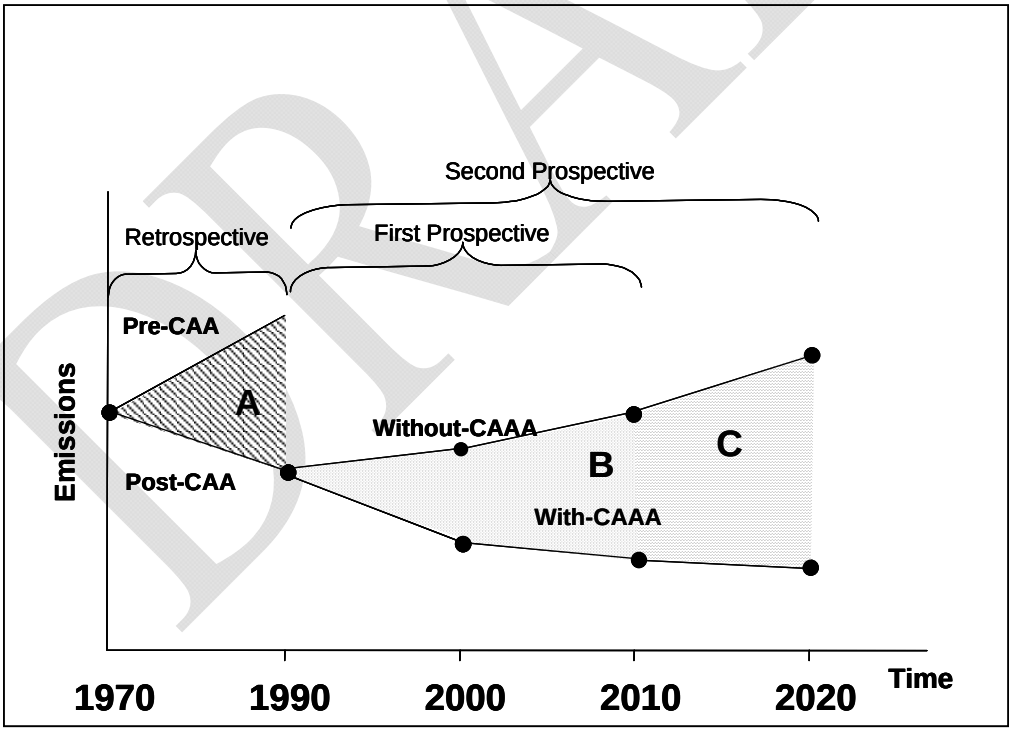
CHAPTER 1 - INTRODUCTION

Section 812 of the Clean Air Act Amendments of 1990 (CAAA) required the U.S. Environmental Protection Agency (EPA) to perform periodic, comprehensive analyses of the total costs and total benefits of programs implemented pursuant to the Clean Air Act (CAA). The first analysis conducted was a retrospective analysis, addressing the original CAA and covering the period 1970 to 1990. The retrospective was completed in 1997. Section 812 also required performance of prospective cost-benefit analyses, the first of which was completed in 1999. The prospective analyses address the incremental costs and benefits of the CAAA. The first prospective covered implementation of the CAAA over the period 1990 to 2010.

EPA's Office of Air and Radiation (OAR) began work on the second prospective with the drafting of an analytical plan for the study. This analytical plan was reviewed by a statutorily-mandated outside peer review group, the Advisory Council for Clean Air Compliance Analysis (Council), and the Council provided comments, which have been incorporated into the technical analysis planning. This report describes the development of quantified and monetized primary benefits associated with emissions reductions estimated for the second prospective section 812 analysis. Exhibit 1-1 below outlines the relationship among the section 812 Retrospective, the First Prospective, and the Second Prospective.

Exhibit 1-1
812 Scenarios: Conceptual Schematic



The scope of this analysis is to estimate the benefits of reducing emissions of criteria pollutants under two scenarios, depicted in schematic form in Exhibit 1-1 above:

1. An historical, "*with-CAAA*" scenario control case that reflects expected or likely future measures implemented since 1990 to comply with rules promulgated through September 2005[1]; and

2. A counterfactual "without CAAA" scenario baseline case that freezes the scope and stringency of emissions controls at their 1990 levels, while allowing for changes in population and economic activity and, therefore, in emissions attributable to economic and population growth.

Criteria pollutant emissions reductions addressed in this analysis include: volatile organic compounds (VOCs), oxides of nitrogen (NO_x), sulfur dioxide (SO_2), particulate matter of 10 microns or less (PM_{10}), and particulate matter with an aerodynamic diameter of 2.5 microns or less ($PM_{2.5}$). Benefits estimates, however, focus not on the emissions but on the ambient air concentration outcomes that result from emissions changes attributed to implementation of the Clean Air Act Amendments. The two major ambient pollutants for which benefits estimates are readily available are fine particulate matter and tropospheric ozone. Air quality changes associated with changes in emissions of lead, the remaining criteria pollutant under the Clean Air Act, are not addressed in this report, and were not addressed in the first prospective analysis, because of the relatively modest impact of CAAA regulations in place by 2005 on lead emissions.[2]

This report presents the results of EPA's analysis of the future effects of implementation of the CAAA's programs on air emissions from the following emission sectors: electricity generating units (EGUs), non-electricity generating unit point sources, nonroad engines/vehicles, on-road vehicles, and nonpoint sources. The study years for the analysis are 1990, 2000, 2010, and 2020. Because the CAAA was signed into law in 1990, emissions and air quality changes attributed to its implementation were not realized until after that point. As a result, benefits are estimated only for the target years 2000, 2010, and 2020.

The purpose of this report is to present the methods used to generate estimates of physical and economic benefits that result from the CAAA, and to present the results of our analyses for each target year. The scope of the benefits analyses conducted to support the second prospective analysis includes the following:

- ***Health Benefits***: These include avoided premature mortality and avoided morbidity associated with reduced human exposures to air pollutants.
- ***Visibility Benefits***: Reductions in air pollutants, particularly fine particulate matter, improve visibility, leading to physical and economic benefits in both recreational and residential settings.

[1] The lone exception is the Coke Ovens Residual Risk rulemaking, promulgated under Title III of the Act in March 2005. We omitted this rule because it has a very small impact on criteria pollutant emissions (less than 10 tons per year VOCs) relative to the with-CAAA scenario. The primary MACT rule for coke oven emissions, however, involves much larger reductions and therefore is included in the with-CAAA scenario.

[2] Lead emissions were effectively controlled under regulations authorized by the original Clean Air Act. As a result, analysis of lead emissions is a major focus of the section 812 retrospective study. Recently finalized revisions to the lead NAAQS could have significant effects on emissions for some localities, but those changes were first proposed on May 1, 2008 and were therefore not included in the scope of this analysis.

- ***Agricultural and Forest Productivity Benefits***: Tropospheric ozone inhibits plant growth; as a result, reduction in ozone concentrations yield physical and economic benefits in the form of enhanced agricultural and forest productivity.
- ***Materials Damage Benefits***: Some materials are susceptible to accelerated deterioration when exposed to air pollution; as a result, reduction in air pollution can extend the life of these materials, yielding physical and economic benefits.
- ***Ecological Benefits***: A wide range of ecological resources are susceptible to damage when exposed to ambient air pollution or deposition of pollutants to terrestrial or aquatic environments. For a small portion of these effects, it is possible to quantify and estimate the economic value of avoided pollutant exposure. As outlined below, quantified and monetized ecological benefits of the CAAA are included in our summary of the benefits of CAAA programs presented later in this chapter. The methods and data used to generate these estimates are not described in this report, but in an accompanying EPA report prepared to support the second prospective analysis.

Relationship of This Report to Other Second Prospective Analyses

The benefits estimates presented in this report rely on results generated in prior analytic components of the overall second prospective effort. As illustrated in Exhibit 1-2, EPA conducted both emissions estimation and air quality modeling analyses to generate data that underlies the benefits estimation approaches. EPA plans to make full reports on each of these major analytic steps available to the public online at the project website, www.epa.gov/oar/sect812. Details on the use of air quality inputs in the health, visibility, agricultural, forestry, and materials damage analyses are provided in the subsequent chapters of this report. In almost all cases, some post-processing of air quality data is involved to estimate pollutant exposures appropriate to the specific benefits analysis.

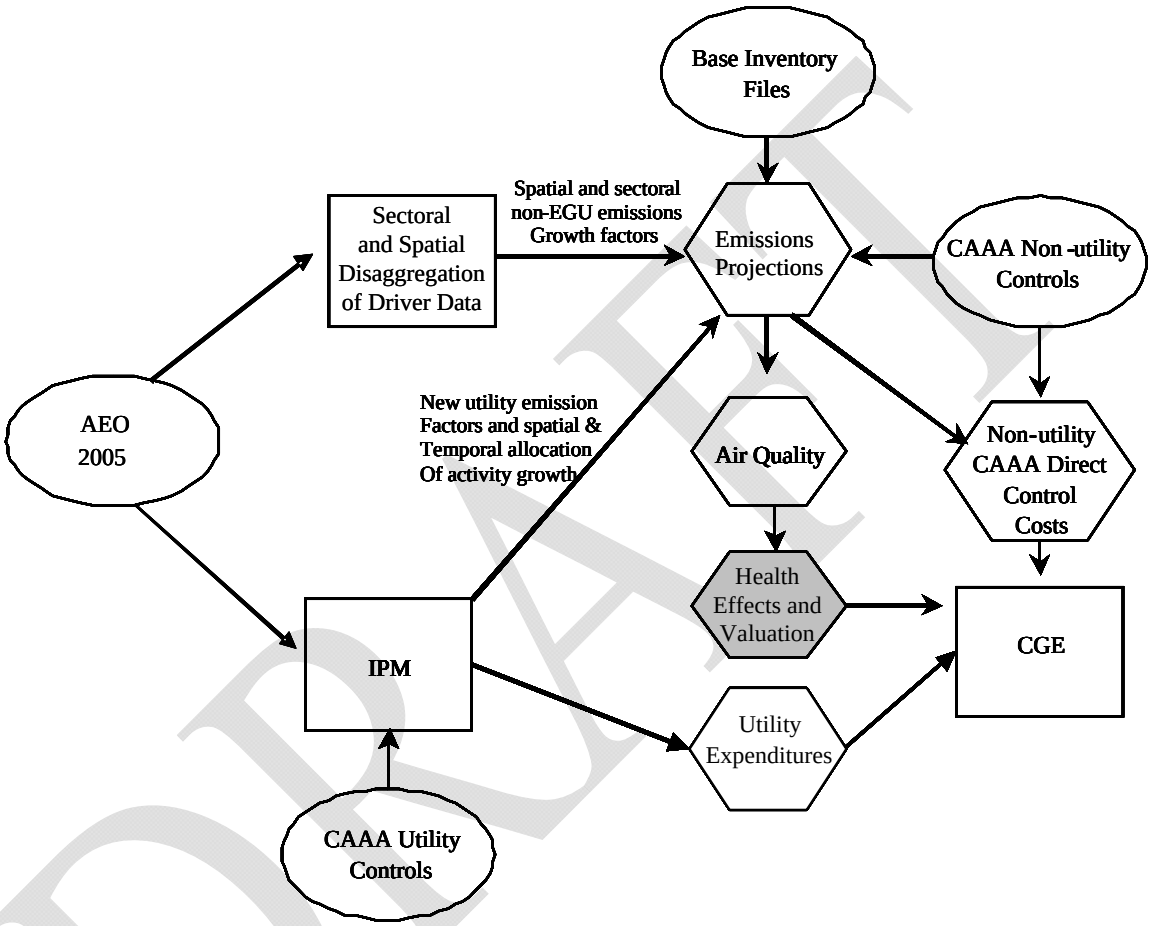
This report focuses on presentation of the primary benefits estimates. The primary benefits estimates are based on EPA's preferred set of analytic assumptions, models, and data sources, many of which have been explicitly reviewed by EPA Science Advisory Board over the course of many years and have been embodied in standard benefits estimation practice as carried out by EPA's Office of Air and Radiation in Regulatory Impact Analyses (RIAs). As an integral part of preparing the primary benefits results, EPA also conducted a series of analyses to estimate uncertainty in the primary results. The methods and results of these uncertainty and sensitivity analyses are described in a separate report, *Uncertainty Analyses to Support the Second Section 812 Prospective Benefit-Cost Analysis of the Clean Air Act*.

In addition, as noted above, estimation of the ecological benefits of the CAAA are described in detail in a separate report, *Ecological Benefits Analyses to Support the Second Section 812 Prospective Benefit-Cost Analysis of the Clean Air Act*. The ecological benefits report addresses the estimation of quantified ecological benefits, including estimates of the value of reduced lake acidification in the Adirondacks region of New York State, but also characterizes a range of unquantifiable ecological impacts through an exhaustive literature review and presentation of maps showing the relation between prevented air pollutant exposure and selected sensitive ecological receptors.

Within each of the following chapters, there is a brief discussion of the scope of quantified and monetized benefits. In addition, we include a brief discussion of other, unquantified benefits of the Clean Air Act. With the completion and review of the benefits analyses, the Agency will prepare an integrated report for the entire project. The integrated report will address each of these major analytic components,

and present comparisons of benefits and costs for each of the target years, as well as uncertainty analyses that characterize confidence in these results.

Exhibit 1-2
May 2003 Analytical Plan - Schematic Flow Chart



Overview of Methods

The methods applied in this report generally follow approaches developed by EPA over many years to support Regulatory Impact Analyses for major Office of Air and Radiation rulemakings, prior Section 812 analyses, and other Agency economic analyses. In a few cases, summarized below, this Second Prospective reflects methodologies, data, or benefits categories that are new to Agency analysis. In general, the primary benefits results presented here reflect methods, data, and benefits that have been vetted through EPA Science Advisory Board review, as well as internal EPA review by OAR economists and benefits analysts.

The general method we apply to quantify and monetize benefits involves four basic steps:

1. ***Access the relevant air quality results from the suite of Second Prospective CMAQ runs***. The Community Multiscale Air Quality (CMAQ) data include estimates of ambient air quality measured as concentrations of particulate matter and ozone, estimates of visibility expressed in deciviews, and estimates of deposition measured as a deposition flux per unit area.

2. ***Estimate exposure for each scenario***. Exposure analyses can vary by endpoint – for example, most health endpoints use an 8-hour maximum measure, while the agricultural analyses use a cumulative measure of ozone exposure over a growing season.

3. ***Estimate changes in physical effects.*** Physical effects are quantified benefits (e.g., cases of chronic bronchitis) attributable to CAAA regulations, and are generated based on differences in exposure between scenarios. A few effects, such as visibility, are estimated for both scenarios, rather than based on differences in exposure.

4. ***Value changes in effects***. In most cases, this step involves application of a unit economic value. The unit values reflect willingness to pay to avoid a small risk of incidence of a health effect; they are not values to avoid a certain health effect. In a few cases, valuation is directly estimated from air quality outcomes, applies avoided cost methods rather than willingness to pay, or is combined with step 3 in an integrated approach or model.

Exhibit 1-3 summarizes our approach to steps 2 through 4 above for each major category of benefits. Detailed descriptions of these approaches are provided in the subsequent chapters.

Exhibit 1-3. Summary of Estimation Approach for Major Benefits Categories

<table>
<tr><th>Benefit Category</th><th>Exposure Estimation</th><th>Physical Effects Estimation</th><th>Economic Value Estimation</th></tr>
<tr><td>Health Effects</td><td>Model Attainment Test Software (MATS) for PM; Enhanced Voronoi Neighbor Averaging (eVNA) for ozone</td><td colspan="2">Benefits Mapping and Analysis Program (BenMAP)</td></tr>
<tr><td>Visibility</td><td colspan="2">CMAQ-derived deciview estimates</td><td>Custom benefits transfer models</td></tr>
<tr><td>Agriculture and Forest Productivity</td><td>eVNA extrapolation , BenMAP procedure, and offline GIS analysis</td><td>NCLAN-based concentration-response functions</td><td>Forest and Agricultural Sector Optimization Model (FASOM)</td></tr>
<tr><td>Materials Damage</td><td colspan="3">Air Pollution Emissions Experiments and Policy (APEEP) model</td></tr>
<tr><td>Lake Acidification</td><td>CMAQ deposition outputs</td><td>Model of Acidification of Groundwater in Catchments (MAGIC)</td><td>Custom random-utility model for Adirondack lakes</td></tr>
<tr><td colspan="4">Note: Models and approaches are described in detail in Chapters 2 through 5 of this report.</td></tr>
</table>

Summary of Results

Exhibit 1-4 below provides a summary of the economic benefits results generated for the categories of benefits address in this report.

Exhibit 1-4. Summary of Mean Primary Benefits Results

Benefit Category	Monetized Benefits (million 2006$) by Target Year			Notes
	2000	2010	2020	
Health Effects				
- PM Mortality	$460,000	$730,000	$1,100,000	- PM mortality estimates based on Pope et. al (2002)
- PM Morbidity	32,000	52,000	76,000	
- Ozone Mortality	4,300	14,000	26,000	- Ozone mortality estimates based on pooled C/R function
- Ozone Morbidity	420	1,300	2,100	
Subtotal Health Effects	$500,000	$800,000	$1,200,000	
Visibility				
- Recreational				
- Residential				
Subtotal Visibility	[Not available for this draft]			
Agricultural and Forest Productivity	[Not available for this draft]			
Materials Damage	[Not available for this draft]			
Ecological	[Not available for this draft]			
Total: all categories	[Not available for this draft]			
Note: See Chapters 2 through 5 of this report for detailed results summaries. All estimates are mean values from distributions of the primary estimate results. Additional, alternative estimates are provided in the separate companion report on uncertainty. Estimates presented with two significant figures.				

The health effects estimates for the second prospective are much larger than the estimates EPA developed for the first prospective. The 2020 estimates are new to the second prospective, but the comparable mean estimate of health benefits in 2000 and 2010 for the first prospective were $71 billion in 2000 and $110 billion in 2010, in 1990$[3] - if updated to 2006$, these estimates would be $110 billion in 2000 and $170 billion in 2010. There are six key reasons we have identified for the nearly five-fold increase in benefits:

1. ***Scenario differences*****:** The with-CAAA scenario, especially for the 2010 target year, includes new rules with substantial additional pollutant reductions that were not included in the comparable first prospective scenario, such as the Clean Air Interstate Rule (CAIR).

2. ***Emissions differences*****:** EPA has substantially improved techniques for measuring emissions inventories and the impacts of regulation on emissions performance. In particular, improved methods for estimating the primary and co-control benefits for directly emitted fine particulates and ammonia have resulted in substantial additional emissions reductions for these pollutants attributed to the CAAA, compared to the first prospective. These two pollutants also tend to have a large effect on estimated ambient fine particulate matter concentrations and, therefore, a large effect on estimated mortality incidence and economic benefits.

3. ***Improved air quality models*****:** The first prospective relied on the Regional Acid Deposition Model/Regional Particulate Model (RADM/RPM) for PM and deposition estimates in the eastern U.S., the Regulatory Modeling System for Aerosols and Acid Deposition (REMSAD) for PM estimates in the western U.S., and the Urban Airshed Model (versions V and IV) at various regional and urban scales to generate ozone estimates. The second prospective relies on the integrated CMAQ modeling tool, which reflects substantial improvements in air quality

[3] See The Benefits and Costs of the Clean Air Act 1990 to 2010, USEPA Office of Air and Radiation and Office of Policy, EPA-410-R-99-001, November 1999.

modeling, provides more comprehensive spatial coverage, and achieves improved model performance.

4. ***Better, more comprehensive exposure estimates*:** The first prospective relied on first generation exposure extrapolation tools to generate monitor-adjusted exposure estimates away from monitors. Since then, the monitor network, availability of speciated data, and the performance of speciated exposure estimation tools have improved substantially.

5. ***Updated dose-response estimates***: Since 1999, health effects research has address endpoints that were not covered in the first prospective. The most notable is premature mortality associated with ozone exposure. In addition, some concentration response functions have been updated (e.g., the PM-premature mortality C/R function).

6. ***New valuation estimates:*** As described in Chapter 2, the estimates reported here make use of an updated Value of Statistical Life based on the Viscusi and Aldy (2003) VSL meta-analysis.

Although the Agency has not yet conducted a rigorous quantitative analysis to assess the impact of these methodology and data improvements, the impact of most of these factors is to increase the estimates of benefits. For example, based on limited analysis of the combined effect of factors 1 and 2, scenario and emissions differences, we estimate that improved methods and data account for a roughly two to three-fold difference in monetized benefits.

Organization of this Report

The remainder of this report is organized as follows. First, we present methods, data, and results for health effects and their valuation. As noted above, the health benefits constitute the majority of the monetized benefits in our analysis. Second, we present benefits associated with changes in visibility in both recreational and residential settings. Third, we present benefits associated with changes in productivity of agricultural crops and commercial forests. Fourth, we present benefits associated with reduced materials damage, including such resources as bridges, architectural coatings, and other materials that can be damaged by air pollution. The report concludes with aggregation and summary of all four of these categories of primary benefits.

[Note: draft results presented for review by the Health Effects Subcommittee of the SAB Council include only Chapters 1 and 2 of this report. Other components of the report will be reviewed by the Ecological Effects Subcommittee, the Council, or both.]

CHAPTER 2 – ESTIMATION OF HUMAN HEALTH EFFECTS AND ECONOMIC BENEFITS

Overview of Approach

This chapter addresses the economic valuation of human health effects realized as a result of the CAAA. The reduced incidence of physical effects is a valuable measure of health benefits for individual endpoints. 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 second prospective section 812 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 visibility, agricultural and ecological benefits, and materials damage, which are covered in Chapters 3 through 5. We obtain valuation estimates from the economic literature and report them in "dollars per case reduced" for health effects. 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 log-normal 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.

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 on the valuation of human health effects attributed to the reduction criteria pollutant emissions.[1] 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. As described in detail in this chapter, there are also significant reductions in short term and chronic health effects and a substantial number of health benefits that we could not quantify or monetize.

Similar to the first section 812 prospective analysis, the study design adopted for this analysis uses 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 with-CAAA scenario and the without-CAAA scenario), as documented in the Emissions Projections for the Clean Air Act Second Section 812 Prospective Analysis.[2] The emissions estimates were input into the Community Multiscale Air Quality (CMAQ) model and, in turn, ambient pollutant concentrations estimated by CMAQ were input into the Environmental Benefits Mapping and Analysis Program (BenMAP).

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[1] OAR's First Prospective analysis of the Costs and Benefits of the Clean Air Act Amendments included a detailed analysis of the health and welfare benefits of Title VI provisions. That analysis concluded that the benefits of the Title VI stratospheric ozone protection programs were very large compared to the costs. For the Second Prospective analysis, EPA has decided that updating the prior analysis likely would provide little in the way of additional insights. As a result, the Second Prospective analysis focuses on benefits and costs of criteria pollutant programs.

[2] See EH Pechan and Industrial Economics, *Emission Projections for the Clean Air Act Second Section 812 Prospective Analysis: Revised Draft Report*, March 2009, available at www.epa.gov/oar/sect 812.

BenMAP is a tool developed by the U.S. Environmental Protection Agency (EPA) for estimating the human health effects and economic benefits associated with changes in ambient air pollution.[3] BenMAP relies on three inputs: 1) forecasted changes in air quality between a baseline and control scenario; 2) health impact functions that quantify the relationship between the forecasted changes in exposure and expected changes in specific health effects; and 3) health valuation functions that assign a monetary value to changes in specific health effects. From these inputs, BenMAP compares changes in pollutant exposure between two scenarios and produces results in terms of avoided health effects and monetary valuation of the willingness to pay to avoid those effects. This chapter begins by discussing methods used to quantify changes in air quality and how that is interpreted for human exposure to specific pollutants, goes on to describe the health impact functions used, and then details the health valuation functions applied. The chapter concludes with a presentation and discussion of the results.

Quantifying Changes in Air Quality

This analysis is the first Section 812 prospective analysis to use an integrated modeling system, the Community Multiscale Air Quality (CMAQ) model, to simulate changes in national and regional-scale pollutant concentrations and deposition. CMAQ has previously been deployed in several EPA economic analyses including the 2008 Ozone National Ambient Air Quality Standards (NAAQS) Regulatory Impact Analysis (RIA) (EPA, 2008) and the 2006 PM NAAQS RIA (EPA, 2006b). The CMAQ model (Byun and Ching, 1999) is a state-of-the-science, regional air quality modeling system that is designed to simulate the physical and chemical processes that govern the formation, transport, and deposition of gaseous and particulate species in the atmosphere. The latest version of CMAQ (Version 4.6) was employed for this analysis.

The CMAQ model was applied for seven core CAAA scenarios that include four different years that span a 30-year period – 1990, 2000, 2010 and 2020. Scenarios that incorporate the emission reductions associated with the CAA are referred to as with-CAAA while those that do not are referred to as without-CAAA. The scenarios include:

Retrospective Base-Year Scenario
- 1990 without-CAAA

Base and Projected Year Scenarios without 1990 CAAA Controls
- 2000 without-CAAA
- 2010 without-CAAA
- 2020 without-CAAA

Base and Projected Year Scenarios with 1990 CAAA Controls
- 2000 with-CAAA
- 2010 with-CAAA
- 2020with-CAAA

An integral component of the modeling analysis is the estimation of future-year emissions for the seven core scenarios – these are described in detail in companion reports available at EPA's Section 812 study website.[4] Emissions for the historical years (1990 and 2000) were based on the best available

[3] This analysis uses BenMAP Version 3.0.16. The current version of BenMAP can be downloaded from http://www.epa.gov/air/benmap/

[4] See www.epa.gov/oar/sect812

emission inventories for these years. Projection to the future years was based on economic growth projections, future-year control requirements (for attainment of NAAQS), and control efficiencies. Different assumptions were applied for the with- and without-CAAA scenarios resulting in a different future-year emissions pathway for each scenario. The emissions data were processed for input to the CMAQ modeling using the Sparse-Matrix Operator Kernel Emissions (SMOKE) emissions processing system.

The model-ready emission inventories for each scenario and year were then used to obtain base- and future-year estimates of the key criteria pollutants, as well as many other species. The air quality modeling analysis was designed to make use of tools and databases that have recently been developed and evaluated by EPA for other national- and regional-scale air quality modeling studies. In particular, model-ready meteorological input files for 2002 were provided by EPA for use in this study. For fine particulate matter ($PM_{2.5}$) and related species, the CMAQ model was applied for an annual simulation period (January through December). A 36-km resolution modeling domain that encompasses the contiguous 48 states was used for the annual modeling (see Exhibit 2-1). For ozone and related species, the CMAQ model was applied for a five-month simulation period that captures the key ozone-season months of May through September. Two 12-km resolution modeling domains (that when combined cover the contiguous 48 U.S. states) were used for the ozone-season modeling (see Exhibit 2-1). Altogether, model-ready emission inventories were prepared and the CMAQ model was applied for a total of 21 simulations (comprising seven core scenarios and three modeling domains).[5]

$PM_{2.5}$ and ozone outputs from CMAQ provide the basis of the air quality inputs needed for BenMAP. The raw CMAQ output is adjusted to take into account monitor data. The $PM_{2.5}$ output is adjusted using the Modeled Attainment Test Software (MATS, Version 2.1.1, Build 807) procedure and the ozone output is adjusted using the enhanced Voronoi Neighbor Averaging (eVNA) routine in BenMAP.

MATS estimates quarterly mean $PM_{2.5}$ chemical component concentrations at monitor locations (point estimates) by conducting a Speciated Modeled Attainment Test (SMAT) analysis. MATS can also estimate quarterly mean concentration estimates for each PM2.5 chemical component concentrations at all grid cells in an Eulerian grid model such as CMAQ using a spatial field gradient interpolation procedure. All PM2.5 concentration estimates for this analysis were prepared using the spatial and temporal relative adjustment method in MATS. PM2.5 concentration estimates in CMAQ grid cells without a monitor were interpolated from nearby monitors using the inverse distance squared weighting option in the Voronoi Neighbor Averaging (VNA) procedure in MATS. The MATS analysis conducted for the PM2.5 used the following input information:

- Observed $PM_{2.5}$ data from 1,336 Federal Reference Method (FRM) monitors with sufficient data in at least one year from 2002 to 2004 (as provided with the MATS Version 2.1.1 installation package);
- Observed chemically speciated fine particle mass data from both the $PM_{2.5}$ Speciated Trends Network (STN) and the Interagency Monitoring of Protected Visual Environments (IMPROVE) network, a total of 420 monitors with sufficient data in as least one year from 2002 to 2004 (as provided with the MATS Version 2.1.1 installation package);

[5] A detailed report on the air quality modeling analyses was prepared for EPA. This description is based on the September 2008 draft report, Second Prospective Analysis of Air Quality in the U.S.: Air Quality Modeling, prepared for James DeMocker of the EPA Office of Policy Analysis and Review by Sharon G. Douglas, Jay L. Haney, A. Belle Hudischewskyj, Thomas C. Myers, and Y. Wei of ICF International.

- Speciated CMAQ estimates for 6 $PM_{2.5}$ species (SO_4, NO_3, elemental carbon, organic carbon, NH_4, and crustal material) at the 36 kilometer PM CMAQ grid cell level for each of the scenarios (from CMAQ speciated output data files provided by ICF/SAI).

Additional detail on the MATS procedure is available in the MATS User Manual (Abt Associates, 2009). MATS produced estimated average quarterly concentrations for each of the CMAQ 36 km grid cells. These estimates were subsequently rewritten to the format required for inputting daily PM2.5 data into EPA's BenMAP software.

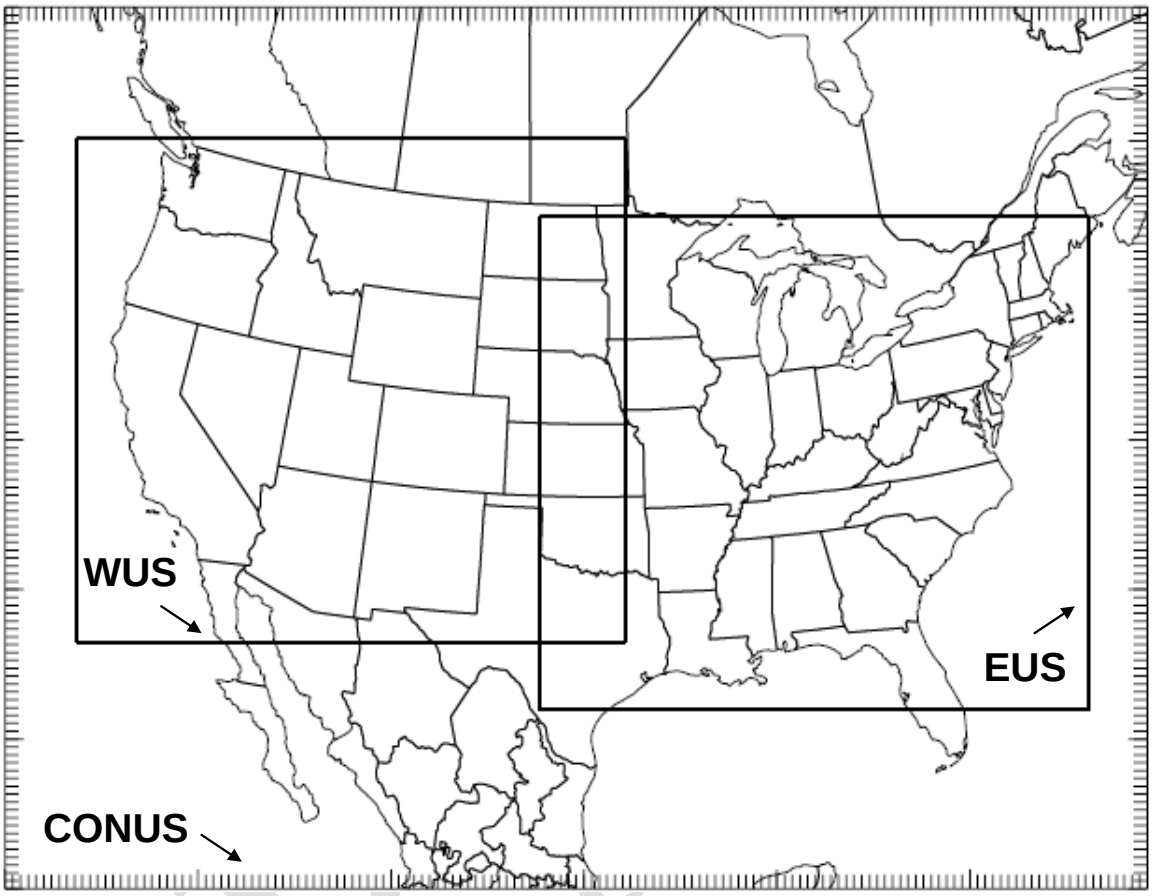
The daily ozone concentration estimates used in this analysis were prepared using a monitor and model relative adjustment procedure, combining the hourly CMAQ estimates with observed ozone monitor data. The monitor and model relative adjustment procedure was conducted using the extended VNA procedure (eVNA) with both spatial and temporal scaling in EPA's BenMAP software. The 1,162 ozone monitors used in the eVNA procedure were the 2002 ozone monitors contained in the BenMAP (ver. 3.0.15) US Setup installation file. The 2002 monitor data was selected because the base case CMAQ analysis ("2000 with Clean Air Act") used a 2002 emission inventory. The CMAQ ozone estimates were prepared for the two separate eastern and western United States domains shown in Exhibit 2-1, each with a 12 kilometer by 12 kilometer grid.

Health Impact Functions[6]

Health impact functions measure the change in a health endpoint of interest, such as hospital admissions, for a given change in ambient ozone or $PM_{2.5}$ concentration. There are several types of data that can support the development of health impact functions relating air pollutant exposure or ambient concentrations to incidence of health outcomes. These sources of data include toxicological studies (including animal and cellular studies), human clinical trials, observational epidemiology studies, and meta-analyses of multiple epidemiology studies. All of these data sources provide important contributions to the weight of evidence surrounding a particular health impact, however, only epidemiology studies provide direct concentration-response (C-R) relationships which can be used to evaluate population-level impacts of reductions in ambient pollution levels.

[6] Portions of this section were derived from the PM NAAQS RIA (EPA, 2006b) and the Ozone NAAQS RIA (EPA, 2008).

Exhibit 2-1. Map of the CMAQ Modeling Domains Used for Second Section 812 Prospective Analysis



Legend:

CONUS: Continental U.S. 36 km grid, PM2.5 and deposition estimates
EUS: Eastern U.S. 12 km grid, ozone estimates
WUS: Western U.S. 12 km grid, ozone estimates

However, standard environmental epidemiology studies provide only a limited representation of the uncertainty associated with a specific health impact function, measuring only the statistical error in the estimates, and usually relating more to the power of the underlying study (driven largely by population size and the frequency of the outcome measure). There are many other sources of uncertainty in the relationships between ambient pollution and population level health outcomes, including many sources of model uncertainty, such as model specification, potential confounding between factors that are both correlated with the health outcome and each other, and many other factors. As such, in recent years, EPA has begun investigating how expert elicitation methods can be used to integrate across various sources of data in developing health impact functions for regulatory benefits analyses.

Expert elicitation is useful in integrating the many sources of information about uncertainty in the health impact function, because it allows experts to synthesize these data sources using their own mental models, and provide a probabilistic representation of their synthesis of the data in the form of a probability distribution of the health impact function. EPA has used expert elicitation to inform the regulatory process in the past (see for example the staff paper for the lead NAAQS (EPA, 1990) and the

PM NAAQS RIA (EPA, 2006b)). In the current analysis, we have used expert elicitation to characterize one representation of the health impact function for the relationship between $PM_{2.5}$ and premature mortality. However, similar methods could be used to characterize health impact functions for other health outcomes.

A standard health impact function has four components: 1) an effect estimate from a particular study; 2) a baseline incidence rate for the health effect (obtained from either the epidemiology study or a source of public health statistics such as the Centers for Disease Control); 3) the size of the potentially affected population; and 4) the estimated change in the relevant ozone or PM summary measures.

A typical health impact function might be of the following generic form:

$$\Delta y = y_0 \cdot \left(e^{\beta \cdot \Delta x} - 1\right),$$

where y_0 is the baseline incidence (the product of the baseline incidence rate times the potentially affected population), β is the effect estimate, and Δx is the estimated change in the summary ozone or $PM_{2.5}$measure. There are other functional forms, but the basic elements remain the same. The ozone and PM air quality inputs to the health impact functions are described in the section above. The following subsections describe the sources for each of the other elements: size of potentially affected populations; effect estimates; and baseline incidence rates.

Potentially Affected Populations

The starting point for estimating the size of potentially affected populations is the 2000 U.S. Census block level dataset (Geolytics 2002). BenMAP incorporates 250 age/gender/race categories to match specific populations potentially affected by ozone and $PM_{2.5}$. The software constructs specific populations matching the populations in each epidemiological study by accessing the appropriate age-specific populations from the overall population database. To estimate population levels for the years after 2000, BenMAP scales the 2000 Census-based population estimate with the ratio of the county-level forecast for the future year of interest over the 2000 county-level population level. Woods & Poole (2007) provides the county-level population forecasts used to calculate the scaling ratios.

Health Effect Estimate Sources

The most significant monetized benefits of reducing ambient concentrations of ozone and PM are attributable to reductions in human health risks. EPA's Ozone and PM Criteria Documents outline numerous health effects known or suspected to be linked to exposure to ambient ozone and PM (EPA, 2006; Anderson et al., 2004). EPA recently evaluated the ozone and PM literature for use in the benefits analyses for the Ozone NAAQS RIA (EPA, 2008) and PM NAAQS RIA (EPA, 2006b), respectively. The discussion of individual effect estimates presented in this section relies heavily on the research done for these RIAs.

Exhibit 2-2 lists the human health effects of ozone and $PM_{2.5}$. Exhibit 2-3 and 2-4 lists the health endpoints associated with ozone and $PM_{2.5}$, respectively, included in this analysis. A number of endpoints that are not health-related may also contribute significant monetized benefits. Welfare benefits such as increased recreational and residential visibility, increased recreational fishing opportunities, increased commercial forest and agriculture productivity, and decreased building materials damage are discussed in Chapters 3 through 5.

Exhibit 2-2. Human Health Effects of Ozone and $PM_{2.5}$

Pollutant/Effect	Quantified and Monetized in Base Estimates[a]	Unquantified Effects[g,h]—Changes in:
PM/Health[b]	Premature mortality based on both cohort study estimates and on expert elicitation[c,d] Bronchitis: chronic and acute Hospital admissions: respiratory and cardiovascular Emergency room visits for asthma Nonfatal heart attacks (myocardial infarction) Lower respiratory symptoms Minor restricted-activity days Work loss days Asthma exacerbations (asthmatic population) Upper Respiratory symptoms (asthmatic population) Infant mortality	Subchronic bronchitis cases Low birth weight Pulmonary function Chronic respiratory diseases other than chronic bronchitis Morphological changes Altered host defense mechanisms Cancer Non-asthma respiratory emergency room Visits UVb exposure (+/-)[e] Stroke/cerebrovascular disease
Ozone/Health[f]	Premature mortality: short-term exposures Hospital admissions: respiratory Emergency room visits for asthma Minor restricted-activity days School loss days Outdoor worker productivity	Cardiovascular emergency room visits Asthma attacks Respiratory symptoms Chronic respiratory damage Increased responsiveness to stimuli Inflammation in the lung Premature aging of the lungs Acute inflammation and respiratory cell damage Increased susceptibility to respiratory infection Non-asthma respiratory emergency room Visits UVb exposure (+/-)[e]

[a] Primary quantified and monetized effects are those included when determining the primary estimate of total monetized benefits of the alternative standards.

[b] In addition to primary economic endpoints, there are a number of biological responses that have been associated with PM health effects including morphological changes and altered host defense mechanisms. The public health impact of these biological responses may be partly represented by our quantified endpoints.

[c] Cohort estimates are designed to examine the effects of long-term exposures to ambient pollution, but relative risk estimates may also incorporate some effects due to shorter term exposures (see Kunzli, 2001 for a discussion of this issue).

[d] While some of the effects of short-term exposure are likely to be captured by the cohort estimates, there may be additional premature mortality from short-term PM exposure not captured in the cohort estimates included in the primary analysis.

[e] May result in benefits or disbenefits.

[f] In addition to primary economic endpoints, there are a number of biological responses that have been associated with ozone health including increased airway responsiveness to stimuli, inflammation in the lung, acute inflammation and respiratory cell damage, and increased susceptibility to respiratory infection. The public health impact of these biological responses may be partly represented by our quantified endpoints.

[g] The categorization of unquantified health effects is not exhaustive.

[h] Health endpoints in the unquantified benefits column include both a) those for which there is not consensus on causality and b) those for which causality has been determined but empirical data are not available to allow calculation of benefits.

Exhibit 2-3. Ozone Related Health Endpoints Basis for the Health Impact Function Associated with that Endpoint, and Sub-Populations for which They Were Computed

Endpoint	Pollutant	Study	Study Population
Premature Mortality			
Premature mortality—nonaccidental	O3 (8-hour max)	Ito et al. (2005) Schwartz (2005) Bell et al. (2004)	All ages
Premature mortality—all cause	O3 (8-hour max)	Bell et al. (2005) Levy et al. (2005)	All ages
Premature mortality—cardiopulmonary	O3 (8-hour max)	Huang et al. (2005)	All ages
Hospital Admissions			
Respiratory	O3 (8-hour max)	Pooled estimate: Schwartz (1995)—ICD 460–519 (all respiratory) Schwartz (1994a; 1994b)—ICD 480–486 (pneumonia) Moolgavkar et al. (1997)—ICD 480–487, 490–496 (pneumonia, COPD) Schwartz (1994b)—ICD 491–492, 494–496 (COPD)	>64 years
Respiratory	O3 (8-hour max)	Burnett et al. (2001)	<2 years
Asthma-related ER visits	O3 (8-hour max)	Pooled estimate: Jaffe et al (2003) Peel et al (2005) Wilson et al (2005)	 5–34 years All ages All ages
Other Health Endpoints			
Minor restricted-activity days	O3 (24-hour avg)	Ostro and Rothschild (1989)	18–64 years
School loss days	 O3 (8-hour avg) O3 (1-hour max)	Pooled estimate: Gilliland et al. (2001) Chen et al. (2000)	5–17 years[a]
Outdoor worker productivity	O3 (8-hour max)	Crocker and Horst (1981)	18–64 years

[a] Gilliland et al. (2001) studied children aged 9 and 10. Chen et al. (2000) studied children 6 to 11. Based on recent advice from the National Research Council and the EPA SAB-HES, we have calculated reductions in school absences for all school-aged children based on the biological similarity between children aged 5 to 17.

Exhibit 2-4. PM Related Health Endpoints Basis for the Health Impact Function Associated with that Endpoint, and Sub-Populations for which They Were Computed

Endpoint	Pollutant	Study	Study Population
Premature Mortality			
Premature mortality—all-cause	PM2.5 (annual avg)	Pope et al. (2002) Laden et al. (2006)	>29 years >24 years
Premature mortality—all-cause[a]	PM2.5 (annual avg)	Expert Elicitation (IEc, 2006)	>29 years
Infant mortality—all-cause	PM2.5 (annual avg)	Woodruff et al. (1997)	Infant (<1 year)
Chronic Illness			
Chronic bronchitis	PM2.5 (annual avg)	Abbey et al. (1995)	>26 years
Nonfatal myocardial infarction	PM2.5 (24-hour avg)	Peters et al. (2001)	Adults (>18 years)
Hospital Admissions			
Respiratory	PM2.5 (24-hour avg)	Pooled estimate: Moolgavkar (2003)—ICD 490–496 (COPD) Ito (2003)—ICD 490–496, 480–487 (COPD, pneumonia)	>64 years
Respiratory	PM2.5 (24-hour avg)	Moolgavkar (2000a)—ICD 490–492, 494-496 (COPD, less asthma)	20–64 years
Respiratory	PM2.5 (24-hour avg)	Sheppard (2003)—ICD 493 (asthma)	<65 years
Cardiovascular	PM2.5 (24-hour avg)	Pooled estimate: Moolgavkar (2003)—ICD 390–429 (all cardiovascular) Ito (2003)—ICD 411–414, 429, 428 (ischemic heart disease, dysrhythmia, heart failure)	>64 years
Cardiovascular	PM2.5 (24-hour avg)	Moolgavkar (2000b)—ICD 390–429 (all cardiovascular)	20–64 years
Asthma-related ER visits	PM2.5 (24-hour avg)	Norris et al. (1999)	<18 years
Other Health Endpoints			
Acute bronchitis	PM2.5 (annual avg)	Dockery et al. (1996)	8–12 years
Lower respiratory symptoms	PM2.5 (24-hour avg)	Schwartz and Neas (2000)	7–14 years
Upper respiratory symptoms	PM2.5 (24-hour avg)	Pope et al. (1991)	9–11 years
Asthma exacerbation	PM2.5 (24-hour avg)	Pooled estimate: Ostro et al. (2001) (cough, wheeze, shortness of breath) Vedal et al. (1998) (cough)	6–18 years[b]
Minor restricted-activity days	PM2.5 (24-hour avg)	Ostro and Rothschild (1989)	18–64 years
Work loss days	PM2.5 (24-hour avg)	Ostro (1987)	18–64 years

[a] Mortality estimates based on the expert elicitation results are omitted from this draft – see text for explanation.

[b] The original study populations were 8 to 13 for the Ostro et al. (2001) study and 6 to 13 for the Vedal et al. (1998) study. Based on advice from the Science Advisory Board Health Effects Subcommittee (SAB-HES), we extended the applied population to 6 to 18, reflecting the common biological basis for the effect in children in the broader age group. See: U.S. Science Advisory Board. 2004. Advisory Plans for Health Effects Analysis in the Analytical Plan for EPA's Second Prospective Analysis – Benefits and Costs of the Clean Air Act, 1990—2020. EPA-SAB-COUNCIL-ADV-04-004. See also National Research Council (NRC). 2002. *Estimating the Public Health Benefits of Proposed Air Pollution Regulations*. Washington, DC: The National Academies Press.

Literature Sources for Ozone Health Effects Functions

Premature Mortality

While PM is the criteria pollutant most clearly associated with premature mortality, recent research suggests that short-term repeated ozone exposure also likely contributes to premature death. The 2006 Ozone Criteria Document states, "Consistent with observed ozone-related increases in respiratory- and cardiovascular-related morbidity, several newer multi-city studies, single-city studies, and several meta-analyses of these studies have provided relatively strong epidemiologic evidence for associations between short-term ozone exposure and all-cause mortality, even after adjustment for the influence of season and PM" (EPA, 2006a: 8-78). The epidemiologic data are also supported by recent experimental data from both animal and human studies, which provide evidence suggestive of plausible pathways by which risk of respiratory or cardiovascular morbidity and mortality could be increased by ambient ozone. With respect to short-term exposure, the Ozone Criteria Document concludes, "This overall body of evidence is highly suggestive that ozone directly or indirectly contributes to non-accidental and cardiopulmonary-related mortality, but additional research is needed to more fully establish underlying mechanisms by which such effects occur" (p. 8-78).

With respect to the time-series studies, the conclusion regarding the relationship between short-term exposure and premature mortality is based, in part, upon recent city-specific time-series studies such as the Schwartz (2005) analysis in Houston and the Huang et al. (2005) analysis in Los Angeles.[7] This conclusion is also based on recent meta-analyses by Bell et al. (2005), Ito et al. (2005), and Levy et al. (2005), and on analyses of the National Morbidity, Mortality, and Air Pollution Study (NMMAPS) data set by Bell et al. (2004), Schwartz (2005), and Huang et al. (2005). Consistent with the methodology used in the Ozone NAAQS RIA (2008), and with more recent advice in NAS (2008), we included ozone mortality in the primary health effects analysis, with the recognition that the exact magnitude of the effects estimate is subject to continuing uncertainty. In this chapter we present the mean of the incidence estimates derived from the three meta-analyses and the three NMMAPS-based studies listed above. The Uncertainty Analysis to Support the Second Section 812 Benefit-Cost analysis of the Clean Air Act (Uncertainty Analysis) includes estimates from all six studies separately. Use of these six studies represents a slight change from the Ozone NAAQS RIA (2008); two NMMAPS-based studies (Schwartz (2005) and Huang et al. (2005)) have been added based on guidance from the National Academy of Sciences (NAS) (2008).

Ozone Exposure Metric. Both the NMMAPS analyses and the individual time series studies upon which the meta-analyses were based use the 24-hour average or 1-hour maximum ozone levels as exposure metrics. The 24-hour average is not the most relevant ozone exposure metric to characterize population-level exposure. Given that the majority of the people tend to be outdoors during the daylight hours and concentrations are highest during the daylight hours, the 24-hour average metric is not appropriate. Moreover, the 1-hour maximum metric uses an exposure window different than that used for the current ozone NAAQS. Together, this means that the most biologically relevant metric, and the one used in the ozone NAAQS since 1997, is the 8-hour maximum standard. Thus, for this analysis, we have converted ozone mortality health impact functions that use a 24-hour average or 1-hour maximum ozone metric to maximum 8-hour average ozone concentration using a procedure described in the BenMAP

[7] For an exhaustive review of the city-specific time-series studies considered in the ozone staff paper, see: U.S. Environmental Protection Agency, 2007. Review of the National Ambient Air Quality Standards for Ozone: Policy Assessment of Scientific and Technical Information. Prepared by the Office of Air and Radiation. Available at http://www.epa.gov/ttn/naaqs/standards/ozone/data/2007_01_ozone_staff_paper.pdf. pp. 5-36.

user's manual (see Abt Associates, 2008). A similar method was used for the final Ozone NAAQS RIA (2008).

Respiratory Hospital Admissions

Detailed hospital admission and discharge records provide data for an extensive body of literature examining the relationship between hospital admissions and air pollution. This is especially true for the portion of the population aged 65 and older, because of the availability of detailed Medicare records. In addition, there is one study (Burnett et al., 2001) providing an effect estimate for respiratory hospital admissions in children less than two years of age.

Because the number of hospital admission studies we considered is so large, we used results from a number of studies to pool some hospital admission endpoints. Pooling is the process by which multiple study results may be combined in order to produce better estimates of the effect estimate, or β.[8] To estimate total respiratory hospital admissions associated with changes in ambient ozone concentrations for adults over 65, we first estimated the change in hospital admissions for each of the different effects categories that each study provided for each city. These cities included Minneapolis, Detroit, Tacoma and New Haven. To estimate total respiratory hospital admissions for Detroit, we added the pneumonia and chronic obstructive pulmonary disease (COPD) estimates, based on the effect estimates in the Schwartz study (1994b). Similarly, we summed the estimated hospital admissions based on the effect estimates the Moolgavkar study reported for Minneapolis (Moolgavkar et al., 1997). To estimate total respiratory hospital admissions for Minneapolis using the Schwartz study (1994a), we simply estimated pneumonia hospital admissions based on the effect estimate. Making this assumption that pneumonia admissions represent the total impact of ozone on hospital admissions in this city will give some weight to the possibility that there is no relationship between ozone and COPD, reflecting the equivocal evidence represented by the different studies. We then used a fixed-effects pooling procedure to combine the two total respiratory hospital admission estimates for Minneapolis. Finally, we used random effects pooling to combine the results for Minneapolis and Detroit with results from studies in Tacoma and New Haven from Schwartz (1995). As noted above, this pooling approach incorporates both the precision of the individual effect estimates and between-study variability characterizing differences across study locations.

Asthma-Related Emergency Room Visits

We used three studies as the source of the C-R functions we used to estimate the effects of ozone exposure on asthma-related emergency room (ER) visits: Peel et al. (2005); Wilson et al. (2005); and Jaffe et al. (2003). We estimated the change in ER visits using the effect estimate(s) from each study and then pooled the results using the random effects pooling technique (see Abt Associates, 2008). The study by Jaffe et al. (2003) examined the relationship between ER visits and air pollution for populations aged five to 34 in the Ohio cities of Cleveland, Columbus and Cincinnati from 1991 through 1996. In single-pollutant Poisson regression models, ozone was linked to asthma visits. We use the pooled estimate across all three cities as reported in the study. The Peel et al. study (2005) estimated asthma-related ER visits for all ages in Atlanta, using air quality data from 1993 to 2000. Using Poisson generalized estimating equations, the authors found a marginal association between the maximum daily 8-hour average ozone level and ER visits for asthma over a 3-day moving average (lags of 0, 1, and 2 days) in a single pollutant model. Wilson et al. (2005) examined the relationship between ER visits for respiratory illnesses and asthma and air pollution for all people residing in Portland, Maine from 1998–2000 and Manchester, New Hampshire from 1996–2000. For all models used in the analysis, the authors restricted the ozone data incorporated into the model to the months ozone levels are usually measured, the spring-

[8] For a complete discussion of the pooling process see Abt Associates, 2008.

summer months (April through September). Using the generalized additive model, Wilson et al. (2005) found a significant association between the maximum daily 8-hour average ozone level and ER visits for asthma in Portland, but found no significant association for Manchester. Similar to the approach used to generate effect estimates for hospital admissions, we used random effects pooling to combine the results across the individual study estimates for ER visits for asthma. The Peel et al. (2005) and Wilson et al. (2005) Manchester estimates were not significant at the 95 percent level, and thus, the confidence interval for the pooled incidence estimate based on these studies includes negative values. This is an artifact of the statistical power of the studies, and the negative values in the tails of the estimated effect distributions do not represent improvements in health as ozone concentrations are increased. Instead, these should be viewed as a measure of uncertainty due to limitations in the statistical power of the study. We included both hospital admissions and ER visits as separate endpoints associated with ozone exposure because our estimates of hospital admission costs do not include the costs of ER visits and most asthma ER visits do not result in a hospital admission.

Minor Restricted-Activity Days

Minor restricted-activity days (MRADs) occur when individuals reduce most usual daily activities and replace them with less-strenuous activities or rest, but do not miss work or school. We estimated the effect of ozone exposure on MRADs using a concentration-response function derived from Ostro and Rothschild (1989). These researchers estimated the impact of ozone and $PM_{2.5}$ on MRAD incidence in a national sample of the adult working population (ages 18 to 64) living in metropolitan areas. We developed separate coefficients for each year of the Ostro and Rothschild analysis (1976–1981), which we then combined for use in EPA's analysis. The effect estimate used in the impact function is a weighted average of the coefficients in Ostro and Rothschild (1989, Table 4), using the inverse of the variance as the weight.

School Loss Days

Children may be absent from school due to respiratory or other acute diseases caused, or aggravated by, exposure to air pollution. Several studies have found a significant association between ozone levels and school absence rates. We use two studies (Gilliland et al., 2001; Chen et al., 2000) to estimate changes in school absences resulting from changes in ozone levels. The Gilliland et al. study estimated the incidence of new periods of absence, while the Chen et al. study examined daily absence rates. We converted the Gilliland et al. estimate to days of absence by multiplying the absence periods by the average duration of an absence. We estimated 1.6 days as the average duration of a school absence, the result of dividing the average daily school absence rate from Chen et al. (2000) and Ransom and Pope (1992) by the episodic absence duration from Gilliland et al. (2001). Thus, each Gilliland et al. period of absence is converted into 1.6 absence days.

Following advice from the National Research Council (2002), we calculated reductions in school absences for the full population of school age children, ages five to 17. We estimated the change in school absences using both Chen et al. (2000) and Gilliland et al. (2001) and then, similar to hospital admissions and ER visits, pooled the results using the random effects pooling procedure.

Outdoor Worker Productivity

To monetize benefits associated with increased outdoor worker productivity resulting from improved ozone air quality, we used information reported in Crocker and Horst (1981). Crocker and Horst examined the impacts of ozone exposure on the productivity of outdoor citrus workers. The study measured productivity impacts. Worker productivity is measuring the value of the loss in productivity for a worker who is at work on a particular day, but due to ozone, cannot work as hard. It only applies to outdoor workers, like fruit and vegetable pickers, or construction workers. Here, productivity impacts are measured as the change in income associated with a change in ozone exposure, given as the elasticity of income with respect to ozone concentration. The reported elasticity translates a ten percent reduction in

ozone to a 1.4 percent increase in income. Given the national median daily income for outdoor workers engaged in strenuous activity reported by the U.S. Census Bureau (2002), $68 per day (2000$), a ten percent reduction in ozone yields about $0.97 in increased daily wages. We adjust the national median daily income estimate to reflect regional variations in income using a factor based on the ratio of county median household income to national median household income. No information was available for quantifying the uncertainty associated with the central valuation estimate. Therefore, no uncertainty analysis was conducted for this endpoint.

Literature Sources for PM Health Effects Functions

Adult Premature Mortality

A substantial body of published scientific literature documents the correlation between elevated PM concentrations and increased mortality rates (US EPA, 2004). Time-series methods have been used 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. Cohort methods have been used to examine the potential relationship between community-level PM exposures over multiple years (i.e., long-term exposures) and community-level annual mortality rates. Researchers have found statistically significant associations between PM and premature mortality using both types of studies. In general, the risk estimates based on the cohort studies are larger than those derived from time-series studies. Cohort analyses are thought to better capture the full public health impact of exposure to air pollution over time, because they capture the effects of long-term exposures and possibly some component of short-term exposures (Kunzli et al., 2001; NRC, 2002). To demonstrate the sensitivity of the benefits estimates to the specific sources of information regarding the impact of $PM_{2.5}$ exposures on the risk of premature death, we provide estimates in our results tables based on studies derived from the epidemiological literature and from the recent EPA sponsored expert elicitation. The studies from which these estimates are drawn are described briefly below.[9]

This analysis relies upon the unadjusted effect estimates (no-threshold) from two epidemiology studies examining the relationship between $PM_{2.5}$ and premature mortality using large population cohorts: the American Cancer Society (ACS) cohort (Pope et al., 2002) and the Harvard Six Cities cohort (Laden et al., 2006). Given their consistent results and broad geographic coverage, and importance in informing the NAAQS development process, the Six-Cities and ACS data have been particularly important in benefits analyses. The credibility of these two studies is further enhanced by the fact that the initial published studies (Pope et al, 1995 and Dockery et al 1993) were subject to extensive reexamination and reanalysis by an independent team of scientific experts commissioned by Health Effects Institute (HEI) (Krewski et al., 2000). Further confirmation and extension of the findings of the 1993 Six City study and the 1995 ACS study were completed using more recent air quality and a longer follow-up period for the ACS cohort has subsequently been published (Pope et al, 2002; Laden et al, 2006). Because of the differences in the study designs and populations considered in the ACS and Harvard Six City studies, we have elected not to pool the results of the studies. Instead, we present a primary estimate based on Pope et al. (2002) and an alternative estimate based on Laden et al. (2006).

The epidemiology-based estimates presented in this analysis reflect EPA's most current interpretation of the scientific literature on $PM_{2.5}$ and mortality, including our updated benefits methodology (i.e., a no-threshold model that calculates incremental benefits down to the lowest modeled

[9] A more complete description of the studies used and issues surrounding the estimation of premature mortality can be found in the PM NAAQS RIA, available at http://www.epa.gov/ttnecas1/ria.html.

$PM_{2.5}$ air quality levels and incorporates two technical updates) compared to the estimates in previous RIAs that did not include these changes.

As part of its efforts to improve the characterization of uncertainties in its benefits estimates, EPA has conducted a study of the C-R relationship between changes in $PM_{2.5}$ exposures and mortality using formally elicited expert judgments. The goal of the study was to elicit from a sample of health experts probabilistic distributions describing uncertainty in estimates of the reduction in mortality among the adult U.S. population resulting from reductions in ambient annual average $PM_{2.5}$ levels. These distributions were obtained through a formal interview protocol using methods designed to elicit subjective expert judgments. The full-scale study involved personal interviews with twelve peer-nominated health experts who have conducted research on the relationship between $PM_{2.5}$ exposures and mortality. The results of the full-scale study consist of twelve individual distributions for the coefficient or slope of the C-R function relating changes in annual average $PM_{2.5}$ exposures to annual, adult all-cause mortality. In prior EPA work, the results have been presented as twelve alternative estimates, and have not been combined in order to preserve the breadth and diversity of opinion on the expert panel (EPA 2006). In separate work presented in the Section 812 report on uncertainty analyses, we are exploring methods that might be applied to combine the estimates but still preserve representation of the breadth and diversity of opinion.

Infant Mortality

Recently published studies have strengthened the case for an association between PM exposure and respiratory inflammation and infection leading to premature mortality in children under 5 years of age. With regard to the cohort study conducted by Woodruff et al. (1997), the Science Advisory Board – Health Effects Subcommittee (SAB-HES) noted several strengths of the study, including the use of a larger cohort drawn from a large number of metropolitan areas and efforts to control for a variety of individual risk factors in infants (e.g., maternal educational level, maternal ethnicity, parental marital status, and maternal smoking status). Based on these findings, the SAB-HES recommended that EPA incorporate infant mortality into the primary benefits estimate and that infant mortality be evaluated using an impact function developed from the Woodruff et al. (1997) study (U.S. EPA-SAB, 2004b). A more recent study by Woodruff et al. (2006) continues to find associations between $PM_{2.5}$ and infant mortality. The study also found the most significant relationships with respiratory-related causes of death. We have not yet sought comment from the SAB on this more recent study and as such for this draft report we continue to rely on the earlier 1997 analysis.

Chronic Bronchitis

Chronic Bronchitis (CB) is characterized by mucus in the lungs and a persistent wet cough for at least 3 months a year for several years in a row. CB affects an estimated 9.1 million Americans annually (American Lung Association, 2009). A limited number of studies have estimated the impact of air pollution on new incidences of CB. Abbey et al. (1995) provide evidence that long-term $PM_{2.5}$ exposure gives rise to the development of CB in the United States.

Nonfatal Myocardial Infarctions (Heart Attacks)

Nonfatal heart attacks have been linked with short-term exposures to $PM_{2.5}$ in the United States (Peters et al., 2001) and other countries (Poloniecki et al., 1997). Other studies, such as Domenici et al. (2006), Samet et al. (2000), and Moolgavkar (2000b), show a consistent relationship between all cardiovascular hospital admissions, including those for nonfatal heart attacks, and PM. Given the lasting impact of a heart attack on long-term health costs and earnings, we provide a separate estimate for nonfatal heart attacks. The estimate used in this analysis is based on the single available U.S. $PM_{2.5}$ effect estimate from Peters et al. (2001).

Respiratory and Cardiovascular Hospital Admissions

Because of the availability of detailed hospital admission and discharge records, there is an extensive body of literature examining the relationship between hospital admissions and air pollution. Because of this, many of the hospital admission endpoints use pooled impact functions based on the results of a number of studies. The two main groups of hospital admissions estimated in this analysis are respiratory admissions and cardiovascular admissions. There is not much evidence linking PM with other types of hospital admissions.

To estimate avoided incidences of $PM_{2.5}$ related cardiovascular hospital admissions in populations aged 65 and older, we use effect estimates from studies by Moolgavkar (2003) and Ito (2003). Moolgavkar (2000a) provides the only separate effect estimate for populations 20 to 64.[10] Total cardiovascular hospital admissions are thus the sum of the pooled estimates from Moolgavkar (2003) and Ito (2003) for populations over 65 and the Moolgavkar (2000a) based impacts for populations aged 20 to 64. Cardiovascular hospital admissions include admissions for myocardial infarctions. To avoid double-counting benefits from reductions in myocardial infarctions when applying the impact function for cardiovascular hospital admissions, we first adjusted the baseline cardiovascular hospital admissions to remove admissions for myocardial infarctions.

To estimate total avoided incidences of respiratory hospital admissions, we used impact functions for several respiratory causes, including COPD, pneumonia, and asthma. Both Moolgavkar (2003) and Ito (2003) provide effect estimates for COPD in populations over 65, allowing us to pool the impact functions for this group. Only Moolgavkar (2000a) provides a separate effect estimate for populations 20 to 64. Total COPD hospital admissions are thus the sum of the pooled estimate for populations over 65 and the single study estimate for populations 20 to 64. In addition, Ito (2003) provides an effect estimate for pneumonia hospital admissions in populations 65 and older and Sheppard (2003) provides an effect estimate for asthma hospital admissions in populations under age 65. The total avoided incidence of respiratory-related hospital admissions is the sum of COPD, pneumonia, and asthma admissions.

Asthma-related Emergency Room Visits

Some studies have examined the relationship between air pollution and emergency room visits. Since most emergency room visits do not result in an admission to the hospital (the majority of people going to the emergency room are treated and return home), we treat hospital admissions and emergency room visits separately, taking account of the fraction of emergency room visits that are admitted to the hospital. The only type of emergency room visits that have been consistently linked to PM in the United States are asthma-related visits. To estimate the effects of PM air pollution reductions on asthma-related ER visits, we use the effect estimate from a study of children 18 and under by Norris et al. (1999). We selected the Norris et al. (1999) effect estimate because it focuses on $PM_{2.5}$, as opposed to PM_{10}.

Acute Health Effects

As indicated in Exhibit 2-4, in addition to mortality, chronic illness, and hospital admissions, a number of acute health effects not requiring hospitalization are associated with exposure to ambient levels of PM. The sources for the effect estimates used to quantify these effects are described below.

[10] Note that the Moolgavkar (2000) study has not been updated to reflect the more stringent GAM convergence criteria. However, given that no other estimates are available for this age group, we chose to use the existing study. Updates have been provided for the 65 and older population, and showed little difference. Given the very small (<5%) difference in the effect estimates for people 65 and older with cardiovascular hospital admissions between the original and reanalyzed results, we do not expect the difference in the effect estimates for the 20 to 64 population to differ significantly. As such, the choice to use the earlier, uncorrected analysis will likely not introduce much bias.

Around 4 percent of U.S. children between the ages of five and 17 experience episodes of acute bronchitis annually (American Lung Association, 2002c). Acute bronchitis is characterized by coughing, chest discomfort, slight fever, and extreme tiredness, lasting for a number of days. According to the MedlinePlus medical encyclopedia, symptoms usually go away without treatment.[11] Incidence of episodes of acute bronchitis in children between the ages of eight and twelve were estimated using an effect estimate developed from Dockery et al. (1996).

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

Because asthmatics have greater sensitivity to stimuli (including air pollution), children with asthma can be more susceptible to a variety of upper respiratory symptoms (e.g., runny or stuffy nose; wet cough; and burning, aching, or red eyes). Research on the effects of air pollution on upper respiratory symptoms has thus focused on effects in asthmatics. Incidences of upper respiratory symptoms in asthmatic children aged nine to eleven are estimated using an effect estimate developed from Pope et al. (1991).[12]

Following recommendations of the SAB-HES, to prevent double-counting, we focused on asthma exacerbation occurring in children and excluded adults from the calculation.[13] Asthma exacerbation occurring in adults is assumed to be captured in the general population endpoints such as work loss days and MRADs. Consequently, including an adult-specific asthma exacerbation estimate would likely double-count incidence for this endpoint. However, because the general population endpoints do not cover children (with regard to asthmatic effects), an analysis focused specifically on asthma exacerbation for children (six to eighteen years of age) could be conducted without concern for double-counting.

[11] See http://www.nlm.nih.gov/medlineplus/bronchitis.html, accessed October 2009.

[12] Pope et al. (1991) estimates the impact of PM_{10} exposure on the incidence of upper respiratory symptoms. The EPA began applying the C-R function derived from Pope et al. (1991) for PM_{10} to $PM_{2.5}$ air quality estimates in 2005 (EPA, 2005). The implicit assumptions of this action are that a) $PM_{2.5}$ is as toxic as the average of all PM_{10} and b) if a single rule or policy action reduced only precursor pollutants, the change in PM_{10} would equal the change in $PM_{2.5}$.

[13] Estimating asthma exacerbations associated with air pollution exposures is difficult, due to concerns about double-counting of benefits. Concerns over double-counting stem from the fact that studies of the general population also include asthmatics, so estimates based solely on the asthmatic population cannot be directly added to the general population numbers without double-counting. In one specific case (upper respiratory symptoms in children), the only study available is limited to asthmatic children, so this endpoint can be readily included in the calculation of total benefits. However, other endpoints, such as lower respiratory symptoms and MRADs, are estimated for the total population that includes asthmatics. Therefore, to simply add predictions of asthma-related symptoms generated for the population of asthmatics to these total population-based estimates could result in double-counting, especially if they evaluate similar endpoints. The SAB-HES, in commenting on the analytical blueprint for the current 812 study, acknowledged these challenges in evaluating asthmatic symptoms and appropriately adding them into the primary analysis (EPA-SAB, 2004b). However, despite these challenges, the SAB-HES recommended the addition of asthma-related symptoms (i.e., asthma exacerbations) to the primary analysis, provided that the studies use the panel study approach and that they have comparable design and baseline frequencies in both asthma prevalence and exacerbation rates. Note also, that the SAB-HES, while supporting the incorporation of asthma exacerbation estimates, did not believe that the association between ambient air pollution, including ozone and PM, and the new onset of asthma is sufficiently strong to support inclusion of this asthma-related endpoint in the primary estimate.

To characterize asthma exacerbations in children, we selected two studies (Ostro et al., 2001; Vedal et al., 1998) that followed panels of asthmatic children. Ostro et al. (2001) followed a group of 138 African-American children in Los Angeles for 13 weeks, recording daily occurrences of respiratory symptoms associated with asthma exacerbations (e.g., shortness of breath, wheeze, and cough). This study found a statistically significant association between $PM_{2.5}$, measured as a 12-hour average, and the daily prevalence of shortness of breath and wheeze endpoints. Although the association was not statistically significant for cough, the results were still positive and close to significance; consequently, we decided to include this endpoint, along with shortness of breath and wheeze, in generating incidence estimates (see below). Vedal et al. (1998) followed a group of elementary school children, including 74 asthmatics, located on the west coast of Vancouver Island for 18 months including measurements of daily peak expiratory flow (PEF) and the tracking of respiratory symptoms (e.g., cough, phlegm, wheeze, chest tightness) through the use of daily diaries. Because it is difficult to translate PEF measures into clearly defined health endpoints that can be monetized, we only included the cough-related effect estimate from this study in quantifying asthma exacerbations. We employed the following pooling approach in combining estimates generated using effect estimates from the two studies to produce a single asthma exacerbation incidence estimate. First, we pooled the separate incidence estimates for shortness of breath, wheeze, and cough generated using effect estimates from the Ostro et al. study, because each of these endpoints is aimed at capturing the same overall endpoint (asthma exacerbations) and there could be overlap in their predictions. The pooled estimate from the Ostro et al. study is then pooled with the cough-related estimate generated using the Vedal et al. study. The rationale for this second pooling step is similar to the first; both studies are attempting to quantify the same overall endpoint (asthma exacerbations).

Minor Restricted-Activity Days

Exposure to air pollution can result in restrictions in activity levels. These restrictions range from relatively minor changes in daily activities to serious limitations that can result in missed days of work (either from personal symptoms or from caring for a sick family member). We include two types of restricted activity days, MRADs and work loss days (WLDs). MRADs result when individuals reduce most usual daily activities and replace them with less strenuous activities or rest, yet not to the point of missing work or school. The effect of $PM_{2.5}$ on MRADs was estimated using an effect estimate derived from Ostro and Rothschild (1989).

Work Loss Days

WLDs due to $PM_{2.5}$ were estimated using an effect estimate developed from Ostro (1987). Ostro (1987) estimated the impacts of $PM_{2.5}$ on the incidence of WLDs, restricted activity days, and respiratory-related restricted activity days in a national sample of the adult working population, ages 18 to 64.

Baseline Incidence Rates

Epidemiological studies of the association between pollution levels and adverse health effects generally provide a direct estimate of the relationship of air quality changes to the *relative risk* of a health effect, rather than estimating the absolute number of avoided cases. For example, a typical result might be that a 10 ppb decrease in daily ozone levels might, in turn, decrease hospital admissions by 3 percent. The baseline incidence of the health effect is necessary to convert this relative change into a number of cases. A baseline incidence rate is the estimate of the number of cases of the health effect per year in the assessment location, as it corresponds to baseline pollutant levels in that location. To derive the total baseline incidence per year, this rate must be multiplied by the corresponding population number. For example, if the baseline incidence rate is the number of cases per year per million people, that number must be multiplied by the millions of people in the total population.

Exhibit 2-5 summarizes the sources of baseline incidence rates and provides average incidence rates for the endpoints included in the analysis. For both baseline incidence and prevalence data, we used age-specific rates where available. We applied C-R functions to individual age groups and then summed over the relevant age range to provide an estimate of total population benefits. In most cases, we used a single national incidence rate, due to a lack of more spatially disaggregated data. Whenever possible, the national rates used are national averages, because these data are most applicable to a national assessment of benefits. For some studies, however, the only available incidence information comes from the studies themselves; in these cases, incidence in the study population is assumed to represent typical incidence at the national level. Regional incidence rates are available for hospital admissions, and county-level data are available for premature mortality. We have projected mortality rates such that future mortality rates are consistent with our projections of population growth (Abt Associates, 2005).

For the set of endpoints affecting the asthmatic population, in addition to baseline incidence rates, prevalence rates of asthma in the population are needed to define the applicable population. Exhibit 2-5 lists the baseline incidence rates and their sources for asthma symptom endpoints. Exhibit 2-6 lists the prevalence rates used to determine the applicable population for asthma symptom endpoints. Note that these reflect current asthma prevalence and assume no change in prevalence rates in future years. It should be noted that current trends in asthma prevalence do not lead us to expect that asthma prevalence rates will be more than 4 percent overall in 2020, or that large changes will occur in asthma prevalence rates for individual age categories (Mansfield et al., 2005).

Exhibit 2-5. Baseline Incidence/Prevalence Rates

		Rate per 100 people per year[d] by Age Group									
Endpoint	**Notes/Source**	**<18**	**18–24**	**25–29**	**30-34**	**35-44**	**45-54**	**55-64**	**65-74**	**75-84**	**85+**
Mortality	CDC Compressed										
All-cause	Mortality File,	0.045	0.093	0.119	0.119	0.211	0.437	1.056	2.518	5.765	15.160
Non-accidental	accessed through	0.025	0.022	0.057	0.057	0.150	0.383	1.006	2.453	5.637	14.859
Cardiopulmonary	CDC Wonder (1996–1998)	0.004	0.005	0.013	0.013	0.044	0.143	0.420	1.163	3.179	9.846
Respiratory Hospital Admissions	1999 NHDS[a] public use data files[b]										
All respiratory		1.066	0.271	0.318		0.446	0.763	1.632	5.200		
Pneumonia		0.308	0.069	0.103		0.155	0.256	0.561	2.355		
Asthma		0.281	0.081	0.110		0.099	0.144	0.161	0.205		
COPD		0.291	0.089	0.124		0.148	0.301	0.711	1.573		
Cardiovascular Hospital Admissions	1999 NHDS public use data files[b]										
All cardiovascular		0.030	0.052	0.146		0.534	1.551	3.385	8.541		
Ischemic heart disease		0.004	0.008	0.031		0.231	0.902	2.021	3.708		
Dysrhythmia		0.011	0.017	0.027		0.076	0.158	0.392	1.387		
Heart failure		0.003	0.005	0.011		0.011	0.160	0.469	2.167		
Asthma ER Visits	2000 NHAMCS public use data files[c]; 1999 NHDS public use data files[b]	1.011	1.087	0.751		0.438	0.352	0.425	0.232		
Chronic Bronchitis	Prevalence 1999 NHIS (American Lung Association, 2002b, Table4)	0.0367					0.0505		0.0587		
	Incidence Abbey et al. (1993, Table 3), for ages 27+	--	--	0.378							
Nonfatal Myocardial Infarction (heart attacks)	Incidence 1999 NHDS public										
Northeast	use data files[b],	0.0000	0.2167						1.6359		
Midwest	adjusted by 0.93 for	0.0003	0.1772						1.4898		
South	probability of	0.0006	0.1620						1.1797		
West	surviving after 29 days (Rosamond et al., 1999)	0.0000	0.1391						1.1971		
Minor Restricted Activity Days	Ostro and Rothschild (1989, p. 243)	--	780						--		
	1996 NIS (Adams et al., 1999, Table 41), U.S. Bureau of Census (1997)	--	197.1	247.5			179.6		--		
School Loss Days—all-cause	National Center for Education Statistics (1996)	990.0	--	--	--	--	--	--	--	--	--

		Rate per 100 people per year[d] by Age Group									
Endpoint	**Notes/Source**	**<18**	**18–24**	**25–29**	**30-34**	**35-44**	**45-54**	**55-64**	**65-74**	**75-84**	**85+**
Acute Bronchitis	Incidence American Lung Association (2002c, Table 11)	4.3	--	--	--	--	--	--	--	--	--
Lower Respiratory Symptoms	Incidence Schwartz et al. (1994, Table 2)	43.8	--	--	--	--	--	--	--	--	--
Upper Respiratory Symptoms	Incidence among asthmatics Pope et al. (1991, Table 2)	12479	--	--	--	--	--	--	--	--	--
Asthma Exacerbation Shortness of breath Wheeze Cough	Incidence (and prevalence) among asthmatic African Americans Ostro et al. (2001)	1350 (0.074) 2774 (0.173) 2445 (0.145)	--	--	--	--	--	--	--	--	--
Asthma Exacerbation Cough	Incidence among asthmatics Vedal et al. (1998)	3139	--	--	--	--	--	--	--	--	--

a The following abbreviations are used to describe the national surveys conducted by the National Center for Health Statistics: HIS refers to the National Health Interview Survey; NHDS—National Hospital Discharge Survey; NHAMCS—National Hospital Ambulatory Medical Care Survey.
b See ftp://ftp.cdc.gov/pub/Health_Statistics/NCHS/Datasets/NHDS/
c See ftp://ftp.cdc.gov/pub/Health_Statistics/NCHS/Datasets/NHAMCS/
d All of the rates reported here are population-weighted. Incidence rates are reported per 100 people per year; prevalence rates are reported as a percentage of the population.
Additional details on the incidence and prevalence rates, as well as the sources for these rates are available upon request.

Exhibit 2-6. Asthma Prevalence Rates Used to Estimate Asthmatic Populations in Health Impact Functions

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

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

Economic Value for Health Outcomes

Reductions in ambient concentrations of air pollution generally lower the risk of future adverse health effects for a large population. Therefore, the appropriate economic measure is willingness-to- pay (WTP) for changes in risk of a health effect rather than WTP for a health effect that would occur with certainty (Freeman, 1993). Epidemiological studies generally provide estimates of the relative risks of a particular health effect that is avoided because of a reduction in air pollution. We converted those to units of avoided statistical incidence for ease of presentation. We calculated the value of avoided statistical incidences by dividing individual WTP for a risk reduction by the related observed change in risk. For example, suppose a pollution-reduction regulation is able to reduce the risk of premature mortality from 2 in 10,000 to 1 in 10,000 (a reduction of 1 in 10,000). If individual WTP for this risk reduction is $100, then the WTP for an avoided statistical premature death is $1 million ($100/0.0001 change in risk).

WTP estimates generally are not available for some health effects, such as hospital admissions. In these cases, we used the cost of treating or mitigating the effect as a primary estimate. These cost-of-illness (COI) estimates generally understate the true value of reducing the risk of a health effect, because they reflect the direct expenditures related to treatment, but not the value of avoided pain and suffering (Harrington and Portney, 1987; Berger, 1987). We provide unit values for health endpoints (along with information on the distribution of the unit value) in Exhibit 2-6. All values are in constant year 2006 dollars, adjusted for growth in real income out to each of the three target years (2000, 2010, and 2020) using the income growth projections contained in BenMAP.[14] Economic theory argues that WTP for most goods, including environmental protection will increase if real income increases. Many of the valuation studies used in this analysis were conducted in the late 1980s and early 1990s. Because real income has grown since the studies were conducted, people's willingness to pay for reductions in the risk of premature death and disease likely has grown as well. We did not adjust cost of illness-based values because they are based on current costs, as parameterized in the BenMAP system. Similarly, we did not adjust the value of school absences, because that value is based on current wage rates. Exhibit 2-7 presents the values for individual endpoints adjusted to year 2020 income levels to illustrate the impact of the adjustment for income growth over time. The discussion below provides additional details on valuation of specific ozone and PM related endpoints.

[14] Projections of income growth in BenMAP are based on data from Standard and Poor's.

Exhibit 2-7. Unit Values for Economic Valuation of Health Endpoints (2006$)

Health Endpoint	Central Estimate of Value Per Statistical Incidence: 1990 Income Level	Central Estimate of Value Per Statistical Incidence: 2020 Income Level	Derivation of Distributions of Estimates
Premature Mortality (Value of a Statistical Life)	$7,400,000	$8,880,000	Mean Value of Statistical Life (VSL) based on the mean of a distribution fitted to 26 "value of statistical life" (VSL) estimates that appear in the economics literature and that have been identified in the Section 812 Reports to Congress as "applicable to policy analysis." The VSL approach and the set of selected studies mirrors that of Viscusi (1992) (with the addition of two studies), and uses the same criteria as Viscusi in his review of value-of-life studies. The central estimate of $6.3 million (2000$) is consistent with Viscusi's conclusion (updated to 2000$) that "most of the reasonable estimates of the value of life are clustered in the $3.8 to $8.9 million range." Five of the 26 studies are contingent valuation (CV) studies, which directly solicit WTP information from subjects; the rest are wage-risk studies, which base WTP estimates on estimates of the additional compensation demanded in the labor market for riskier jobs. The fitted distribution is a Weibull with $\alpha=5.32\text{x}10^{-6}$ and $\beta=1.509588$. See Abt Associates, 2008 for more details.
Chronic Bronchitis (CB)	$399,000	$490,000	The WTP to avoid a case of pollution-related CB is calculated as $WTP_x = WTP_{13} \cdot e^{-\beta \cdot (13-x)}$, where *x* is the severity of an average CB case, WTP_{13} is the WTP for a severe case of CB, and β is the parameter relating WTP to severity, based on the regression results reported in Krupnick and Cropper (1992). The distribution of WTP for an average severity-level case of 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, with the most likely value at severity level 6.5 and endpoints at 1.0 and 12.0; 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)). This process and the rationale for choosing it is described in detail in the Costs and Benefits of the Clean Air Act, 1990 to 2010 (EPA, 1999).
Nonfatal Myocardial Infarction (heart attack) 7% discount rate Age 0–24 Age 25–44 Age 45–54 Age 55–65 Age 66 and over	 $84,171 $93,802 $98,366 $166,222 $84,171		No distributional information available. Age-specific cost-of-illness values reflect lost earnings and direct medical costs over a 5-year period following a nonfatal MI. Lost earnings estimates are based on Cropper and Krupnick (1990). Direct medical costs are based on simple average of estimates from Russell et al. (1998) and Wittels et al. (1990). Lost earnings: Cropper and Krupnick (1990). Present discounted value of 5 years of lost earnings (2006$): age of onset: at 7%[a] 25–44 $9,631 45–54 $14,195 55–65 $82,051 Direct medical expenses: An average of (2006$): 1. Wittels et al. (1990) ($141,124—no discounting) 2. Russell et al. (1998), 5-year period ($28,787 at 3% discount rate; $21,113 $27,217 at 7% discount rate)

Health Endpoint	Central Estimate of Value Per Statistical Incidence		Derivation of Distributions of Estimates
	1990 Income Level	2020 Income Level	
Hospital Admissions			
All respiratory (ages 65+)	$23,711	$23,711	No distributions available. The COI point estimates (lost earnings plus direct medical costs) are based on ICD-9 code level information (e.g., average hospital care costs and average length of hospital stay) reported in Agency for Healthcare Research and Quality, 2000 (www.ahrq.gov). As noted in the text, no adjustments are made to cost of illness values for income growth.
All respiratory (ages 0–2)	$10,002	$10,002	
Chronic Obstructive Pulmonary Disease (COPD) (ages 65+)	$17,308	$17,308	
Asthma Admissions (ages <65)	$10,040	$10,040	
Pneumonia Admissions (ages 65+)	$23,004	$23,004	
COPD, less asthma (ages 20–64)	$15,903	$15,903	
All Cardiovascular (ages 65+)	$27,319	$27,319	
All Cardiovascular (ages 20–64)	$29,364	$29,364	
Ischemic Heart Disease (ages 65+)	$33,357	$33,357	
Dysrhythmia (ages 65+)	$19,643	$19,643	
Congestive Heart Failure (ages 65+)	$19,619	$19,619	
Emergency Room Visits for Asthma	$369	$369	No distributional information available. Simple average of two unit COI values (2006$): (1) $401.62, from Smith et al. (1997) and (2) $336.03, from Stanford et al. (1999). As noted in the text, no adjustments are made to cost of illness values for income growth.
Respiratory Ailments Not Requiring Hospitalization			
Upper Respiratory Symptoms (URS)	$28.8	$30.7	Combinations of the three symptoms for which WTP estimates are available that closely match those listed by Pope et al. result in seven different "symptom clusters," each describing a "type" of URS. A dollar value was derived for each type of URS, using mid-range estimates of WTP (IEc, 1994) to avoid each symptom in the cluster and assuming additivity of WTPs. In the absence of information surrounding the frequency with which each of the seven types of URS occurs within the URS symptom complex, we assumed a uniform distribution between $10.8 and $50.5 (2006$).

Health Endpoint	Central Estimate of Value Per Statistical Incidence: 1990 Income Level	Central Estimate of Value Per Statistical Incidence: 2020 Income Level	Derivation of Distributions of Estimates
Lower Respiratory Symptoms (LRS)	$18	$19	Combinations of the four symptoms for which WTP estimates are available that closely match those listed by Schwartz et al. result in 11 different "symptom clusters," each describing a "type" of LRS. A dollar value was derived for each type of LRS, using mid-range estimates of WTP (IEc, 1994) to avoid each symptom in the cluster and assuming additivity of WTPs. The dollar value for LRS is the average of the dollar values for the 11 different types of LRS. In the absence of information surrounding the frequency with which each of the 11 types of LRS occurs within the LRS symptom complex, we assumed a uniform distribution between $8.1 and $28.6 (2006$).
Asthma Exacerbations	$50	$54	Asthma exacerbations are valued at $45 per incidence, based on the mean of average WTP estimates for the four severity definitions of a "bad asthma day," described in Rowe and Chestnut (1986). This study surveyed asthmatics to estimate WTP for avoidance of a "bad asthma day," as defined by the subjects. For purposes of valuation, an asthma exacerbation is assumed to be equivalent to a day in which asthma is moderate or worse as reported in the Rowe and Chestnut (1986) study. The value is assumed have a uniform distribution between $18.3 and $82.9 (2006$).
Acute Bronchitis	$416	$512	Assumes a 6-day episode, with the distribution of the daily value specified as uniform with the low and high values based on those recommended for related respiratory symptoms in Neumann et al. (1994). The low daily estimate of $20.5 (2006$) is the sum of the mid-range values recommended by IEc (1994) for two symptoms believed to be associated with acute bronchitis: coughing and chest tightness. The high daily estimate was taken to be twice the value of a minor respiratory restricted activity day, or $118 (2006$). The low and high daily values are multiplied by six to get the 6-day episode values.
Work Loss Days (WLDs)	Variable (U.S. median = $149)		No distribution available. Point estimate is based on county-specific median annual wages divided by 50 (assuming 2 weeks of vacation) and then by 5—to get median daily wage. U.S. Year 2000 Census, compiled by Geolytics, Inc.
Minor Restricted Activity Days (MRADs)	$61	$64	Median WTP estimate to avoid one MRAD from Tolley et al. (1986). Distribution is assumed to be triangular with a minimum of $24 and a maximum of $94, with a most likely value of $59 (2006$). 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 $24) 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.
School Loss Days	$89	$89	No distribution available. Point estimate is based on (1) the probability that, if a school child stays home from school, a parent will have to stay home from work to care for the child, and (2) the value of the parent's lost productivity. Calculated using U.S. Bureau of Census data.

[a] These values are presented using a seven percent discount rate for this draft report, however these results will be presented using a five percent discount rate in the final report.

Mortality Valuation

To estimate the monetary benefit of reducing the risk of premature death, we used the "value of statistical lives" saved (VSL) approach, which is a summary measure for the value of small changes in mortality risk for a large number of people. The VSL approach applies information from several published value-of-life studies to determine a reasonable monetary value of preventing premature mortality. The mean value of avoiding one statistical death is estimated to be approximately $7.4 million at 1990 income levels (2006$), and $8.8 million (2006$) at 2020 income levels. This value is the mean of a distribution fitted to 26 VSL estimates that appear in the economics literature and that have been identified in the Section 812 Reports to Congress as "applicable to policy analysis." This represents an intermediate value from a variety of estimates, and it is a value EPA has frequently used in RIAs as well as in the Section 812 Retrospective and Prospective Analyses of the Clean Air Act.

The VSL approach and the set of selected studies mirrors that of Viscusi (1992) (with the addition of two studies), and uses the same criteria as Viscusi in his review of value-of-life studies. The $7.4 million estimate is consistent with Viscusi's conclusion (updated to 2006$) that "most of the reasonable estimates of the value of life are clustered in the $4.4 to $10.4 million range." 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. Because this VSL-based unit value does not distinguish among people based on the age at their death or the quality of their lives, it can be applied to all premature deaths.

Chronic Bronchitis

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

We use the mean of a distribution of WTP estimates as the central tendency estimate of WTP to avoid a pollution-related case of CB in this analysis. The distribution incorporates uncertainty from three sources: the WTP to avoid a case of severe CB, as described by Viscusi et al.; the severity level of an average pollution-related case of CB (relative to that of the case described by Viscusi et al.); and the elasticity of WTP with respect to severity of the illness. Based on assumptions about the distributions of each of these three uncertain components, we derive a distribution of WTP to avoid a pollution-related case of CB by statistical uncertainty analysis techniques. The expected value (i.e., mean) of this distribution, which is about $399,000 (2006$), is taken as the central tendency estimate of WTP to avoid a PM-related case of CB.

Nonfatal Myocardial Infarction Valuation

We were not able to identify a suitable WTP value for reductions in the risk of nonfatal heart attacks. Instead, we use a COI unit value with two components: the direct medical costs and the opportunity cost (lost earnings) associated with the illness event. Because the costs associated with a myocardial infarction extend beyond the initial event itself, we consider costs incurred over five years. We used age-specific annual lost earnings estimated by Cropper and Krupnick (1990). Cropper and

Krupnick (1990) do not provide lost earnings estimates for populations under 25 or over 65. As such, we do not include lost earnings in the cost estimates for these age groups.

Three sources were consulted for direct medical costs of myocardial infarction: Wittels et al. (1990), Eisenstein et al. (2001), and Russell et al. (1998). Because the wage-related opportunity cost estimates from Cropper and Krupnick (1990) cover a 5-year period, we used estimates for medical costs that similarly cover a 5-year period (i.e., estimates from Wittels et al. (1990) and Russell et al. (1998)). We used a simple average of the two 5-year estimates.[15]

Hospital Admissions Valuation

In the absence of estimates of societal WTP to avoid hospital visits/admissions for specific illnesses, estimates of total cost of illness (total medical costs plus the value of lost productivity) typically are used as conservative, or lower bound, estimates. These estimates are biased downward, because they do not include the willingness-to-pay value of avoiding pain and suffering.

The International Classification of Diseases (ICD-9, 1979) code-specific COI estimates used in this analysis consist of estimated hospital charges and the estimated opportunity cost of time spent in the hospital (based on the average length of a hospital stay for the illness). We based all estimates of hospital charges and length of stays on statistics provided by the Agency for Healthcare Research and Quality (AHRQ, 2000). We estimated the opportunity cost of a day spent in the hospital as the value of the lost daily wage, regardless of whether the hospitalized individual is in the workforce. To estimate the lost daily wage, we divided year 2000 median annual wage by (52*5) to get median daily wage and inflated the result to year 2006$ using the EPA standard inflator wage index. The resulting estimate is $149. The total cost-of-illness estimate for an ICD code-specific hospital stay lasting *n* days, then, was the mean hospital charge plus $109 multiplied by *n.*

Asthma-Related Emergency Room Visits Valuation

To value asthma emergency room visits, we used a simple average of two estimates from the health economics literature. The first estimate comes from Smith et al. (1997), who reported approximately 1.2 million asthma-related emergency room visits in 1987, at a total cost of $186.5 million (1987$). The average cost per visit that year was $155; in 2006$, that cost was $401.62 (using the EPA standard inflator medical cost index). The second estimate comes from Stanford et al. (1999), who reported the cost of an average asthma-related emergency room visit at $336.03 (adjusted to 2006$), based on 1996–1997 data. A simple average of the two estimates yields a (rounded) unit value of $369.

Minor Restricted Activity Days Valuation

No studies are reported to have estimated WTP to avoid a minor restricted activity day. However, one of EPA's contractors, IEc (1993) has derived an estimate of willingness to pay to avoid a minor *respiratory* restricted activity day, using estimates from Tolley et al. (1986) of WTP for avoiding a combination of coughing, throat congestion and sinusitis. The IEc estimate of WTP to avoid a minor respiratory restricted activity day is about $61 ($2006).

[15] In this draft analysis a seven percent discount rate is used to discount costs incurred over the 5-year period. The Project Team is currently working on incorporating values based on a five percent discount rate into BenMAP for use in the final analysis.

Although Ostro and Rothschild (1989) statistically linked ozone and minor restricted activity days, it is likely that most MRADs associated with ozone exposure are, in fact, minor *respiratory* restricted activity days. For the purpose of valuing this health endpoint, we used the estimate of mean WTP to avoid a minor respiratory restricted activity day.

School Loss Days

To value a school absence, we: (1) estimated the probability that if a school child stays home from school, a parent will have to stay home from work to care for the child; and (2) valued the lost productivity at the parent's wage. To do this, we estimated the number of families with school-age children in which both parents work, and we valued a school-loss day as the probability that such a day also would result in a work-loss day. We calculated this value by multiplying the proportion of households with school-age children by a measure of lost wages.

We used this method in the absence of a preferable WTP method. However, this approach suffers from several uncertainties. First, it omits willingness to pay to avoid the symptoms/illness that resulted in the school absence; second, it effectively gives zero value to school absences that do not result in work-loss days; and third, it uses conservative assumptions about the wages of the parent staying home with the child. Finally, this method assumes that parents are unable to work from home. If this is not a valid assumption, then there would be no lost wages.

For this valuation approach, we assumed that in a household with two working parents, the female parent will stay home with a sick child. From the Statistical Abstract of the United States (U.S. Census Bureau, 2001), we obtained: (1) the numbers of single, married and "other" (widowed, divorced or separated) working women with children; and (2) the rates of participation in the workforce of single, married and "other" women with children. From these two sets of statistics, we calculated a weighted average participation rate of 72.85 percent.

Our estimate of daily lost wage (wages lost if a mother must stay at home with a sick child) is based on the year 2000 median weekly wage among women ages 25 and older (U.S. Census Bureau, 2001). This median weekly wage is $551 (2000$). Dividing by five gives an estimated median daily wage of $103. To estimate the expected lost wages on a day when a mother has to stay home with a school-age child, we first estimated the probability that the mother is in the workforce then multiplied that estimate by the daily wage she would lose by missing a workday: 72.85 percent times $103, for a total loss of $75 in 2000$, or $89 in 2006$. This valuation approach is similar to that used by Hall et al. (2003).

Results and Implications

Ozone Benefit Estimates

Ozone benefit estimates are calculated for a "stitched" National domain, created by merging results from the two original modeling domains, Eastern United States (EUS) and Western United States (WUS), and eliminating double-counting in the areas of overlap (see Exhibit 2-1). Exhibit 2-8 summarizes the valuation of ozone benefits for the nation. Exhibits 2-9 through 2-11 give detailed ozone benefit estimates in each target year for the nation. In addition to the mean incidence and valuation estimates, we have included 5^{th} and 95^{th} percentile estimates when available.

Based in part on prior SAB advice, EPA has typically assumed that there is a time lag between changes in pollution exposures and the total realization of changes in health effects. Within the context of

benefits analyses, this term is often referred to as "cessation lag". The existence of such a lag is important for the valuation of premature mortality incidence because economic theory suggests that benefits occurring in the future should be discounted. In this analysis, we apply a twenty-year distributed lag to PM mortality reductions - this method is consistent with the most recent recommendation by the EPA's Science Advisory Board (EPA – SAB, 2004a) – but not to premature mortality reduction attributed to reduced ozone exposure. Alternative cessation lag structures for PM-related mortality risk are explored in the accompanying Second Prospective uncertainty analysis report. For the primary results, a five percent discount rate is used to discount future benefits back to the target year of the analysis (i.e., 2000, 2010, or 2020).

Benefits of reduced morbidity account for roughly eight percent of the total primary ozone benefits in the EUS and five percent in the WUS. Exhibit 2-12 presents a more detailed comparison of the primary ozone morbidity estimates. Benefits of reduced mortality make up the remainder of the total ozone benefits.

Exhibit 2-8. EUS Summary Ozone Valuation Results

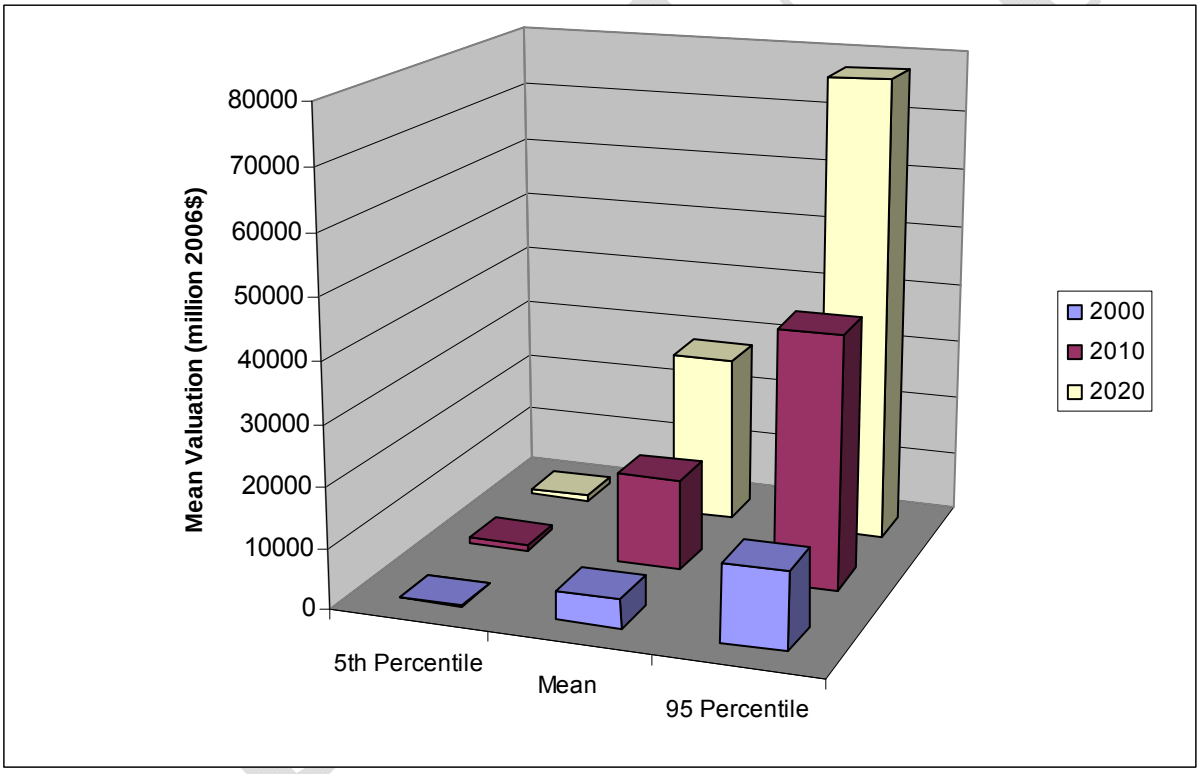


Exhibit 2-9. National Ozone Benefits of CAAA in 2000

Endpoint Group	Incidence			Valuation (million 2006$)		
	Percentile 5	Mean	Percentile 95	Percentile 5	Mean	Percentile 95
Mortality – Pooling of Bell et al. (2004) and Schwartz (2005)	160	560	1,100	$60	$4,300	$12,000
Hospital Admissions, Respiratory (>64)	100	3,000	5,700	$2.5	$70	$140
Hospital Admissions, Respiratory (<2)	1,400	3,000	4,600	$14	$30	$46
Emergency Room Visits, Respiratory	0	2,200	6,200	$0	$0.81	$2.2
Minor Restricted Activity Days	1,300,000	3,100,000	4,800,000	$70	$180	$330
School Loss Days	480,000	1,200,000	1,900,000	$43	$110	$170
Outdoor Worker Productivity				$30	$30	$30

Notes:
1. Results are rounded to two significant figures.
2. Mortality results from Bell et al. (2004) and Schwartz (2005) are pooled using inverse variance weights.

Exhibit 2-10. National Ozone Benefits of CAAA in 2010

Endpoint Group	Incidence			Valuation (million 2006$)		
	Percentile 5	Mean	Percentile 95	Percentile 5	Mean	Percentile 95
Mortality – Pooling of Bell et al. (2004) and Schwartz (2005)	570	1,800	3,400	$460	$14,000	$40,000
Hospital Admissions, Respiratory (>64)	740	9,900	18,000	$17	$230	$440
Hospital Admissions, Respiratory (<2)	4,300	9,000	14,000	$43	$90	$140
Emergency Room Visits, Respiratory	0	6,600	18,000	$0	$2.4	$6.4
Minor Restricted Activity Days	4,400,000	9,500,000	15,000,000	$230	$560	$1,000
School Loss Days	1,400,000	3,200,000	5,100,000	$120	$290	$450
Outdoor Worker Productivity				$100	$100	$100

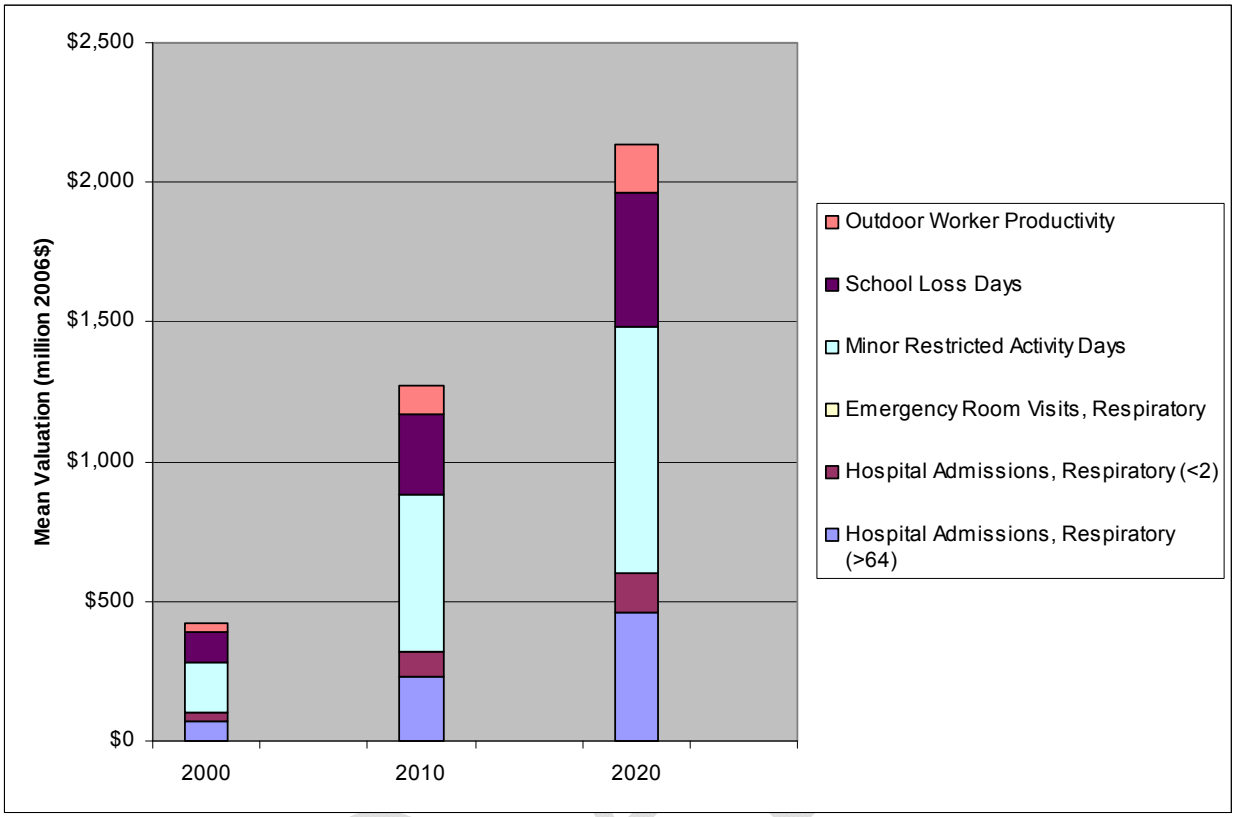
Notes:
1. Results are rounded to two significant figures.
2. Mortality results from Bell et al. (2004) and Schwartz (2005) are pooled using inverse variance weights.

Exhibit 2-11. National Ozone Benefits of CAAA in 2020

Endpoint Group	Incidence			Valuation (million 2006$)		
	Percentile 5	Mean	Percentile 95	Percentile 5	Mean	Percentile 95
Mortality – Pooling of Bell et al. (2004) and Schwartz (2005)	900	3,000	5,700	$67	$26,000	$74,000
Hospital Admissions, Respiratory (>64)	990	19,000	36,000	$23	$460	$860
Hospital Admissions, Respiratory (<2)	6,600	14,000	22,000	$65	$140	$220
Emergency Room Visits, Respiratory	0	11,000	31,000	$0	$4.1	$11
Minor Restricted Activity Days	6,400,000	15,000,000	23,000,000	$330	$880	$1,600
School Loss Days	2,200,000	5,400,000	8,600,000	$190	$480	$770
Outdoor Worker Productivity				$170	$170	$170

Notes:
1. Results are rounded to two significant figures.
1. Mortality results from Bell et al. (2004) and Schwartz (2005) are pooled using inverse variance weights.

Exhibit 2-12. National Ozone Morbidity Benefits



PM Benefit Estimates

PM benefit estimates are calculated at the national level for the contiguous 48 states. Exhibit 2-13 summarizes the valuation of PM benefits for the two alternative mortality estimates. Exhibits 2-14 through 2-16 give detailed PM benefit estimates in each target year. In addition to the mean incidence and valuation estimates, we have included 5^{th} and 95^{th} percentile estimates when available.

Benefits of reduced morbidity account for between three and six percent of the total PM benefits, depending on the mortality incidence estimate used. Exhibit 2-17 presents a more detailed comparison of the PM morbidity estimates. Benefits of reduced mortality make up the remainder of the total PM benefits.

Exhibit 2-13. Summary PM Valuation Results

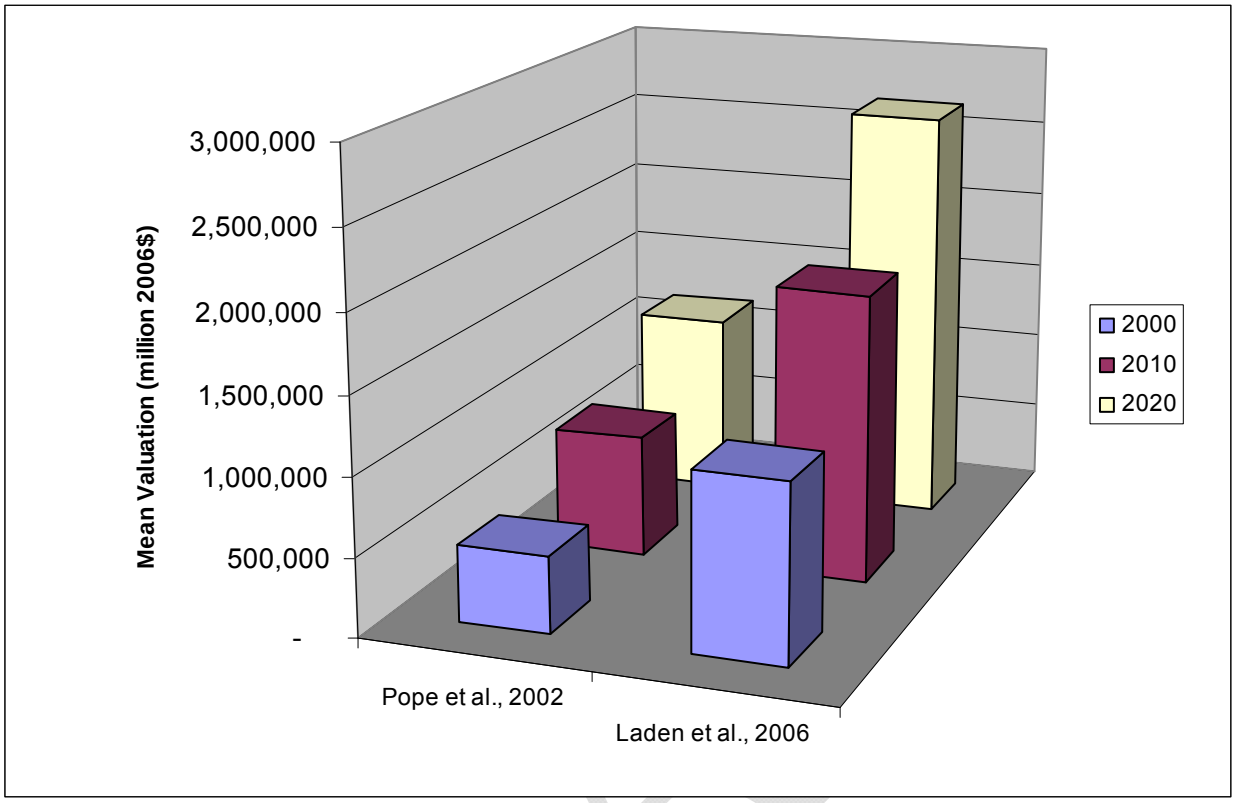


Exhibit 2-14. National PM Benefits of CAAA in 2000

Endpoint Group	Incidence			Valuation (million 2006$)		
	Percentile 5	Mean	Percentile 95	Percentile 5	Mean	Percentile 95
Mortality - Pope et al., 2002	27,000	68,000	110,000	$64,000	$460,000	$1,200,000
Mortality - Laden et al., 2006	94,000	170,000	240,000	$170,000	$1,100,000	$2,800,000
Infant Mortality - Woodruff et al., 1997	95	190	290	$740	$1,500	$2,400
Chronic Bronchitis	6,400	39,000	70,000	$1,300	$16,000	$58,000
Nonfatal Myocardial Infarction	35,000	89,000	140,000	$2,600	$9,100	$22,000
Hospital Admissions, Respiratory	7,800	16,000	23,000	$110	$220	$320
Hospital Admissions, Cardiovascular	23,000	30,000	37,000	$620	$860	$1,100
Emergency Room Visits, Respiratory	38,000	63,000	86,000	$13.0	$23.0	$35.0
Acute Bronchitis	0	110,000	210,000	$0	$48.0	$120
Lower Respiratory Symptoms	700,000	1,400,000	2,000,000	$10.0	$25.0	$46.0
Upper Respiratory Symptoms	360,000	1,100,000	1,900,000	$9.80	$35.0	$74.0
Asthma Exacerbation	150,000	1,400,000	3,900,000	$8.20	$70.0	$210
Minor Restricted Activity Days	45,000,000	54,000,000	62,000,000	$1,900	$3,200	$4,600
Work Loss Days	8,200,000	9,300,000	11,000,000	$1,200	$1,400	$1,600

Notes:
1. Results are rounded to two significant figures.

Exhibit 2-15. National PM Benefits of CAAA in 2010

Endpoint Group	Incidence			Valuation (million 2006$)		
	Percentile 5	Mean	Percentile 95	Percentile 5	Mean	Percentile 95
Mortality - Pope et al., 2002	41,000	100,000	160,000	$100,000	$730,000	$1,900,000
Mortality - Laden et al., 2006	140,000	250,000	360,000	$270,000	$1,800,000	$4,500,000
Infant Mortality - Woodruff et al., 1997	130	260	390	$1,100	$2,200	$3,400
Chronic Bronchitis	9,700	59,000	110,000	$2,200	$26,000	$93,000
Nonfatal Myocardial Infarction	58,000	140,000	220,000	$4,500	$15,000	$36,000
Hospital Admissions, Respiratory	12,000	24,000	37,000	$170	$340	$500
Hospital Admissions, Cardiovascular	37,000	49,000	59,000	$1,000	$1,400	$1,800
Emergency Room Visits, Respiratory	54,000	87,000	120,000	$18.0	$32.0	$48.0
Acute Bronchitis	0	150,000	270,000	$0	$68.0	$160
Lower Respiratory Symptoms	940,000	1,800,000	2,700,000	$14.0	$34.0	$61.0
Upper Respiratory Symptoms	490,000	1,600,000	2,600,000	$13.00	$48.0	$100.0
Asthma Exacerbation	210,000	1,900,000	5,500,000	$12.00	$100.0	$310
Minor Restricted Activity Days	70,000,000	82,000,000	94,000,000	$2,900	$4,900	$7,000
Work Loss Days	12,000,000	14,000,000	16,000,000	$1,900	$2,100	$2,400

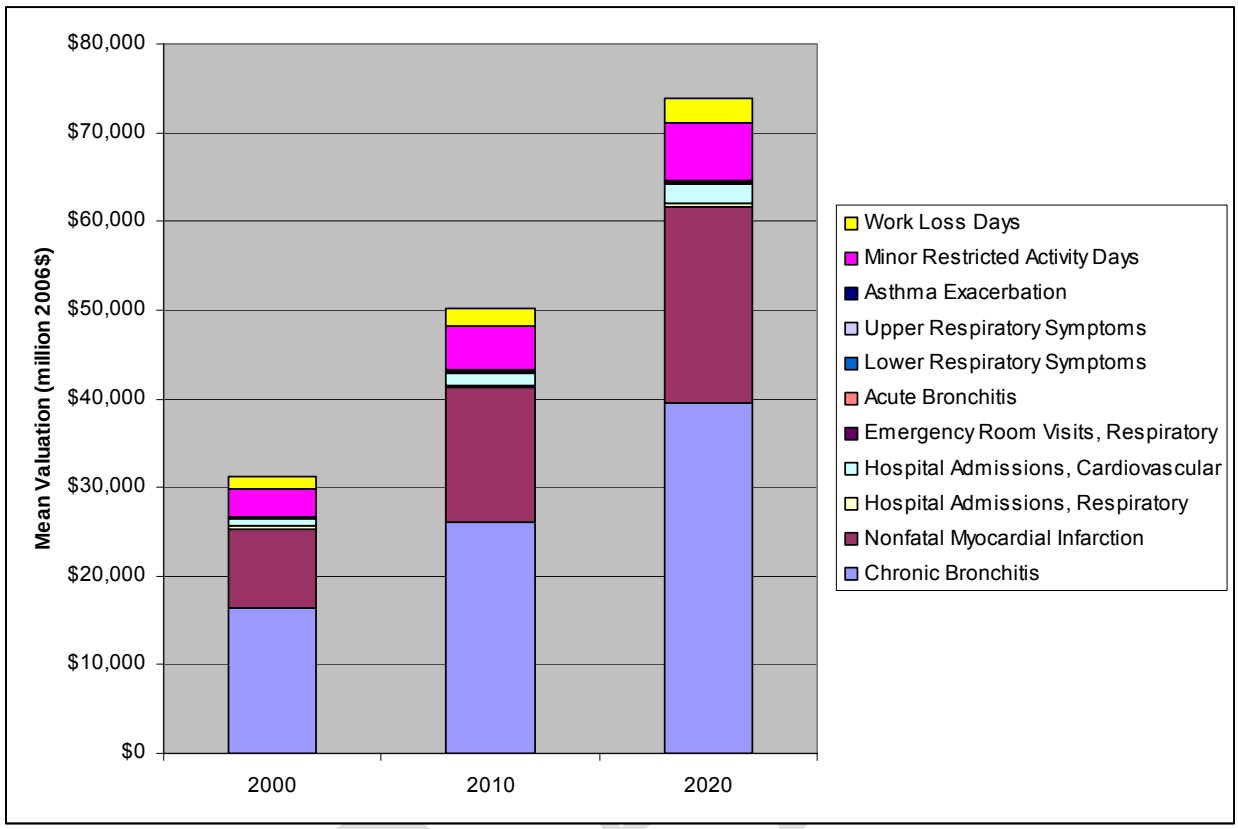
Notes:
1. Results are rounded to two significant figures.

Exhibit 2-16. National PM Benefits of CAAA in 2020

Endpoint Group	Incidence			Valuation (million 2006$)		
	Percentile 5	Mean	Percentile 95	Percentile 5	Mean	Percentile 95
Mortality - Pope et al., 2002	56,000	140,000	220,000	$150,000	$1,100,000	$2,800,000
Mortality - Laden et al., 2006	190,000	340,000	480,000	$400,000	$2,600,000	$6,500,000
Infant Mortality - Woodruff et al., 1997	150	310	460	$1,400	$2,800	$4,400
Chronic Bronchitis	14,000	81,000	140,000	$3,300	$39,000	$140,000
Nonfatal Myocardial Infarction	86,000	210,000	320,000	$6,600	$22,000	$52,000
Hospital Admissions, Respiratory	18,000	35,000	52,000	$250	$500	$730
Hospital Admissions, Cardiovascular	57,000	75,000	90,000	$1,600	$2,100	$2,800
Emergency Room Visits, Respiratory	71,000	110,000	160,000	$24.0	$42.0	$62.0
Acute Bronchitis	0	200,000	360,000	$0	$100.0	$240
Lower Respiratory Symptoms	1,300,000	2,500,000	3,600,000	$19.0	$46.0	$83.0
Upper Respiratory Symptoms	680,000	2,200,000	3,600,000	$19.00	$66.0	$140.0
Asthma Exacerbation	290,000	2,600,000	7,400,000	$16.00	$140.0	$420
Minor Restricted Activity Days	92,000,000	110,000,000	120,000,000	$3,800	$6,400	$9,300
Work Loss Days	16,000,000	19,000,000	21,000,000	$2,500	$2,800	$3,200

Notes:
1. Results are rounded to two significant figures.

Exhibit 2-17. National PM Morbidity Benefits



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