not immediately affect service flows received from that ecosystem. By focusing on acute impacts it is possible to lose sight of ecosystem-level changes to structure and function that could eventually lead to large-scale impacts far greater in degree and geographic extent than the contemporaneous, acute effects.

Ecosystems generally maintain multiple interchangeable elements that may drive a particular process, as hypothesized by Odum (1985) for forest ecosystems and Howarth (1991) for aquatic systems. This allows for natural variation in these elements as well as long-term cycles in which some elements dominate over others. Explicit in the definition of ecological structure and function is the ability of an ecosystem to adapt to natural changes in its environment. When pollution affects ecosystem functions such as nutrient cycling, water filtration, biological diversity, and provision of habitat, it may also be precluding the system's ability to adapt and respond to change and perform these functions in the future. The ultimate effects of such changes in ecosystems are sometimes unpredictable in scale and nature. Ecosystems impacted by mankind may respond in a discontinuous manner around critical thresholds that are boundaries between locally stable equilibria (Common and Perrings 1992; Constanza et al 1993). Complexity in ecosystems prevents analysts from using linear methods to "add up" the discrete ecological effects of pollution.

Additional research is also needed to develop economic valuation methods that can adequately characterize the monetized benefits of maintaining ecosystem structure and function in their current states. Contingent valuation approaches may prove valuable, but the scientific basis for evaluating changes in ecosystems needs to be sufficiently advanced that analysts can construct plausible scenarios of alternative ecosystem outcomes for respondents to react to. To lay the groundwork for these efforts, there is an immediate need to identify the key attributes of ecosystems that are most valued by individuals. The results of those types of scoping analyses might be useful in targeting subsequent scientific and ecological research, with the goal of developing pilot analyses that integrate robust and realistic characterizations of the changes in ecological resources attributed to air pollution with careful economic valuation approaches to assess the value of avoiding those changes.

The isolation of service flows, while a useful interim tool for quantifying and monetizing the effects of air pollutants on ecosystems, may imply an oversimplified cause and effect relationship between pollution and the provision of the service flow. As our analysis suggests, often the service flow is affected by complex non-linear relationships that govern ecosystem structure and function. Pursuit of the short-term goal of enhancing our understanding of ecological effects on service flows may ultimately provide new insights into our understanding of these complex relationships. At the same time, we suggest that it will continue to be important to pursue methods to estimate the effects of air pollution on other ecological indicators of concern, including those that may not be directly linked to service flows, recognizing that accurate assessment of changes in nutrient cycling, water filtration, biological diversity, provision of habitat, and other valuable aspects of ecosystems may ultimately demand a broader view of these effects.

## Economic Valuation

The importance of the economic valuation step in our benefits assessment is highlighted by our analysis of the influence of key variables on the overall range of uncertainty from our statistical simluation modeling analysis. Our analysis clearly shows that uncertainty in the measurement of the value of statistical life dominates the quantifiable uncertainty in our overall

estimates. In addition, there remain several important non-quantified uncertainties in the use of labor market studies to value avoidance of environmental risks from air pollution. These uncertainties were also highlighted in the section 812 retrospective analysis report, and as a result many are currently being pursued as part of federally sponsored and independent research studies.

First, to the extent we must continue to rely on the value of statistical life (VSL) approach to value avoided mortality risks, we need more advanced methods for discerning an appropriate VSL from the extensive literature on this topic. The application of meta-analysis techniques may help us better understand the impact of important methodological and measurement choices that are made in conducting these studies, and provide a basis for narrowing the range of appropriate VSL estimates for environmental risk estimates. Second, we need to develop a better means for adjusting VSL estimates to address the key benefits transfer issues from the risk scenarios presented in existing studies to the specific mortality risk presented by particulate matter air pollution. Pursuit of this goal would include developing better adjustment techniques for differences in age, health status, and the risk context, including such attributes of risk perception as dread and the involuntary nature of environmental exposures.

Third, and perhaps most importantly, we need to develop a better literature basis for directly valuing the commodity provided by air pollution reductions, that is, reductions in the risk of dying prematurely or, put another way, changes in individual's survival probabilities. Several research efforts are currently underway that are attempting to directly value life extensions similar in magnitude to those provided by air quality improvements. These research efforts necessarily rely on stated preference methods, which in most cases are considered less reliable than the revealed preference estimates used as the basis for VSL approaches. Substantial analytic challenges remain in making the risk reduction scenarios presented to respondents clear and understandable to the lay person, but the results of this new work will need to be carefully considered for their implications for a new paradigm for valuation of mortality risk reduction.

Beyond the valuation of avoided premature mortality, there are several other areas of research that, if pursued, can enhance our ability to value the health outcomes of reductions in air pollutants. For example, we must develop a broader research base for valuation of avoided effects to children, including construction of an overall framework for considering the welfare and utility of children within the broader family context, to better characterize the effects of air pollutants to this important sub-population. It may also be fruitful to pursue the potential cost-effective advantages of developing a more flexible means of valuing health effects through health-state utility approaches. More research is needed to assess the trade-offs in accuracy and precision of these results with the advantages of a broader set of WTP estimates to apply to relevant endpoints (for example, to value the avoidance of hospital admissions).

Additional research is also needed to enhance our ability to value important welfare effects. Visibility continues to be one of the most important welfare endpoints for analyses of particulate air pollutants, but it would be useful if the literature base were periodically updated to better reflect the current state-of-the-art in stated preference technique. For example, an important research direction would be to pursue development of additional estimates for residential visibility valuation to corroborate those currently available, and develop insights into the potential for double-counting in application of the location-specific residential and recreational visibility valuation estimates. The literature on materials damage valuation is also in need of updating. We chose not to include an estimate for household soiling effects in our primary benefits estimates because of the age of the original research, its reliance on an older measure of particulate air pollution (total suspended particulates), and its reliance on outdated household expenditure data. Updating existing estimates of the effects of air pollutants on household soiling expenditures would be a relatively straightforward research project. Agricultural analyses could also benefit from a broader assessment of the crops

potentially affected by ozone and other pollutants, and by the joint analysis of not only the damaging effects of some pollutants but also the yield-enhancing effects of others (e.g., nitrogen deposition).

The results of this first prospective analysis continue to suggest that our nation's investment in clean air has been a wise one. At the same time, we recognize that we should continue to assess the progress of the clean air program, as implemented under the Clean Air Act, to ensure that benefits are achieved in the most cost-effective means possible. Pursuit of the research goals outlined above will continue to enhance our ability to provide accurate and timely assessments of the costs and benefits of all provisions of the Clean Air Act.

# Text of the November 19, 1999 Final Advisory by the Advisory Council on Clean Air Compliance Analysis on the 1999 Prospective Study of Costs and Benefits of Implementation of the Clean Air Act Amendments (CAAA)

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

November 19, 1999

EPA-SAB-COUNCIL-ADV-00-003

OFFICE OF THE ADMINISTRATOR
SCIENCE ADVISORY BOARD

Honorable Carol M. Browner
Administrator
U.S. Environmental Protection Agency
401 M Street, SW
Washington, DC 20460

RE: Final Advisory by the Advisory Council on Clean Air Compliance Analysis on the 1999 Prospective Study of Costs and Benefits (1999) of Implementation of the Clean Air Act Amendments (CAAA)

Dear Ms. Browner:

On October 1, 1999, the Advisory Council on Clean Air Compliance Analysis (Council) held a public teleconference to review a draft Agency document, The Benefits and Costs of the Clean Air Act, 1990 to 2010; EPA Report to Congress (U.S. EPA, Office of Air and Radiation and Office of Policy, September 1999) and held a follow-up teleconference on October 15, 1999 to review an October draft of that same document. These two closure meetings represented the culmination of a multi-year series of review meetings during which the Council provided advice to the Agency on the study design, methodologies, and intermediate results. The Council submits this Advisory to complete its review responsibilities as defined in Section 812 of the CAAA.[1]

The Council believes that The Benefits and Costs of the Clean Air Act, 1990 to 2010 is a serious, careful study that, in general, employs sound methods and data. While we do not endorse all details of the study, we believe that the study's conclusions are generally consistent with the weight of available evidence. The Council also appreciates the Agency's responsiveness over the many years of this study's development to advice conveyed by the Council and its technical subcommittees. While the Project Team has not followed our advice in every instance, we believe that they have done a remarkable job on an extremely difficult project.

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[1] Specifically, subsection (g) of CAA §312 (as amended by §812 of the amendments) states: "(g) The Council shall -- (1) review the data to be used for any analysis required under this section and make recommendations to the Administrator on the use of such data, (2) review the methodology used to analyze such data and make recommendations to the Administrator on the use of such methodology; and (3) prior to issuance of a report required under subsection (d) or (e), review the findings of such report, and make recommendations to the Administrator concerning the validity and utility of such findings."

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We would, however, like to bring to your attention two major issues that arose in our review of the study. These pertain to the study's measurement of costs and representation of uncertainty regarding costs. Following our discussion of these points, we present suggestions to improve future Prospective Studies. Because of their importance, we would like to highlight these suggestions here:

a) We believe that benefits and costs must be disaggregated by individual provision of the Clean Air Act if benefit-cost analysis is to be useful in informing regulation.

b) Future studies must attempt to quantify uncertainties about regulatory costs, as well as uncertainties about the benefits of regulations. Failure to quantify cost uncertainties may give the impression that costs cannot exceed point estimates.

c) Cost estimates should include tax-interaction effects; i.e., they should reflect the fact that environmental regulations may exacerbate the disincentive effects of the personal and corporate income taxes. This may raise cost estimates considerably.

d) The Agency should revise its estimates of the Value of a Statistical Life.

e) The impact of air quality regulations should be stated in terms of a Net Cost per Life Saved and a Net Cost per Life-year Saved to facilitate comparisons with other health and safety regulations.

f) Attempts should be made to increase the set of ecosystem benefits valued and to improve estimates of the exposure and effects of air toxics.

## 1. Comments on the Drafts Provided for Council Review

a) The Relationship between Direct and Social Costs of Compliance. Social cost is the type of cost that is most relevant to the evaluation of the 1990 Clean Air Act. However, the draft Prospective Study relies primarily on estimates of the "direct" compliance costs for affected industries or pollution sources. The reliance on direct costs is understandable, since it is more difficult to assess the social costs. At the same time, it is important to articulate clearly and without bias the relationship between direct and social costs.

The Council believes that the study's discussion of this issue lacks balance and is prone to misinterpretation. The study describes in detail two factors that might cause direct costs to overstate true social costs (absence of attention to producer and consumer responses, and the assumption of a static technology). The October review draft contained a discussion of the tax-interaction effect, which can cause direct costs to understate social costs, possibly by a very large amount. In the Council's view, this effect merited discussion in the text and should not be relegated to a footnote. There is now a substantial body of published theoretical

and empirical research that indicates that, under typical conditions, tax-interactions can cause social costs to exceed direct costs by at least 25 percent, and in some cases by 100 percent or more. Table 3-3 further contributes to potential misinterpretation. It explicitly mentions a factor that would cause direct cost to overstate social cost (lack of attention to producer and consumer responses) but fails to mention explicitly the factor (tax interactions) that works in the opposite direction. By minimizing attention to the tax-interaction effect, the study gives readers the erroneous impression that the EPA's use of direct costs is likely to overstate social costs.

Tax interactions occur when environmental regulations exacerbate the distortions in labor and capital markets caused by prior income, profit, or sales taxes. These interactions may result from any regulations that raise production costs and thereby lower the real purchasing power stemming from given real wages. Even "small" regulations can produce significant tax-interaction effects. The Prospective Study fails to indicate the general relevance of these effects. The study states that general equilibrium effects are important where the regulatory action is known to have an impact on many sectors of the US economy. Although this statement is technically correct, it allows for the impression that such general equilibrium effects are unusual. It fails to point out the key finding from the tax-interaction literature: namely, that all regulatory actions have impacts on other sectors (particularly labor and capital markets) and that these general equilibrium impacts, under typical conditions, raise social costs substantially relative to direct costs.

In sum, the Council would urge the EPA to give more attention to the tax-interaction effect in order to achieve a more balanced and straightforward presentation of the relationship between direct costs and overall social cost. This is necessary to avoid giving the false impression that direct costs are likely to overstate social cost.

b) Characterization of Uncertainty with Respect to Cost Estimates. The main results of the first Prospective Study are summarized in a table of costs and benefits that appears both in the Executive Summary and in Chapter 8. Uncertainties about the benefits of the CAAA are nicely illustrated by a lower bound and an upper bound (90% confidence interval). In contrast, the cost of this environmental protection is represented only by a central estimate. (Cost uncertainties are discussed via sensitivity analyses in other tables, but these uncertainties are not combined into an overall set of bounds on the central cost estimate.)

Thus the benefit-cost ratios in that main summary table vary only with uncertainties about benefits. These ratios would vary even more if they incorporated some uncertainty about costs. Since costs are indeed uncertain, the table implicitly understates the true degree of uncertainty about the net benefits of the CAAA.

Even rough representations of uncertainty about these costs would be better than the current implication that costs are certain. One possibility would be to assume a uniform distribution about each element of cost, ranging from 50% to 150% of the central estimate. A second possibility is to show an additional row of benefit-cost ratios where the costs have been multiplied by 1.3 to account for the tax-interaction effect. A third possibility is suggested by reference to the fact that the Retrospective Study produced a central estimate of direct cost equal to $523 billion, while the modeling approach provided welfare effects between $493 billion and $621 billion. Since these bounds are 6% below and 19% above the central estimate, the same percentage bounds could be applied to the central estimate of costs in the Prospective Study. True bounds on costs in the Prospective Study would be preferred, but one of these rough estimates of bounds is better than using no bounds at all.

## 2. Suggestions to Improve Future Prospective Studies

a) Disaggregate Benefits and Costs by Title or Provision. The Council reiterates its strong recommendation for presenting the benefits as well as the costs of the CAAA by title and, preferably, by provision, in future studies. Without this level of disaggregation, the study cannot be used directly to identify how the CAAA might be improved in the future. The Council recognizes that a thorough disaggregation analysis was not feasible for the current study since resources were not available for exercising several air quality models to create the needed data base for the analysis. Future studies should not be limited in this regard since more universal and versatile platforms for air quality modeling, such as Models-3, are expected to be available. With careful design, using such a system, a small number of additional comprehensive modeling simulations can provide the information needed for a thorough bottom-up assessment of the CAAA benefits by individual title and even by some provisions. If, in the design phase of the next prospective study, it becomes apparent that resources cannot be allocated for these analyses, then an alternative design strategy combining use of top-down or screening model approaches combined with carefully selected essential comprehensive model simulations should be pursued.

b) Characterize Uncertainty about Costs. The costs imposed by air pollution regulations are highly uncertain. For example, the costs of sulfur dioxide abatement under the 1990 Clean Air Act have turned out to be a fraction of what was estimated in 1990. Unfortunately, uncertainty can lead to higher as well as lower costs.

EPA has relied on engineering estimates of abatement costs. Even if these estimates were accurate estimates of the cost of equipment and operating costs, they would understate social costs because of tax-interaction and other effects. EPA needs to discuss and to quantify the following sources of uncertainty:

(1) Uncertainty in the engineering cost estimates.

(2) Costs in addition to the engineering estimates, such as tax-interactions.

(3) Technical change due to the technology forcing that lowers costs.

(4) Changes in the wage rate or prices of materials due to the changes in demand.

c) Include Tax-Interaction Effects in Future Cost Estimates. One of the most important insights to emerge in Environmental Economics in the past 25 years is that regulations, by exacerbating existing distortions in the economy, can have social costs considerably in excess of direct compliance costs. An environmental regulation that raises the price of purchased goods and lowers the real wage will tend to, other things equal, cause a substitution of leisure for labor. This compounds the deadweight loss of the tax system, which, by driving a wedge between the gross and net of tax wages, causes individuals to substitute leisure for labor. This tax-interaction effect can, in some cases, double the costs of a regulation (Goulder et al. 1999, Parry et al. 1999).[2]

It is important for tax-interaction effects to be included in future Prospective Studies for two reasons. First, these costs are real. They represent real losses in output, and they occur even for small regulations. Second, the tax-interaction effect can at least to some degree be offset if the environmental program raises revenues, which are used to reduce the rates of other, distorting taxes. This implies that the costs of a program will depend on how a standard is achieved, which has implications for the choice of regulatory approach. For example, a permit market will have lower social costs if permits are auctioned and revenues recycled than if permits are given away (Goulder et al. 1997; Parry 1997).[3]

d) Revise Mortality Risk Estimates. The Council is uncomfortable with the Agency's use of $4.8 million (1990 U.S. dollars) for the Value of a Statistical Life (VSL) and $293,000 for the Value of a Statistical Life-year (VSLY) to value mortality risk reductions from reduced air pollution. We question the

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[2] Goulder, Lawrence H., Ian W. H. Parry, Roberton C. Williams III, and Dallas Burtraw, 1999. "The Cost-Effectiveness of Alternative Instruments for Environmental Protection in a Second-Best Setting," Journal of Public Economics 72(3):329-360; and Parry, Ian, W. H., R. C. Williams III, and L. H. Goulder, 1999. "When Can Carbon Abatement Policies Increase Welfare? The Fundamental Role of Distorted Factor Markets," Journal of Environmental Economics and Management 37:52-84.

[3] Goulder, Lawrence H., Parry, Ian W. H., and Dallas Burtraw, "Revenue-Raising vs. Other Approaches to Environmental Protection: The Critical Significance of Pre-Existing Tax Distortions," RAND Journal of Economics, Winter 1997; and Parry, Ian W. H., "Environmental Taxes and Quotas in the Presence of Distorting Taxes in Factor Markets," Resource and Energy Economics, Winter 1997,19:203-20.

appropriateness of the $4.8 million VSL even as a measure of prime-aged individuals' willingness to pay (WTP) for risk reductions, and we question the application of a WTP estimate for prime-aged individuals to a population of older individuals and people who are in poor health. Time limitations did not permit a thorough treatment of this issue prior to completing the first Prospective Study; hence we recommended that the Project Team use the $4.8 million figure. For future studies, however, we recommend that the Agency revisit the literature on the value of mortality risk reductions. The following points should be kept in mind when examining this literature:

(1) It is WTP for risk reductions that is the appropriate concept when valuing the mortality benefits of environmental regulations. The costs of environmental regulations are spread broadly over many individuals who, indeed, are paying for the resulting risk reductions.

(2) Labor market studies measure willingness to accept (WTA) compensation for increased risk of death. This is likely to exceed what people will pay (WTP) for the same risk reductions.

(3) Averting behavior and consumer product safety studies, which are omitted from the current list of 26 studies, do measure WTP. These studies should be considered in the review.

(4) In reviewing studies the population whose preferences are measured should be noted, as should the magnitude of the risk reduction valued. Studies should be identified that measure WTP for risk reductions among the populations that benefit from air quality regulation, especially older people, and that value risk reductions of the same magnitude as those in future Prospective Studies.

(5) There should be well-defined criteria for selecting studies, which are clearly stated and consistently applied. For example, compensating wage studies should adequately control for inter-industry wage differentials; contingent valuation studies should test for sensitivity to scope.

e) Present Cost-Effectiveness Results. Improvements in human health remain a major motivation behind air quality regulation and account for over 90% of the quantified benefits from Titles I-IV of the 1990 CAAA. Reductions in premature mortality, in turn, account for over 90% of these health benefits. Because mortality risk reductions are such a large component of the benefits from air quality regulation, the Council urges the Agency to express the outcomes of the CAAA in terms of: (1) Net Cost per Life Saved, and (2) Net Cost per Life-Year Saved. These are calculated by subtracting the value of non-mortality benefits from costs and dividing the result by: (1) the number of statistical lives saved, and (2) the number of statistical life-years saved.

By taking this approach the Agency would: (1) provide a measure of program effectiveness that avoids the use of flawed measures of VSL and VSLY and, more generally, avoids the controversies surrounding the valuation of mortality risk change; (2) be in line with standard practice in the public health community, where different programs are routinely compared using cost-effectiveness analysis; and (3) facilitate comparisons of the cost effectiveness of various health and safety programs with health-based environmental regulations. The Council feels such comparisons are necessary for improving public decisions about the allocation of society's scarce resources among competing ends.

f) Increase Set of Ecosystem Benefits Valued. The current Prospective Study has made important advances in identifying ecosystem services that can be linked to air pollution, and in trying to value these endpoints. For the purposes of valuation, it is convenient to categorize the impact of pollution on ecosystems as follows: (1) impacts that occur through markets (e.g., impacts of pollution on commercial timber stands or fish populations); (2) impacts that affect recreation (e.g., damage to National Parks from air pollution or to recreational fishing from acid rain); (3) impacts on ecosystems for which people have well-defined non-use values (e.g., damage to forest canopy, the value of reduced fish populations to non-anglers); and (4) other impacts on ecosystem functions and services, not otherwise classified, for example water and nutrient recycling, maintenance of biodiversity, climate stabilization. These indirect and more subtle effects may not be well understood or even perceived by people; yet they may have important impacts on human well being.

Techniques for valuing the first 3 categories of benefits are well-established, but the set of applied studies is sparse. The Agency might consider funding new studies, after determining which categories of benefits are likely to have the largest impact on regulatory decisions. When commissioning studies to measure non-use values, care should be taken in defining: (1) the geographical scope of what is to be valued (for example, are people asked only for non-use values in their state?); (2) the nature of substitutes (i.e., conditions at other locations); and (3) how many endpoints to value at the same time. For example, in regard to this last point, if a regulation to reduce nitrogen oxides (NOx) affects forests through ozone and fish population through acid rain, people should be asked to value the entire package of ecosystem benefits brought about by NOx reduction. Adding up WTP values from individual studies might overstate the value of the NOx reduction program if there are important substitution effects across ecosystem services.

A problem for policy analysis is that the endpoints that affect markets or for which people have well-defined use (recreation) and non-use values (e.g., damages to forests, fish and wildlife populations) do not capture the totality of ecosystem damages associated with pollution control decisions. In particular, they do not capture ecosystem functions and services such as nutrient recycling

and habitat provision. Nor do they capture the more subtle changes in ecosystem functioning that may to lead to non-marginal changes in ecosystem performance. Before these changes can be valued, however, it is essential that ecologists characterize the ecosystem outcomes (or indicators) that are important to ecosystem functioning and then relate these outcomes (or indicators) to particular activities or pollutants. This information is an essential foundation for economic valuation.

g) Estimate Exposure and Effects of Air Toxics. The Retrospective Study and the first Prospective Study do not contain any quantitative benefit-cost analyses of Toxic Air Pollutants (TAP). As the Council's Health and Ecological Effects Subcommittee (HEES) has stated,[4] the Agency does not currently have analytical methodologies available to establish population exposure estimates, or to define realistic risk estimates for the general population. The HEES, with approval by the full Council, suggested an approach to identifying the research and methodological developments needed to overcome these deficiencies. The effort requires coordination with the SAB Executive Committee, various SAB Committees, the Office of Research and Development and Office of Air Quality Planning and Standards. Implementation of the plan of action outlined for the Agency will begin a process that can lead to quantitative estimates of health, and possibly ecological benefits for the next Prospective Study.

## 3. Conclusion

The purpose of conducting benefit-cost analyses is to improve the efficiency of regulation. The suggestions we have made in this Advisory are designed to help achieve this goal. Increasing the accuracy of benefit-cost analyses will entail measuring certain categories of benefits (e.g., certain ecological benefits, benefits of reduced exposure to hazardous air pollutants) and costs (tax-interaction effects) not included in the current Prospective Study. It will also entail refining estimates of the value of mortality benefits, which continue to dominate the monetized benefits of improved air quality. Of all the suggestions made above, however, we believe that disaggregating the benefits and costs of individual provisions of the CAA is, perhaps, the most important. If our recommendation to provide more disaggregated benefit-cost estimates can be implemented, the specific programs which have the highest potential payoff to society can be more readily identified. We strongly encourage the Agency to make the research investment and analytical commitments required to ensure this objective is met in the next prospective study.

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[4] See HEES Letter Advisories, "The Clean Air Act Amendments (CAAA) Section 812 Prospective Study of Costs and Benefits (1999): Advisory by the Health and Ecological Effects Subcommittee on Initial Assessments of Health and Ecological Effects, Part 1", EPA-SAB-COUNCIL-ADV-99-012 and "Part 2", EPA-SAB-COUNCIL-ADV-00-001.

We thank the Agency for the opportunity to review the first Prospective Study and to make recommendations to improve the methods and data to be used in future prospective studies. We look forward to your response to this Advisory.

Sincerely,

Maureen L. Cropper

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