TECHNICAL ADDENDUM: METHODOLOGIES FOR THE BENEFIT ANALYSIS OF THE CLEAR SKIES ACT OF 2003

September 2003

1. INTRODUCTION

1.1. Background

On February 14, 2002, President Bush announced the Clear Skies Initiative, a proposal to reduce emissions from electric power generating sources. The proposal was embodied in legislative form as the Clear Skies Act of 2002, which was introduced in the House of Representatives and in the Senate in July 2002. The Clear Skies Act was reintroduced in the US House of Representatives (H.R. 999) and the US Senate (S. 485) as the Clear Skies Act of 2003 on February 27, 2003.

If enacted, the Clear Skies Act of 2003 would reduce emissions of sulfur dioxide (SO_2), nitrogen oxides (NO_x), and mercury from fossil fuel-fired combustion units by approximately 70% from current levels at full implementation.[1] These mandatory emission reductions would be achieved through a cap and trade program, modeled on the current Acid Rain Program for SO_2. Federally enforceable emissions limits, or national caps, for each pollutant would be established. Sources would be allowed to transfer these authorized emission limits among themselves to achieve the required reductions for all three pollutants at the lowest overall cost. This proposal would alleviate many of the remaining environmental and health problems associated with power generation.

This document reports the methods and results of an analysis of the environmental and health benefits of the Clear Skies Act of 2003.[2] It presents quantitative estimates of the health improvements and monetary benefits that would be achieved by this proposal.

1.2. Summary of the Benefits Analysis Methods and Results

The Clear Skies Act would provide significant benefits to public health and the environment. Emissions reductions would start before 2010 and would increase significantly between 2010 and 2020. Based on these emissions reductions, the cumulative health benefits of the program across the next two decades would be significant. The key results of this analysis of the Clear Skies Act are summarized in Tables 1a and 1b.

As shown in Tables 1a and 1b, we have used two approaches to provide health and environmental benefits (Base Estimate and Alternative Estimate). While there is a substantial difference in the specific estimates, both approaches show that the monetary benefits of the Clear

[1] The Clear Skies Act would cut sulfur dioxide (SO2) emissions by 73 percent, from year 2000 emissions of 11 million tons to caps of 4.5 million tons in 2010 and 3 million tons in 2018. It would cut emissions of nitrogen oxides (NOx) by 67 percent, from year 2000 emissions of 5 million tons to caps of 2.1 million tons in 2008 and 1.7 million tons in 2018. Mercury emissions would be reduced by 69 percent, from year 1999 emissions of 48 tons to caps of 26 tons in 2010 and 15 tons in 2018. Because sources can reduce emissions early, earn allowances for these actions, and use the allowances later, emissions are projected to be higher than the cap in the first years under each cap.

[2] This document is an update of an earlier technical addendum, which described the methods and results of an EPA's analysis of the Clear Skies Act of 2002.

Skies Act are well in excess of the estimated costs of $4.3 billion (1999 $) in 2010 and $6.3 billion in 2020.[3]

Table 1a

Reductions in Incidence of PM and Ozone-related Adverse Health Effects Associated with the Clear Skies Act

Health Effect	Avoided Incidence[A] (cases/year)	
	2010	2020
Premature mortality		
Base estimate: Long-term exposure	7,900	14,000
Alternative estimate: Short-term exposure	4,700	8,400
Chronic bronchitis	5,400	8,800
Non-fatal myocardial infarctions	13,000	23,000
Hospital admissions – Respiratory [C]	5,200	11,000
Hospital admissions – Cardiovascular [D]	3,200	5,800
Emergency Room Visits for Asthma	8,300	14,000
Acute bronchitis	13,000	20,000
Lower respiratory symptoms	140,000	230,000
Upper respiratory symptoms	110,000	180,000
Work loss days	1,100,000	1,600,000
Minor restricted activity days	6,600,000	10,000,000
School absence days	81,000	200,000

[A] Incidences are rounded to two significant digits.
[B] Premature mortality associated with ozone is not separately included in this analysis
[C] Respiratory hospital admissions for PM includes admissions for COPD, pneumonia, and asthma.
[D] Cardiovascular hospital admissions for PM includes total cardiovascular and subcategories for ischemic heart disease, dysrhythmias, and heart failure.

[3] Detailed information on the costs of Clear Skies can be found in the Clear Skies Act: Technical Support Package (2003) available at www.epa.gov/clearskies.

Table 1b

Results of Human Health and Welfare Benefits Valuation for the Clear Skies Analyses, 2010 and 2020

Endpoint	Monetary Benefits [a] (millions 1999$)	
	2010 Mean	2020 Mean
Health Endpoints		
Premature mortality [b]		
Base estimate: Long-term exposure, (adults, 30 and over)		
3% discount rate	$50,000	$100,000
7% discount rate	$47,000	$97,000
Alternative estimate: Short-term exposure (all ages)		
3% discount rate	$7,900	$16,000
7% discount rate	$9,000	$19,000
Chronic bronchitis		
Base estimate: Willingness-to-pay	$2,000	$3,800
Alternative estimate: Cost-of-illness		
3% discount rate	$590	$1,100
7% discount rate	$380	$680
Non-fatal myocardial infarctions		
3% discount rate	$1,100	$1,900
7% discount rate	$1,000	$1,800
Hospital admissions – Respiratory Causes	$73	$150
Total hospital admissions – Cardiovascular Causes	$66	$120
Emergency Room Visits for Asthma	$2	$4
Acute bronchitis	$5	$8
Lower respiratory symptoms	$2	$4
Upper respiratory symptoms	$3	$5
Work loss days	$130	$200
Minor restricted activity days	$350	$540
School absence days	$6	$15
Worker productivity	$10	$22
Welfare Endpoints		
Recreational visibility	$1,100	$2,900
Agricultural crop damage	$32	$31
Monetized Total		
Base estimate		
3% discount rate	$55,000	$113,000
7% discount rate	$52,000	$107,000
Alternative estimate		
3% discount rate	$11,300	$23,000
7% discount rate	$12,100	$25,000

[a] Ozone-related mortality is not included in the estimate of premature mortality. Respiratory hospital admissions for PM includes admissions for COPD, pneumonia, and asthma, and ozone-related respiratory admissions includes all respiratory causes and subcategories for COPD and pneumonia. Cardiovascular hospital admissions for PM includes total cardiovascular and subcategories for ischemic heart disease, dysrhythmias, and heart failure.

[b] Long-term exposure mortality was modeled as lagged, both in the base analysis and in the sensitivity analyses. The values shown here were adjusted to take this into account. For example, the base analysis assumes that 25 percent of premature deaths occur in the first year, 25 percent occur in the second year, and 16.7 percent occur in each of the three subsequent years after exposure. Using this lag structure, to account for the preferences of individuals for current risk reductions relative to future risk reductions, we discount the value of avoided premature mortalities occurring beyond the analytical year (2010 or 2020) using three and seven percent discount rates. No lag adjustment is necessary for the Alternative Estimate, which focuses on premature mortality occurring within a few days of the PM exposure.

The first approach presented, the Base Estimate, is a peer-reviewed method developed for previous risk and benefit-cost assessments carried out by the Environmental Protection Agency. This method was used in the Regulatory Impact Analyses (RIAs) of the Heavy Duty Diesel and Tier II Rules, the first Prospective Section 812 Report to Congress on the Costs and Benefits of the Clean Air Act and the draft RIA for the proposed Nonroad Diesel Engines Rule. Following the approach of these earlier assessments, we present various sensitivity analyses on the Base Estimate that alter select subsets of variables along with the results of the Base Estimate; these sensitivity analyses yield results as much as 12 percent lower to over 170 percent higher. By far, the largest component of these monetized benefits is related to premature mortality from long-term exposure to particulate matter ($50 billion and $100 billion in 2010 and 2020, respectively), followed by chronic bronchitis ($2.0 billion and $3.8 billion in 2010 and 2020, respectively), and non-fatal myocardial infarctions ($1.1 billion and $1.9 billion in 2010 and 2020, respectively).

In order to provide some insight into the potential importance of the key elements underlying estimates of the benefits of reducing SOx and NOx emissions, the Administration has also developed an Alternative Estimate using different choices of data, methods, and assumptions that are detailed in Section 2. This Alternative approach was also used in the draft RIA of the Nonroad Diesel Engines Rule. A similar approach was first developed in the context of the analysis of the Clear Skies Act of 2002 As indicated in Table 1, the differences between the Alternative and Base Estimates are found in (1) the estimation of the impact of fine particle reductions on premature mortality, (2) the valuation of reducing the risk of premature mortality, and (3) the valuation of reducing the risk of chronic bronchitis. The Alternative Estimate of the impact of fine particle reductions on premature mortality relies on recent scientific studies finding an association between daily mortality and acute (days to weeks) exposure to particulate matter, while the Base Estimate relies on a recent reanalysis of earlier studies that found associations between chronic exposure to fine particles and premature mortality. The Alternative approach also uses different data to value reductions in the risk of premature mortality and chronic bronchitis and makes adjustments relating to the health status and potential longevity of the populations most likely affected by PM. Even using these alternative assumptions, the benefits of Clear Skies still outweigh the projected costs of the proposal.

All such benefit estimates are subject to a number of assumptions and uncertainties, which are discussed in Section 3 of this report. For example, key assumptions underlying the Base and Alternative Estimates for the mortality category include the following: (1) Inhalation of fine particles is causally associated with premature death at concentrations near those experienced by most Americans on a daily basis. While biological mechanisms for this effect have not yet been definitively established, the weight of the available epidemiological evidence supports an assumption of causality; (2) All fine particles, regardless of their chemical composition, are equally potent in causing premature mortality. This is an important assumption because fine particles from power plant emissions are chemically different from directly emitted fine particles from both mobile sources and other industrial facilities, but no clear scientific grounds exist for supporting differential effects estimates by particle type; (3) The concentration-response (C-R) function for fine particles is approximately linear within the range of ambient concentrations under consideration. Thus, the estimates include health benefits from reducing fine particles in areas with varied concentrations of particulate matter, including both regions that

are in attainment with fine particle standard and those that do not meet the standard; (4) The forecasts for future emissions and associated air quality modeling are valid. Although recognizing the difficulties, assumptions and inherent uncertainties in the overall enterprise, these analyses are based on peer-reviewed scientific literature and up-to-date assessment tools, and we believe the results are highly useful in assessing this proposal.

In addition to the quantified and monetized benefits summarized above, there are a number of additional categories that are not currently amenable to quantification or valuation. See Section 2.4.1 for details. Unquantified benefits include: reduced mercury accumulation in fish and other wildlife; reduced exposure to mercury through fish consumption; reduced acid and particulate deposition damage to cultural monuments and other materials; reduced ozone effects on forested ecosystems; and acidification in lakes and streams; and reduced eutrophication in coastal areas. Additionally, we have not quantified a number of known or suspected health effects linked with PM and ozone for which appropriate concentration-response functions are not available or which do not provide easily interpretable outcomes (i.e. changes in lung function, as measured by forced expiratory volume (FEV_1)). As a result, both the Base and Alternative monetized benefits estimates underestimate the total benefits attributable to the Clear Skies Act.

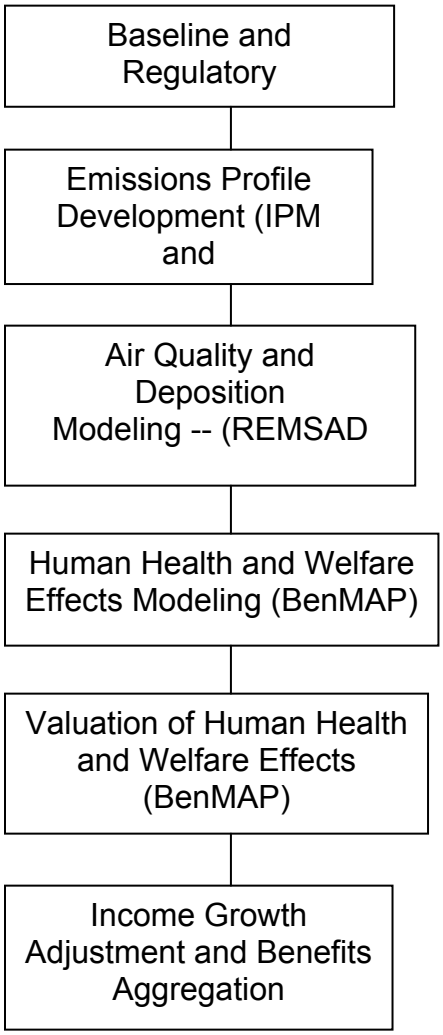
2. ANALYTICAL APPROACH

The framework for the benefits analysis of the Clear Skies Act of 2003 is the same as that used in the analysis of the Clear Skies Act of 2002. This framework was also used in four recent state-of-the-art EPA regulatory analyses: the Section 812 Prospective Report to Congress on the Benefits and Costs of the Clean Air Act (U.S. EPA, 1999a); the Tier II Motor Vehicle Emissions Standards/Gasoline Sulfur Rules Regulatory Impact Analysis (RIA) (U.S. EPA, 1999b); the Heavy-Duty Engine Vehicle Standards/Diesel Fuel Sulfur Rules RIA (U.S. EPA, 2000b); and the Nonroad Diesel Engines Rule Draft RIA (U.S. EPA, 2003). This analysis uses the same health effect and valuation functions employed in the most recent of these analyses, the Nonroad Diesel Engines Draft RIA. The analytical approach can be described as a sequence of six steps, summarized below and described in detail later in this report. These steps, listed in order of completion, are:

1. Scenario development
2. Emissions modeling
3. Air quality modeling
4. Human health and visibility effects estimation
5. Economic valuation
6. Adjustments for income growth and benefits aggregation

Figure 1 outlines the analytical framework used to study the benefits of the Clear Skies Act.

Figure 1
Analytic Sequence for
Multi-Emissions Reduction
Proposal Benefits
Analysis



The first step in the benefits analysis is the specification of the regulatory scenarios that will be evaluated. Typically, an analysis will include a baseline scenario that simulates future conditions in the absence of the proposed regulation and one or more control scenarios that simulate conditions under the regulations being evaluated. The benefits of a proposed regulation are then estimated as the difference in benefit outcomes (e.g., adverse health effects) between the control and baseline scenarios. For this analysis, the baseline scenarios for 2010 and 2020 assume no additional emissions control regulation beyond the continuing effects of Title IV of the Clean Air Act Amendments, the NO_x State Implementation Plan (SIP) Call, other promulgated state rules and federal rules issued under the Clean Air Act up through March 2003, and the proposed Nonroad Diesel Engines Rule. For each year (2010 and 2020), our analysis evaluates a single control scenario, as described below.

After scenario development, the second step of the benefits analysis is the estimation of the effect of the Clear Skies Act on emissions sources. Current emissions inputs were derived from the 1996 National Toxics Inventory and the 1996 National Emissions Inventory, updated to a 2001 base year inventory. Using the current emissions inventory, baseline future year emissions projections were generated for sectors except electric generating units using economic and population growth projections along with emission reductions from recent regulations. The Integrated Planning Model (IPM) was used to derive all future projections of electricity generation source emissions. IPM generated emissions estimates for the Clear Skies Act control scenario using the same set of economic activity projections as the baseline but with additional emissions controls consistent with the Clear Skies Act caps.

After the emissions inventories are developed, they are translated into estimates of future-year air quality conditions under each scenario. We employed two sophisticated computer models, the Regulatory Modeling System for Aerosols and Deposition (REMSAD) and the Comprehensive Air Quality Model with Extensions (CAMx) to estimate changes to the concentration of particulate matter and ozone, respectively, resulting from the Clear Skies Act. The REMSAD model was also used to estimate changes in visibility associated with those changes in particulate matter concentrations and to estimate changes in deposition of sulfur, nitrogen, and mercury.

The air quality modeling results serve as inputs to a modeling system that translates air quality changes to changes in health outcomes (e.g., premature mortality, emergency room visits) through the use of concentration-response functions. Scientific literature on the health effects of air pollutants provides the source of these concentration-response functions. At this point, we derive estimates of the differences between the two scenarios in terms of incidences of a range of human health effects that are associated with exposure to ambient particulate matter and ozone.

In the next step, economic valuation models or coefficients are used to estimate a dollar value for the reduced incidence of those adverse effects amenable to monetization. For example, analysis of estimates derived from the economic literature provides an estimate of the value of reductions in mortality risk. Finally, the benefit values are adjusted for expected income growth through 2010 and 2020 and aggregate the benefits to the appropriate geographic level.

As noted in Section 1.2, Base and Alternative estimates are presented for mortality and chronic bronchitis benefits. The different methodologies and assumptions for these approaches are discussed in separate subsections in the effects estimation and valuation sections below.

2.1. Baseline and Regulatory Scenario Development

This analysis looks at the impacts of the multi-pollutant reductions that are part of the Clear Skies Act for two future target years, 2010 and 2020. Avoided health effects and visibility improvements are quantified by comparing two scenarios:

(1) A baseline scenario (Base Case) that reflects the continuing effects of Title IV of the Clean Air Act Amendments (the Acid Rain Program) as well as other promulgated

federal rules issued under Clean Air Act authority that are expected to affect electric generating units (EGUs). It also includes promulgated federal rules that will affect other sources of emissions (e.g. the NO_x SIP call and the Tier II and Heavy Duty Diesel Rules for mobile sources). Finally, it includes the proposed Nonroad Diesel Engines Rule.

(2) A control scenario that reflects the emissions reductions expected under the baseline scenario plus implementation of the Clear Skies Act in the target year.

2.2. Emissions Profile Development

Emission inventories were developed to support the benefits analysis for the Clear Skies Act. Emissions profiles were generated for the following cases: 2001 Base Year, 2010 Base Case, 2010 Clear Skies, 2020 Base Case, and 2020 Clear Skies.

These national inventories were prepared for the 48 contiguous states at the county-level for electric generating unit (EGU) sources, non-EGU point sources, stationary area sources, and mobile sources. The approach used to create inventories was the same as that used for the Heavy-Duty Engine/Diesel Fuel Rule RIA (US EPA, 2000d). Modifications were made to reflect emission and modeling advances since that analysis.

Power generation emissions of SO_2 and NOx for each of the scenarios are presented in The Clear Skies Act: Technical Support Package.[4] Table 2 presents total national emissions of NO_x and SO_2 from all sectors, including electric power generation.

Table 2
National SO_2 and NOx Emissions Projections for Base and Clear Skies Scenarios
(million tons)

Scenario	SO_2 Emissions, All sectors	NOx Emissions, All sectors
2001 Base Year	16.6	23.0
2010 Base Case	15.3	17.7
2010 Clear Skies	11.7	16.0
2020 Base Case	14.7	14.6
2020 Clear Skies	10.1	12.3

[4] The Clear Skies Act: Technical Support Package (July 11, 2003) describes EPA's analysis of the Clear Skies Act of 2003, including program elements, health and environmental benefits, costs, and impacts on the power sector. The full document can be found at www.epa.gov/clearskies/ 03technical_packagetofc.pdf.

2.2.1 Emissions Inventories from Non-EGUs

Due to significant reductions in both SO_2 and NOx emissions and measured ambient concentration data between 1996 and 2001, the base year inventory was updated from the 1996 base year inventory (used in several rulemakings and in the analysis of the Clear Skies Act of 2002) to a 2001 base year inventory. The 2001 base year modeling inventory for criteria pollutants was developed from the 1996 National Emissions Inventory (NEI) used for the Heavy Duty Diesel Vehicle Rule and the proposed Nonroad Diesel Engines Rule, using the 2010 projection of that 1996 inventory and the preliminary 2001 NEI inventory. The EGU inventory was developed by applying State level ratios of the 1996 and 2001 NEI inventories to the 1996 modeling inventory. The mobile and nonroad inventories were developed by applying ratios of the 1996 and 2001 NEI inventories by grid cell. The area source, livestock, and non-EGU point source sectors for 2001 were developed from a linear interpolation between the 1996 and 2010 modeling inventories for each grid cell.

For mercury, the 1996 National Toxics Inventory (NTI) was used for all sources except the three largest categories: EGUs, Municipal Waste Combustors (MWCs), and Medical Waste Incinerators (MWIs). Coal-fired EGU mercury was estimated using industry provided information for a 1999 base year. MWC and MWI mercury was based on preliminary NTI information also for a 1999 base year as developed for the MACT program for these categories. No adjustment for growth was made to this hybrid 1996/1999 mercury inventory to represent the 2001 base year.

The 2010 and 2020 Base Case inventories for all sectors except EGUs were developed using economic and population growth projections along with emission reductions from recent regulations, including the NOx SIP Call, Tier II vehicle standards, Heavy Duty Diesel vehicle standards, and the proposed Nonroad Diesel Vehicle standards. [The mobile and nonroad sectors are projected using the MOBILE and NONROAD models respectively.]

2.2.2. Emissions Projections from EGUs: The Integrated Planning Model (IPM)

The EGU portion of the future base and control cases were developed using the Integrated Planning Model (IPM). IPM predicts future emissions outputs from EGUs affected by the Clear Skies Act. These outputs are used to develop the emissions inventories.

IPM is a linear programming model of the electricity sector that finds the most efficient (i.e. least cost) approach to operating the electric power system over a given time period subject to specific constraints (e.g. pollution caps or transmission limitations). The model, which was developed for EPA by ICF Resources, Inc., selects investment strategies given the cost and performance characteristics of available options, forecasts of customer demand for electricity, and reliability criteria. System dispatch, which determines the proper and most efficient use of the existing and new resources available to utilities and their customers, is optimized given the resource mix, unit operating characteristics, and fuel and other costs. Unit and system operating constraints provide system-specific realism to the outputs of the model.

The IPM is dynamic; it has the capability to use forecasts of future conditions, requirements, and option characteristics to make decisions for the present. This ability replicates, to the extent possible, the perspective of utility managers, regulatory personnel, and the public in reviewing important investment options for the utility industry and electricity consumers. Decisions are made based on minimizing the net present value of capital and operating costs over the full planning horizon. IPM also models a variety of environmental market mechanisms, such as emissions caps, allowances, trading, and banking.[5]

IPM's projections for electric utilities under the Base Case include power sector emissions under Title IV of the 1990 Clean Air Act Amendments (The Acid Rain Program), which caps SO_2 emissions at 8.95 million tons/year beginning in 2010. In addition, IPM's projections for electric utilities under the Base Case include the NO_X SIP Call with a cap on summertime NO_X emissions in SIP Call states beginning in 2004 and state limits on NO_X from EGUs in Texas, Connecticut, Missouri, Massachusetts, New Hampshire, and North Carolina. The Base Case also includes state limits on SO_2 from EGUs in Connecticut, Massachusetts, New Hampshire, North Carolina, Texas, Wisconsin. In addition, the Base Case includes EGU mercury limits in Wisconsin.

IPM was also used to project emissions for electric utilities in the future under a Clear Skies scenario. The 2010 and 2020 Clear Skies Act profile includes all of the programs included in the Base Case. In addition, the Clear Skies projections include a 4.5 million ton per year national cap on EGUs beginning in 2010 for SO_2 emissions, which would be lowered to a 3 million ton cap in 2018; a 2.1 million ton per yr cap beginning in 2008 for NO_X emissions, which would be lowered to a 1.7 million ton cap in 2018; and a 26 ton per yr cap beginning in 2010 for mercury emissions, which would be lowered to a 15 ton cap in 2018. Because sources can reduce emissions early, earn allowances for these actions, and use the allowances later, sources are expected to achieve some emissions reductions before the cap comes into effect. However, because of these early emissions reductions, actual emissions are projected to be higher than the cap in the first years under each cap.

2.3. Air Quality and Deposition Modeling

Air quality modeling is a critical analytical step that provides the link between emissions changes and the physical effects that affect human health and the environment. Using emissions inventories developed for the Base and Control cases, this step of the analysis employs complex computer models that simulate the transport and transformation of emitted pollutants in the atmosphere. The results of these model runs are predictions of pollutant concentrations under each of the emission control scenarios specified above. These predicted concentrations are then used as inputs to the human health effect estimation model discussed in the next section.

Air quality modelers face two key challenges in attempting to translate emission inventories into pollutant concentrations. First, they must model the dispersion and transport of

[5] Complete documentation of the IPM model can be found at http://www.epa.gov/airmarkets/epa-ipm/index.html

pollutants through the atmosphere. Second, they must model pertinent atmospheric chemistry and other pollutant transformation and removal processes. These challenges are particularly difficult for those pollutants that are not emitted directly but instead form through secondary processes. Ozone is the best example; it forms in the atmosphere through a series of complex, non-linear chemical interactions of precursor pollutants, particularly certain classes of volatile organic compounds (VOCs) and nitrogen oxides (NO_x). Modelers face similar challenges when estimating fine particle concentrations. Atmospheric transformation of gaseous sulfur dioxide and nitrogen oxides to particulate sulfates and nitrates, respectively, contributes significantly to ambient concentrations of fine particulate matter. In addition to recognizing the complex atmospheric chemistry relevant for some pollutants, air quality modelers also must deal with uncertainties associated with variable meteorology and the spatial and temporal distribution of emissions.

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

2.3.1. Regional Modeling System for Aerosols and Deposition (REMSAD)

The change in particulate matter (PM) concentrations due to the Clear Skies Act was modeled using REMSAD. REMSAD was also used to estimate the changes in visibility and deposition of mercury, nitrogen, and sulfur. REMSAD is a three-dimensional, grid-based Eulerian air quality model designed to simulate long-term (e.g., annual) concentrations and deposition fluxes of atmospheric pollutants over large spatial scales (e.g., over the contiguous U.S.). Air pollution issues meant to be addressed by REMSAD include long-term $PM_{2.5}$ ambient concentrations; visibility; ambient concentrations and deposition fluxes of several hazardous air pollutants, including mercury; deposition fluxes of nutrient nitrogen; and deposition of acids such as sulfuric acid and nitric acid.

REMSAD has been developed under funding from the U.S. Environmental Protection Agency over the past five years. REMSAD consists of three components: (1) a meteorological data pre-processor; (2) the core aerosol and toxic deposition model (ATDM); and (3) post-processing programs. The horizontal grid size can be on the order of a few kilometers (km) for an urban-scale simulation up to about 100 km for a continental-scale simulation. For large-scale simulations, one-way nesting of fine and coarse grids can be performed to allow simulation of sensitive areas with strong pollution spatial gradients using a fine grid resolution. The vertical structure of REMSAD covers the whole troposphere from the surface up to about 15 km. The physical and chemical processes simulated by REMSAD include emissions of pollutants from surface and elevated sources, advective transport, horizontal turbulent diffusion, vertical mixing via turbulent diffusion and convective transport, cloud processes, gas-phase and aqueous-phase chemistry, $PM_{2.5}$ formation, dry deposition and wet deposition.

Version 7.06 of REMSAD was employed for this analysis. Previous versions of REMSAD have been used to estimate PM for EPA's Section 812 Prospective Report to Congress (U.S. EPA, 1999a); the Heavy-Duty Engine/Diesel Fuel RIA (U.S. EPA, 2000b); the 2002 analysis of the Clear Skies Act; and the Nonroad Diesel Engines Rule Draft RIA (U.S. EPA, 2003). REMSAD Version 7.06 reflects updates in the following areas to improve performance and address comments from the 1999 peer-review:

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

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

3. Aqueous phase chemistry updates to incorporate the oxidation of SO_2 by O_3 and O_2 and to include the cloud and rain liquid water content from MM5 meteorological data in sulfate production and deposition calculations.

Additional updates were recently made to REMSAD (version 7.06) to fix an error in the secondary organic aerosol mechanism and to revise certain aspects of the dry deposition code.

The REMSAD modeling domain selected for the Clear Skies Act consists of 36 km x 36 km grid cells covering the 48-contiguous United States, and REMSAD can perform a full-year simulation, generating predictions of hourly PM concentrations (including both $PM_{2.5}$ component species and PM_{10}) at each grid cell. These hourly predictions form the basis for estimates of daily and annual PM air quality metrics (e.g., annual mean PM concentration) as inputs to the health and welfare C-R functions of the benefits analysis. REMSAD also yields estimates of visibility, which are used as an input into the visibility damage function.

For the health and welfare benefits analysis, we applied REMSAD to the entire U.S. for four future-year scenarios: a 2010 Base Case, a 2020 Base Case, a 2010 Clear Skies Act Case, and a 2020 Clear Skies Act Case. Air quality monitoring data for 2001 were adjusted using these REMSAD results to estimate PM concentrations in each grid cell under each scenario (see Section 2.4.2). The difference in grid cell PM concentrations between Base and Clear Skies scenarios represents the expected change in PM due to the emission controls under the Clear Skies Act exposure for the population living in that grid cell.

2.3.2. Comprehensive Air Quality Model with Extensions (CAMx)

We modeled changes in ozone in the eastern U.S. CAMx. This model was also used in to model ozone changes in the analysis of the Clear Skies Act of 2002 and in the Nonroad Diesel Engines Rule Draft RIA. CAMx is an Eulerian photochemical dispersion model designed to assess air pollution over many scales, from urban to super-regional. The model estimates concentrations of both inert and chemically reactive pollutants by simulating the physical and

chemical processes in the atmosphere that affect ozone formation. Because it accounts for spatial and temporal variations as well as differences in the reactivity of emissions, the CAMx is useful for evaluating the impacts of the Clear Skies Act on U.S. ozone concentrations. Although the model tends to underestimate observed ozone, especially over the western U.S., it exhibits less bias and error than any past regional ozone modeling application conducted by EPA (i.e., OTAG, Tier-2, and Heavy Duty Diesel). The latest version of the model, CAMx 3.10, provides full support for parallel processing for increased computational performance, as well as new algorithms for gas phase chemistry (CAMx v3.10 User's Guide, April 2002).

The modeling domain for this analysis encompasses most of the eastern U.S., bounded on the east by the 67 degrees west longitude and on the west by the 99 degrees west longitude. The horizontal resolution for the outer grid is approximately 36 km. The horizontal resolution for the inner grid is approximately 12 km (see below for further description). The vertical resolution for both grids consists of nine layers. The top of the modeling domain is 4,000 meters above ground level. Recognizing the relationship between grid cell resolution and the certainty of results, we sought to estimate pollutant concentrations in more populated areas using higher resolution models. Similarly, we used an intermediate resolution grid (12 km x 12 km) to model ozone in "inner OTAG" states where population density is high and ozone transport is a major problem.[6] This approach makes CAMx well-suited to estimate effects based on a range of ozone averaging times, an important capability for benefits assessment applications.

This study extracted hourly, surface-layer ozone concentrations for each grid-cell from the standard CAMx output file containing hourly average ozone values. These model predictions are used in conjunction with the observed concentrations obtained from the Aerometric Information Retrieval System (AIRS) to generate ozone concentrations for the entire ozone season.[7, 8] The predicted changes in ozone concentrations from the Base Case to the Clear Skies Act serve as inputs to the health and welfare C-R functions of the benefits analysis, (i.e., BenMAP).

In order to estimate ozone-related health and welfare effects for the eastern U.S., full-season ozone data are required for every CAMx grid-cell. Given available ozone monitoring data, we generated full-season ozone profiles for each location by combining monitored observations and modeled ozone predictions to interpolate hourly ozone concentrations to an air quality grid, as will be described in the Human Health and Environmental Effects Modeling section.[9] For the analysis of ozone impacts on agriculture, we use a similar approach except air quality is interpolated to county centroids as opposed to population grid-cells. We report ozone

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

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

[8] Based on AIRS, there were 949 ozone monitors with sufficient data, i.e., at least 9 hourly observations per day (8 am to 8 pm) in a given season.

[9] This approach is a generalization of planar interpolation that is technically referred to as enhanced Voronoi Neighbor Averaging (EVNA) spatial interpolation (See Abt Associates (2000) for a more detailed description).

concentrations as a cumulative index called the SUM06. The SUM06 is the sum of the ozone concentrations for every hour that exceeds 0.06 parts per million (ppm) within a 12-hour period from 8 am to 8 pm in the months of May to September. These methods are described in detail in the Heavy Duty Diesel Fuel RIA (USEPA, 2000b).

2.4. Human Health and Environmental Effects Modeling

To estimate health and environmental benefits from the Clear Skies Act, we used the same general approach used in recent major EPA regulatory analyses (U.S. EPA, 1999a, 1999b, 2000b and 2003). This approach takes the estimates of changes in ambient pollutant concentrations predicted by air quality modeling for each scenario (relative to the baseline scenario) and converts them into estimates of changes in the incidence of adverse health effects using concentration-response (C-R) functions. The model we use to generate these estimates is BenMAP (the Environmental Benefits Mapping and Analysis Program).

BenMAP aggregates population to air quality model grids and calculates changes in air pollution metrics (e.g., daily averages) for input into C-R functions. C-R functions are equations that relate the change in the number of individuals in a population exhibiting a "response" (in this case an adverse health effect such as respiratory disease) to a change in pollutant concentration experienced by that population. In general, the C-R functions used in BenMAP require four input values: (1) the grid-cell-specific change in pollutant concentration; (2) the grid-cell affected population (i.e. asthmatic children); (3) the baseline incidence rate of the health effects; and (4) an estimate of the change in the number of individuals that suffer an adverse health effect per unit change in air quality. Both the form of the C-R function and the estimate of the change in the number of individuals that suffer an adverse health effect per unit change in air quality are derived from epidemiological studies in the scientific literature that link pollutant exposures with adverse health effects.

In addition to our national benefits results, we generated regional estimates of the benefits of the Clear Skies Act using the same benefits estimation procedure used to generate the national estimates. The REMSAD and CAMx air quality models provide information on the improvements in ambient air concentrations throughout the country within the REMSAD/CAMx gridboxes. This information is used in subsequent exposure, dose-response, and valuation steps. This "bottom-up" approach provides a more accurate representation of regional benefits estimates than a comparable "top-down", emissions-weighted approach might, particularly given the importance of long-range transport for the major pollutants controlled by the Clear Skies Act (SO_2 and NO_x, as well as mercury).

Recreational visibility benefits can also be geographically disaggregated, based on either the location of the recreational Class I area where visibility is improved or on the state of origin of visitors to these sites. For this analysis, we disaggregated benefits based on the state of origin of visitors, reflecting the notion that many of the recreational sites with the highest visitation rates are valued by individuals throughout the country, not only by those individuals who live closest to the site. The results of the regional analysis of visibility benefits indicate that benefits are realized throughout the country, with a higher concentration of benefits in those areas of higher population density.

2.4.1. Health and Ecological Endpoints: Quantified and Unquantified Effects

As part of the evaluation of the effects of various scenarios concerning SO_2 and NO_x emissions, we have identified and, where possible, developed quantitative, monetized estimates of health benefits. Our analysis also looked at several environmental endpoints, including the benefits associated with visibility improvements, ozone damage to agriculture, and changes in acidification in lakes and streams.

Table 3 provides a list of the health effects for which we estimate quantified benefits as part of our analysis and a list of the health effects for which we are unable to quantify benefits at this time. The unquantified benefits for ozone and PM fall into two categories: (1) those for which the scientific literature does not provide an established C-R function capable of estimating health effects with reasonable certainty and (2) those effects that may double-count benefits (e.g., hospital admissions for specific cardiovascular illnesses). The direct health effects of nitrogen oxide gases and sulfur dioxide gases are also unquantified. Although C-R functions are available to estimate health effects of exposure to nitrogen oxides and sulfur dioxide gases, these effects were not estimated in this analysis because of modeling and resource limitations.

The health and environmental effects of mercury exposure are also not quantified. EPA is currently investigating methods to quantify and monetize the human health related benefits of reductions in air emissions of mercury. However, there are still major gaps in the science of mercury fate, transport, and transformation that make such an assessment difficult at time. Methods for mercury benefits analyses are still under development and do not yet provide a means to estimate the mercury-related benefits of the Clear Skies Act.

Table 4 provides a list of the welfare effects associated with the emissions targeted by Clear Skies. As stated earlier, most of these effects have not been quantified as part of our analysis, due to data or modeling limitations. We have, however, monetized effects of changes in ambient ozone on some agricultural production and changes in particulate matter on visibility.

Table 3:
Human Health Effects of Air Pollutants Affected by Clear Skies

Pollutant	Quantified Health Effects	Quantified and/or Monetized Effects in Sensitivity Analyses	Unquantified Health Effects
PM	Premature mortality – long term exposures Premature mortality – short term exposures Bronchitis – chronic and acute Hospital admissions - respiratory and cardiovascular Emergency room visits for asthma Non-fatal heart attacks (myocardial infarction) Lower and upper respiratory illness Minor restricted activity days Work loss days	Asthma attacks (asthmatic population)[a] Respiratory symptoms (asthmatic population) Infant mortality	Low birth weight Changes in pulmonary function Chronic respiratory diseases other than chronic bronchitis Morphological changes Altered host defense mechanisms Cancer Non-asthma respiratory emergency room visits Changes in cardiac function (e.g. heart rate variability) Allergic responses (to diesel exhaust)
Ozone	Hospital admissions - respiratory Emergency room visits for asthma Minor restricted activity days School loss days Worker productivity	Asthma attacks (asthmatic population)[a]	Increased airway responsiveness to stimuli Inflammation in the lung Chronic respiratory damage Premature aging of the lungs Acute inflammation and respiratory cell damage Increased susceptibility to respiratory infection Non-asthma respiratory emergency room visits Chronic asthma[b] Cardiovascular emergency room visits Premature mortality – acute exposures[c] Acute respiratory symptoms
Mercury			Neurological disorders Learning disabilities Retarded development Cardiovascular effects Altered blood pressure regulation Increased heart rate variability Myocardial infarctions Reproductive effects
Nitrogen Oxides			Lung irritation Lowered resistance to respiratory infection Hospital Admissions for respiratory and cardiac diseases
Sulfur Dioxide			Hospital admissions for respiratory and cardiac diseases Respiratory symptoms in asthmatics

[a] For adults only, asthma attacks may be represented in the primary estimate of minor restricted activity days.
[b] While no causal mechanism has been identified linking new incidences of chronic asthma to ozone exposure, two epidemiological studies shows a statistical association between long-term exposure to ozone and incidences of chronic asthma in exercising children and some non-smoking men (McConnell, 2002; McDonnell, et al., 1999).
[c] Premature mortality associated with ozone is not separately included in the calculation of total monetized benefits.

Table 4
Welfare Effects of Air Pollutants Affected by Clear Skies

Pollutant	Quantified Effects	Quantified and/or Monetized Effects in Sensitivity Analyses	Unquantified Effects
Particulate Matter (PM_{10}, $PM_{2.5}$)	Visibility in California, Southwestern, and Southeastern Class I areas	Visibility in Northern, Northwestern, and Midwestern Class I areas Visibility in residential and non-Class I areas Household soiling	
Ozone	Decreased yields for commercial and non-commercial crops		Decreased commercial forest productivity Decreased yields for fruits and vegetables Damage to urban ornamental plants Impacts on recreational demand from damaged forest aesthetics Damage to ecosystem functions Decreased outdoor worker productivity
Nitrogen and Sulfate Deposition	Impacts to some freshwater aquatic habitats in the Adirondacks are quantified but not monetized		Impacts of acidic sulfate and nitrate deposition on commercial forests Impacts of acidic deposition on commercial freshwater fishing Impacts of acidic deposition on recreation in terrestrial ecosystems Impacts of nitrogen deposition on commercial fishing, agriculture, and forests Impacts of nitrogen deposition on recreation in estuarine ecosystems Reduced existence values for currently healthy ecosystems
Mercury Deposition			Impacts on birds and mammals (e.g. reproductive effects) Impacts to commercial, subsistence, and recreational fishing Reduced existence values for currently healthy ecosystems

Tables 5 and 6 provide a list of the health effect endpoints we quantified as part of our analysis of the Clear Skies Act, as well as references to the studies that serve as the basis for the C-R functions. As with emissions and air quality estimates, our estimates of the effect of ambient pollution levels on all of these endpoints represent the best science and analytical tools available. With the exception of short-term mortality, myocardial infarctions, and school loss days, the choice of C-R functions and the majority of the analytical assumptions used to develop our estimates have been reviewed and approved by EPA's Science Advisory Board (SAB). The C-R functions in Table 5 only capture effects related to exposures to particulate matter and ozone; they do not include human health effects related to exposures to SO_2, NO_x, or mercury. As a result, for these exposures, we have underestimated the total health benefits attributable to Clear Skies emissions reductions.

Table 5
PM-Related Health Endpoints Used in Base and Alternative Estimates

Health Effect	Applied Ages	Pollutant	Source of Effect Estimate	Source of Baseline Incidence [1]
Mortality				
Long-term – Base Estimate	30+	$PM_{2.5}$	Krewski et al. (2000), reanalysis of Pope et al. (1995) using the annual mean and all-cause mortality	CDC Wonder (1996-1998) [2]
Short-term – Alternative Estimate	All	$PM_{2.5}$	Schwartz et al. (1996) adjusted using ratio of distributed lag to single day coefficients from Schwartz (2000b)	
Chronic Illness				
Chronic bronchitis	27+	$PM_{2.5}$	Abbey et al (1995)	1999 HIS (American Lung Association, 2002b, Table 4); Abbey et al. (1993, Table 3)
Non-fatal heart attacks	18+	$PM_{2.5}$	Peters et al. (2001)	1999 NHDS public use data files;[3] adjusted by 0.93 for prob. of surviving after 28 days (Rosamond et al., 1999)
Hospital Admissions				
Respiratory	65+	$PM_{2.5}$	Pooled estimate: Moolgavkar (2000b) - ICD 490-496 (COPD) Lippmann et al. (2000) - ICD 490-496 (COPD)	1999 NHDS public use data files[3]
	20-64	$PM_{2.5}$	Moolgavkar (2000b) - ICD 490-496 (COPD)	
	65+	$PM_{2.5}$	Lippmann et al. (2000) - ICD 480-486 (pneumonia)	
	<65	$PM_{2.5}$	Sheppard, et al. (1999) - ICD 493 (asthma)	
Cardiovascular	65+	$PM_{2.5}$	Pooled estimate: Moolgavkar (2000a) - ICD 390-429 (all cardiovascular) Lippmann et al. (2000) - ICD 410-414, 427-428 (ischemic heart disease, dysrhythmia, heart failure)	
	20-64	$PM_{2.5}$	Moolgavkar (2000a) - ICD 390-429 (all cardiovascular)	

Table 5 PM-Related Health Endpoints Used in Base and Alternative Estimates				
Health Effect	**Applied Ages**	**Pollutant**	**Source of Effect Estimate**	**Source of Baseline Incidence** [1]
Emergency Room Visits				
Asthma ER Visits	0-18	$PM_{2.5}$	Norris et al. (1999)	2000 NHAMCS public use data files;[4] 1999 NHDS public use data files[3]
Other Health Effects				
Acute bronchitis	Ages 8-12	$PM_{2.5}$	Dockery et al. (1996)	American Lung Association (2002a, Table 11)
Upper respiratory symptoms	Asthmatics, ages 9-11	PM_{10}	Pope et al. (1991)	Pope et al. (1991, Table 2)
Lower respiratory symptoms[5]	7-14	$PM_{2.5}$	Schwartz et al. (1994)	Schwartz (1994, Table 2)
Work loss days	18-64	$PM_{2.5}$	Ostro (1987)	1996 HIS (Adams et al., 1999, Table 41); U.S. Bureau of the Census (2000)
Minor restricted activity day	18-64	$PM_{2.5}$	Ostro and Rothschild (1989)	Ostro and Rothschild (1989, p. 243)

1. 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.
2. See http://wonder.cdc.gov/.
3. See ftp://ftp.cdc.gov/pub/Health_Statistics/NCHS/Datasets/NHDS/
4. See ftp://ftp.cdc.gov/pub/Health_Statistics/NCHS/Datasets/NHAMCS/
5. Lower Respiratory Symptoms are defined as ≥2 of the following: cough, chest pain, phlegm, wheeze.

Table 6 Ozone-Related Health Endpoints in Base and Alternative Estimates				
Health Effect	**Applied Ages**	**Pollutant**	**Description**	**Source of Baseline Incidence**[1]
Hospital Admissions				
Respiratory	65+	O_3	Pooled estimate: Schwartz (1995) - ICD 460-519 (all resp) Schwartz (1994a; 1994b) - ICD 480-486 (pneumonia) Moolgavkar et al. (1997) - ICD 480-487 (pneumonia) Schwartz (1994b) - ICD 491-492, 494-496 (COPD) Moolgavkar et al (1997) - ICD 490-496 (COPD)	1999 NHDS public use data files[2]
	<2	O_3	Burnett et al. (2001)	
Emergency Room Visits				
Asthma ER Visits	All	O_3	Pooled estimate: Weisel et al. (1995) Cody et al. (1992) Stieb et al. (1996)	2000 NHAMCS public use data files[3]; 1999 NHDS public use data files[2]
Other Health Effects				
School Loss Days[4]	9-10 6-11	O_3	Pooled estimate: Gilliland et al (2001) Chen et al (2000)	National Center for Education Statistics (1996)
Worker productivity	18-65	O_3	Crocker and Horst (1981) and EPA (1994). Applied to outdoor workers, defined as those engaged in the farming, fishing, and forestry occupations.	NA

1. The following abbreviations are used to describe the national surveys conducted by the National Center for Health Statistics: NHDS - National Hospital Discharge Survey; NHAMCS - National Hospital Ambulatory Medical Care Survey.
2. See ftp://ftp.cdc.gov/pub/Health_Statistics/NCHS/Datasets/NHDS/
3. See ftp://ftp.cdc.gov/pub/Health_Statistics/NCHS/Datasets/NHAMCS/
4. The estimate of daily illness-related school absences excludes school loss days associated with injuries to match the definition in the Gilliland et al. (2001) study.

2.4.2. Air Quality Changes

To estimate changes in pollutant concentrations for particulate matter, we used the results of the REMSAD model for each scenario. The difference in REMSAD-modeled PM concentrations for these scenarios represents the expected change in PM due to the emission controls under the Clear Skies Act. To estimate changes in pollutant concentrations for ozone, we used the results of CAMx for each scenario.

To forecast population exposure in each grid cell, for both ozone and PM, air quality monitor data are combined with the modeling results in BenMAP. At each PM and ozone monitor, we quantified the relationship between REMSAD and CAMx modeled levels of PM and ozone at the monitor for a base year (2001) and the future year (2010 or 2020). These adjustment ratios are applied to the actual monitoring data to generate estimates of PM and ozone levels at the monitor for the future scenarios. Note that we do not use the modeling data

directly to estimate future-year air quality. Instead, we use them in a relative sense to simply adjust actual, 2001 PM and ozone monitor levels to project future Base Case or Clear Skies concentrations. This provides a better estimate than the REMSAD or CAMx modeling data itself. To calculate population exposure to PM and ozone, each REMSAD or CAMx grid cell was assigned a distance-weighted average of adjusted PM or ozone levels from nearby monitors. This approach is a generalization of planar interpolation that is technically referred to as enhanced Voronoi Neighbor Averaging (eVNA) spatial interpolation (See Abt Associates, 2000 for a more detailed description).

2.4.3. Population

Health benefits are related to the change in air pollutant exposure experienced by individuals. Because the expected changes in pollutant concentrations vary from location to location, individuals in different parts of the country may not experience the same level of air quality improvement under the control scenario. We apportioned benefits among individuals by using BenMAP to match the change in air pollutant concentration in each REMSAD or CAMx grid cell with the size and composition (e.g., age distribution) of the population that experiences that change.

Integral to the estimation of such benefits is a reasonable estimate of future population projections. We extrapolated grid cell population estimates for future years using population projections based on county level allocations of national population projections from the U.S. Census Bureau (Hollman, Mulder and Kallan, 2000). County-level allocations of populations by age, race, and sex are based on economic forecasting models developed by Woods and Poole, Inc., which account for patterns of economic growth and migration (U.S. EPA, 2003). Growth factors are calculated using the Woods and Poole data and then applied to 2000 U.S. Census data.

An epidemiological study typically focuses on a particular age cohort (e.g., adults age 30 and older), and the C-R relationship found in a particular study can not necessarily be generalized across broader age categories. Therefore, to avoid overestimating the benefits of reduced pollution levels, we applied C-R relationships only to those age groups corresponding to the cohorts included in the given epidemiological study. For outcomes where the study population reflects data limitations and not the age-specificity of a health effect, this assumption may lead us to underestimate the benefits of reductions in pollutant exposures to the entire, exposed population.

2.4.4. Baseline Incidence Rates

Most C-R functions (those expressed as a change relative to baseline conditions) require baseline incidence data associated with ambient levels of pollutants. The baseline incidences for health outcomes used in our analyses are selected and adapted to match the specific populations studied. For example, we use age- and county-specific baseline total mortality rates in the estimation of PM-related premature mortality. County-level incidence rates are not available for other endpoints. We used national or regional incidence rates whenever possible because these data are most applicable to a national assessment of benefits. However, for some studies, the

only available incidence information comes from the studies themselves; in these cases, incidence in the study population is assumed to represent typical incidence at the national level. Sources of baseline incidence rates are reported in Table 5 and Table 6.

Additional information on the baseline incidence rates used in this analysis is available in the Nonroad Diesel Engines Rule Draft RIA (EPA, 2003).

2.4.5. Health Effects Concentration-Response Functions

Fundamental to the estimation of health benefits was our utilization of the PM and ozone epidemiology literature. We rely upon C-R functions derived from published epidemiological studies that relate health effects to ambient concentrations of PM and ozone. The specific studies from which C-R functions are drawn are listed in Tables 5 and 6. While a broad range of serious health effects have been associated with exposure to elevated PM and ozone levels, we include only a subset of health effects in this benefit analysis due to limitations in available C-R functions and concerns about double-counting of overlapping effects. Since the analysis of the Clear Skies Act of 2002, we have added several new endpoints, which are listed in Section 2.7 and described in detail in the Nonroad Diesel Engines Rule Draft RIA (U.S. EPA, 2003).

The C-R functions for PM and ozone exposure are the same as used for the Nonroad Diesel Engines Rule Draft RIA. We present information below on the selection of C-R functions for several of the most significant health effects evaluated (in terms of monetized benefits), premature mortality, chronic bronchitis, and myocardial infarctions. Detailed information on the selection and application of C-R functions for other endpoints in Tables 5 and 6 is available in the Nonroad Diesel Engines Rule Draft RIA (U.S. EPA, 2003). Alternative assumptions about these judgements may lead to substantially different results and they are explored using appropriate sensitivity analyses provided in Section 5.

Quantifiable health benefits of the modeled preliminary control options may be related to ozone only, PM only, or both pollutants. Decreased worker productivity, respiratory hospital admissions for children under two, and school absences are related to ozone but not PM. PM-only health effects include premature mortality, non-fatal heart attacks, asthma emergency room visits, chronic bronchitis, acute bronchitis, upper and lower respiratory symptoms, and work loss days.[10] Because of concern about overstating of benefits and because the evidence associating mortality with exposure to PM is currently stronger than for ozone, only the benefits related to the long-term exposure study (Krewkski, et al, 2000) of mortality are included in the total primary benefits estimate. Health effects related to both PM and ozone include hospital admissions and minor restricted activity days.

We relied on the available published scientific literature to ascertain the relationship

[10] Some evidence has been found linking both PM and ozone exposures with premature mortality. The SAB has raised concerns that mortality-related benefits of air pollution reductions may be overstated if separate pollutant-specific estimates, some of which may have been obtained from models excluding the other pollutants, are aggregated. In addition, there may be important interactions between pollutants and their effect on mortality (EPA-SAB-Council-ADV-99-012, 1999).

between particulate matter and ozone exposure and adverse human health effects. We evaluated studies using the selection criteria summarized in Table 7. In general, we selected C-R functions that 1) most closely match the pollutants of interest, i.e. $PM_{2.5}$ and ozone, 2) cover the broadest potentially exposed population (i.e. all ages functions would be preferred to adults 27 to 35), 3) have appropriate model specification (e.g. control for confounding pollutants), 4) have been peer-reviewed, and 5) are biologically plausible. Other factors may also affect our selection of C-R functions for specific endpoints, such as premature mortality.

Table 7

Summary of Considerations Used in Selecting C-R Functions

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

Concentration-response relationships between a pollutant and a given health endpoint are

applied consistently across all locations nationwide. This applies to both C-R relationships defined by a single C-R function and those defined by a pooling of multiple C-R functions. Although the C-R relationship may, in fact, vary from one location to another (for example, due to differences in population susceptibilities or differences in the composition of PM), location-specific C-R functions are generally not available. A single function applied everywhere may result in overestimates of incidence changes in some locations and underestimates elsewhere, but these location-specific biases will negate each other, to some extent, when the total incidence change is calculated. It is not possible to know the extent or direction of the bias in the total incidence change based on the general application of a single C-R function everywhere.

While there is a consistent body of evidence supporting a relationship between a number of adverse health effects and PM and ozone exposure, there is often only a single study of a specific endpoint covering a specific age group. There may be multiple estimates examining subgroups (i.e. asthmatic children). However, for the purposes of assessing national population-level benefits, we chose the most broadly applicable C-R function to more completely capture health benefits in the general population. Estimates for subpopulations are provided in the results section of this document.

C-R functions may also be estimated with or without explicit thresholds. Air pollution levels below the threshold for each health effect studied are assumed not to cause the effect. When no threshold is assumed, as is often the case in epidemiological studies, any exposure level is assumed to pose a non-zero risk of response to at least one segment of the population. In the benefits analyses for some recent RIAs (e.g., the Regional Haze RIA and the NOx SIP Call RIA), the low-end estimate of benefits assumed a threshold in PM health effects at 15 $\mu g/m^3$.

Based on a review of the recent literature on health effects of PM exposure (Daniels et al., 2000; Pope et al, 2002; Rossi et al., 1999; Schwartz, 2000a), we chose for the purposes of this analysis to assume that PM-related health effects occur down to natural background (i.e. there is no health effects threshold). We assume that all of the C-R functions are continuous and differentiable down to natural background levels. In addition, we explore this important assumption in a sensitivity analysis described in Section 5.3.

Recently, the Health Effects Institute (HEI) reported findings by investigators at Johns Hopkins University and others that have raised concerns about aspects of the statistical methods used in a number of recent time-series studies of short-term exposures to air pollution and health effects (Greenbaum, 2002a). Some of the concentration-response functions used in this benefits analysis were derived from such short-term studies. The estimates derived from the long-term exposure studies, which account for a major share of the benefits in the Base Estimate, are not affected. Similarly, the time-series studies employing generalized linear models (GLMs) or other parametric methods, as well as case-crossover studies, are not affected. As discussed in HEI materials provided to sponsors and to the Clean Air Scientific Advisory Committee of the SAB (Greenbaum, 2002a, 2002b), these investigators found problems in the default "convergence criteria" used in Generalized Additive Models (GAM) and a separate issue first identified by Canadian investigators about the potential to underestimate standard errors in the same statistical package. These and other investigators have begun to reanalyze the results of several important time series studies with alternative approaches that address these issues and have found a

downward revision of some results. For example, the mortality risk estimates for short-term exposure to PM_{10} from NMMAPS were overestimated (this study was not used in this benefits analysis of fine particle effects) (HEI, 2003). However, both the relative magnitude and the direction of bias introduced by the convergence issue is case-specific. In most cases, the concentration-response relationship may be overestimated; in other cases, it may be underestimated. The preliminary reanalyses of the mortality and morbidity components of NMMAPS suggest that analyses reporting the lowest relative risks appear to be affected more greatly by this error than studies reporting higher relative risks (Dominici et al., 2002; Schwartz and Zanobetti, 2002).

Our examination of the original studies used in this analysis finds that the health endpoints that are potentially affected by the GAM issues include: reduced hospital admissions in both the Base and Alternative Estimates; reduced lower respiratory symptoms in the both the Base and Alternative Estimates; and reduced premature mortality due to short-term PM exposures in the Alternative Estimate. The preliminary results from reanalyses of some of the studies we use in our Clear Skies analyses (Dominici et al, 2002; Schwartz and Zanobetti, 2002; Schwartz, personal communication 2002) suggested a more modest effect of the S-plus error than reported for the NMMAPS PM_{10} mortality study. A number of researchers submitted reanalysis reports, and final report on these reanalyses was released by the Health Effects Institute in May 2003 (HEI, 2003). The final report found that the impact of the reanalyses varied greatly across the studies. In some studies, the reanalysis had little impact, while the impact was substantial in a few studies. The reanalyses did not meaningfully change the conclusions of any of the studies, as the studies generally showed a smaller but continuing association between air pollution and health. The results of these reanalyses were incorporated in the fourth external review draft of EPA's Criteria Document released in June 2003 (US EPA, 2003b). While we wait for further clarification from the scientific community, we have chosen not to remove these results from the Clear Skies benefits estimates, nor have we elected to apply any interim adjustment factor based on the preliminary reanalyses. EPA will continue to monitor the progress of this concern, and make appropriate adjustments as further information is made available.

While a broad range of serious health effects have been associated with exposure to elevated ozone and PM levels (as noted for example in Table 3 and described more fully in the ozone and PM Criteria Documents (US EPA, 1996a, 1996b, 2003b), we include only a subset of health effects in this quantified benefit analysis. Health effects are excluded from this analysis for three reasons: (i) the possibility of double counting (such as hospital admissions for specific respiratory diseases); (ii) uncertainties in applying effect relationships based on clinical studies to the affected population; or (iii) a lack of an established C-R relationship.

In general, the use of results from more than a single study can provide a more robust estimate of the relationship between a pollutant and a given health effect. However, there are often differences between studies examining the same endpoint which make it difficult to pool the results in a consistent manner. For example, studies may examine different pollutants, or different age groups. For this reason, we consider very carefully the set of studies available examining each endpoint, and select a consistent subset that provides a good balance of population coverage and match with the pollutant of interest. In many cases, either due to a lack

of multiple studies, consistency problems, or clear superiority in the quality or comprehensiveness of one study over others, a single published study is selected as the basis of the C-R relationship.

When several estimated C-R relationships between a pollutant and a given health endpoint have been selected, they are quantitatively combined or pooled to derive a more robust estimate of the relationship. The benefits TSD for the Nonroad Diesel Engines Draft RIA provides details of the procedures used to combine multiple C-R functions (Abt Associates, 2003). In general, we use fixed or random effects models to pool estimates from different studies of the same endpoint. Fixed effects pooling simply weights each studies estimate by the inverse variance, giving more weight to studies with greater statistical power (lower variance). Random effects pooling accounts for both within-study variance and between-study variability, due for example to differences in population susceptibility. We use the fixed effects model as our null hypothesis, and then determine whether the data suggest that we should reject this null hypothesis, in which case we would use the random effects model.[8] Pooled C-R functions are used to estimate hospital admissions related to PM and asthma-related emergency room visits related to ozone. For more details on methods used to pool incidence estimates and a complete discussion of the C-R functions used for this analysis and information about each endpoint, see the benefits TSD for the Nonroad Diesel Engines Draft RIA (Abt Associates, 2003). Basic information on several of the endpoints (premature mortality, chronic bronchitis, and nonfatal heart attacks) is presented below.

Premature Mortality (Particulate Matter)

Both long and short-term exposures to ambient levels of air pollution have been associated with increased risk of premature mortality. The size of the mortality risk estimates from these epidemiological studies, the serious nature of the effect itself, and the high monetary value ascribed to prolonging life make mortality risk reduction the most important health endpoint quantified in this analysis. Because of the importance of this endpoint and the considerable uncertainty among economists and policymakers as to the appropriate way to value reductions in mortality risks, this section discusses some of the issues surrounding the estimation of premature mortality. Additional discussion is found in the section on uncertainties.

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

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

significant associations between PM and premature mortality using both types of studies. In general, the risk estimates based on the long-term exposure studies are larger than those derived from short-term studies. Cohort analyses are better able to capture the full public health impact of exposure to air pollution over time (Kunzli, 2001; NRC, 2002) than other epidemiological study designs. The Alternative Estimate is based on time-series studies demonstrating the effect of short-term exposures. This section discusses some of the issues surrounding the estimation of premature mortality.

Base Estimate

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

Given their consistent results and broad applicability to general US populations, the Six-City and ACS data have been of particular importance in benefits analyses. The credibility of these two studies is further enhanced by the fact that they were subject to extensive reexamination and reanalysis by an independent scientific analysis team of experts compiled by the Health Effects Institute (Krewski et al., 2000). The final results of the reanalysis were then independently peer reviewed by a Special Panel of the HEI Health Review Committee. The results of these reanalyses confirmed and expanded those of the original investigators. This intensive independent reanalysis effort was occasioned both by the importance of the original findings as well as concerns that the underlying individual health effects information has never been made publicly available. The HEI re-examination lends credibility to the original studies but also found unexpected sensitivities concerning (a) which pollutants are most important, (b) the role of education in mediating the association between pollution and mortality, and (c) the

magnitude of the association depending on how spatial correlation was handled. Further confirmation and extension of the overall findings using more recent air quality and ACS health information was recently published in the Journal of the American Medical Association (Pope et al., 2002). In general, the risk estimates based on the long-term mortality studies are substantially greater than those derived from short-term studies.

In developing and improving the methods for estimating and valuing the potential reductions in mortality risk over the years, EPA has consulted with a panel of the Science Advisory Board (SAB). That panel recommended use of long-term prospective cohort studies in estimating mortality risk reduction (EPA-SAB-COUNCIL-ADV-99-005, 1999). This recommendation has been confirmed by a recent report from the National Research Council, which stated that "it is essential to use the cohort studies in benefits analysis to capture all important effects from air pollution exposure (NRC, 2002, p. 108)." More specifically, the SAB recommended emphasis on Pope, et al. (1995) because it includes a much larger sample size and longer exposure interval, and covers more locations (e.g. 50 cities compared to 6 cities examined in the Harvard data) than other studies of its kind. As explained in the RIA for the Heavy-Duty Engine/Diesel Fuel rule (U.S. EPA, 2000b), more recent EPA benefits analyses have relied on an improved specification from this data set that was developed in the HEI reanalysis of this study (Krewski et al., 2000). The particular specification estimated a C-R function based on changes in mean levels of $PM_{2.5}$, as opposed to the function in the original study, which used median levels. This specification also includes a broader geographic scope than the original study (63 cities versus 50). Specifically, the relative risk from which the Base estimate is derived is 1.12 per 24.5 $\mu g/m^3$ for all-cause mortality (Krewski, et al. 2000, Part II, page 173, Table 31). The SAB has recently agreed with EPA's selection of this specification for use in analyzing mortality benefits of PM reductions (EPA-SAB-COUNCIL-ADV-01-004, 2001).

Alternative Estimate

To reflect concerns about the inherent limitations in the number of studies supporting a causal association between long-term exposure and mortality, an Alternative benefit estimate was derived from the large number of time-series studies that have established a likely causal relationship between short-term measures of PM and daily mortality statistics. A particular strength of such studies is the fact that potential confounding variables such as socio-economic status, occupation, and smoking do not vary on a day-to-day basis in an individual area. A number of multi-city and other types of studies strongly suggest that these effects-relationships cannot be explained by weather, statistical approaches, or other pollutants. The risk estimates from the vast majority of the short-term studies include the effects of only one or two-day exposure to air pollution. More recently, several studies have found that the practice of examining the effects on a single day basis may significantly understate the risk of short-term exposures (Schwartz, 2000; Zanobetti et al., 2002). These studies suggest that the short-term risk can double when the single-day effects are combined with the cumulative impact of exposures over multiple days to weeks prior to a mortality event.

The fact that the PM-mortality coefficients from the cohort studies are far larger than the coefficients derived from the daily time-series studies provides some evidence for an independent chronic effect of PM pollution on health. Indeed, the Base Estimate presumes that

the larger coefficients represent a more complete accounting of mortality effects, including both the cumulative total of short-term mortality as well as an additional chronic effect. This is, however, not the only possible interpretation of the disparity. Various reviewers have argued that 1) the long-term estimates may be biased high and/or 2) the short-term estimates may be biased low. In this view, the two study types could be measuring the same underlying relationship.

Reviewers have noted some possible sources of upward bias in the long-term studies. Some have noted that the less robust estimates based on the Six-Cities Study are significantly higher than those based on the more broadly distributed ACS data sets. Some reviewers have also noted that the observed mortality associations from the 1980's and 90's may reflect higher pollution exposures from the 1950's to 1960's. While this would bias estimates based on more recent pollution levels upwards, it also would imply a truly long-term chronic effect of pollution.

With regard to possible sources of downward bias, it is of note that the recent studies suggest that the single day time series studies may understate the short-term effect on the order of a factor of two. These considerations provide a basis for considering an Alternative Estimate using the most recent estimates from the wealth of time-series studies, in addition to one based on the long-term cohort studies.

In essence, the Alternative Estimate addresses the above noted uncertainties about the relationship between premature mortality and long-term exposures to ambient levels of fine particles by assuming that there is no mortality effect of chronic exposures to fine particles. Instead, it assumes that the full impact of fine particles on premature mortality can be captured using a concentration-response function relating daily mortality to short-term fine particle levels. Specifically, a concentration-response function based on Schwartz et al. (1996) is employed, with an adjustment to account for recent evidence that daily mortality is associated with particle levels from a number of previous days (Schwartz, 2000). Previous daily mortality studies (Schwartz et al., 1996) examined the impact of $PM_{2.5}$ on mortality on a single day or over the average of two or more days. Recent analyses have found that impacts of elevated $PM_{2.5}$ on a given day can elevate mortality on a number of following days (Schwartz, 2000; Samet et al., 2000). Multi-day models are often referred to as "distributed lag" models because they assume that mortality following a PM event will be distributed over a number of days following or "lagging" the PM event.[11]

There are no $PM_{2.5}$ daily mortality studies which report numeric estimates of relative risks from distributed lag models; only PM_{10} studies are available. Daily mortality C-R functions for PM_{10} are consistently lower in magnitude than $PM_{2.5}$-mortality C-R functions because fine particles are believed to be more closely associated with mortality than the coarse fraction of PM. Given that the emissions reductions under the Clear Skies Act result primarily in reduced ambient concentrations of $PM_{2.5}$, use of a PM_{10} based C-R function results in a significant downward bias in the estimated reductions in mortality. To account for the full potential multi-day mortality impact of acute $PM_{2.5}$ events, we use the distributed lag model for PM_{10} reported in Schwartz (2000) to develop an adjustment factor which we then apply to the

$PM_{2.5}$ based C-R function reported in Schwartz et al. (1996).

If most of the increase in mortality is expected to be associated with the fine fraction of PM_{10}, then it is reasonable to assume that the same proportional increase in risk would be observed if a distributed lag model were applied to the $PM_{2.5}$ data. The distributed lag adjustment factor is constructed as the ratio of the estimated coefficient from the unconstrained distributed lag model to the estimated coefficient from the single-lag model reported in Schwartz (2000). The unconstrained distributed lag model coefficient estimate is 0.0012818 and the single-lag model coefficient estimate is 0.0006479. The ratio of these estimates is 1.9784. This adjustment factor is then multiplied by the estimated coefficients from the Schwartz et al. (1996) study. There are two relevant coefficients from the Schwartz et al. (1996) study, one corresponding to all-cause mortality, and one corresponding to chronic obstructive pulmonary disease (COPD) mortality (separation by cause is necessary to implement the life years lost approach detailed below). The adjusted estimates for these two C-R functions are:

All cause mortality = 0.001489 * 1.9784 = 0.002946

COPD mortality = 0.003246 * 1.9784 = 0.006422

Note that these estimates, while approximating the full impact of daily pollution levels on daily death counts, do not capture any impacts of long-term exposure to air pollution. As discussed earlier, EPA's Science Advisory Board, while acknowledging the uncertainties in estimation of a PM-mortality relationship, has repeatedly recommended the use of a study that does reflect the impacts of long-term exposure. The omission of long-term impacts accounts for approximately 40 percent reduction in the estimate of avoided premature mortality in the Alternative Estimate relative to the Base Estimate.

Chronic Bronchitis

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

Nonfatal Myocardial Infarctions (Heart Attacks)

Non-fatal heart attacks have been linked with short term exposures to $PM_{2.5}$ in the U.S. (Peters et al. 2001) and other countries (Poloniecki et al. 1997). We use a recent study by Peters et al. (2001) as the basis for the C-R function estimating the relationship between $PM_{2.5}$ and non-fatal heart attacks. Peters et al. is the only available U.S. study to provide a specific estimate for heart attacks. Other studies, such as Samet et al. (2000) and Moolgavkar et al. (2000b), show a

consistent relationship between all cardiovascular hospital admissions, including for non-fatal heart attacks, and PM. Given the lasting impact of a heart attack on longer-term health costs and earnings, we choose to provide a separate estimate for non-fatal heart attacks based on the single available U.S. C-R function. The finding of a specific impact on heart attacks is consistent with hospital admission and other studies showing relationships between fine particles and cardiovascular effects both within and outside of the U.S. These studies provide a weight of evidence for this type of effect. Several epidemiological studies (Liao et al, 1999; Gold et al, 2000; Magari et al, 2001) have shown that heart rate variability (an indicator of how much the heart is able to speed up or slow down in response to momentary stresses) is negatively related to PM levels. Heart rate variability is a risk factor for heart attacks and other coronary heart diseases (Carthenon et al, 2002; Dekker et al, 2000; Liao et al, 1997, Tsuji et al. 1996). As such, significant impacts of PM on heart rate variability are consistent with an increased risk of heart attacks.

2.4.6. Visibility Improvements

One of the direct consequences of the reductions in fine particles that accompany implementation of the SO_2 and NO_x emissions caps is an improvement in atmospheric clarity and visibility. Changes in the emissions of SO_2 and NO_x caused by the Clear Skies Act will change the level of visibility in much of the U.S by reducing concentrations of sulfate and nitrate particles. Fine particles absorb and scatter light, impairing visibility. Visibility directly affects people's enjoyment of a variety of daily activities both in the places they live and work and in the places they travel to for recreation. The Clean Air Act recognizes visibility as an important public good in naming visibility as one of the aspects of public welfare to be protected in setting secondary NAAQS. In Sections 165 and 169, the Act places particular value on protecting visibility in 156 national parks and wilderness areas (e.g. Shenandoah, Acadia, and Grand Canyon) that are termed class I Federal areas. As noted above, the REMSAD modeling estimates regional and national visibility improvements associated with Clear Skies. As discussed in a subsequent section, this analysis also provides partial estimates of the potential economic value of these visibility improvements.

A number of related measures can be used to measure changes in visibility associated with reduced fine particle concentrations. A key such measure is light "extinction," a measure of the amount of light scattered and absorbed by particles suspended in air. This light scattering and absorption reduces atmospheric clarity and is perceived as haze. Changes in fine particulate mass components are used directly to estimate changes in extinction. Decreasing extinction (in units of inverse distance) can in turn be used to estimate quantitative measures more directly related to human perception such as contrast of distant targets and visual range. More recently, Sisler (1996) created a unitless measure of visibility based directly on the degree of measured light absorption called the *deciview*. Deciviews, like the analogous term decibel, employ a logarithmic scale to evaluate relative changes in visibility that is more directly related to human perception. Sisler characterized a change in light extinction of one deciview as "a small but perceptible scenic change under many circumstances." For this analysis, REMSAD version 7.06 was used to predict the change in visibility, measured in deciviews and presented graphically, of the areas affected by the Clear Skies Act.

2.5. Economic Valuation of Benefits

The overall approach applied in our estimates of the benefits of the Clear Skies Act closely parallels that used in prior EPA analyses, including the Section 812 series of Reports to Congress (U.S. EPA, 1997 and 1999a), the Heavy-Duty Engine/Diesel Fuel RIA (U.S. EPA, 2000b) and the recent Nonroad Diesel Engines Draft RIA (U.S. EPA 2003a). As in those analyses, the EPA has not conducted extensive new primary research to measure economic benefits for individual rulemakings. As a result, our estimates are based on the best available methods of benefits transfer. Benefits transfer is the science and art of adapting primary benefits research from similar contexts to obtain the most accurate measure of benefits for the environmental quality change under analysis. Where appropriate, we have made adjustments to existing primary research for the level of environmental quality change, the sociodemographic and economic characteristics of the affected population, and other factors in order to improve the accuracy and robustness of benefits estimates.

2.5.1. Valuation of Health Effects

Reductions in ambient concentrations of air pollution generally lower the risk of future adverse health affects by a fairly small amount for a large population. The appropriate economic measure is therefore willingness-to-pay (WTP) for changes in risk prior to the regulation (Freeman, 1993). For some health effects, such as hospital admissions, WTP estimates are generally not available. In these cases, we use the cost of treating or mitigating the effect as a primary estimate. These costs of illness (COI) estimates generally understate the true value of reductions in risk of a health effect, reflecting the direct expenditures related to treatment but not the value of avoided pain and suffering from the health effect (Harrington and Portney, 1987; Berger, 1987). Unit values for health endpoints are provided in Table 8. To be consistent with estimates of the costs of Clear Skies and with the analysis of the Clear Skies Act of 2002, all values are in constant year 1999 dollars.

For two endpoints, premature mortality and chronic bronchitis, we provide both a Base valuation estimate, reflecting the best available scientific literature and methods, and an Alternative Estimate, reflecting different assumptions about the value of reducing risks of premature death and chronic bronchitis. Following the advice of the Environmental Economics Advisory Committee of the Science Advisory Board, the Base Estimate uses the "value of a statistical life saved" (VSL) approach in calculating the primary estimate of mortality benefits because we believe this calculation to provide the most reasonable single estimate of an individual's willingness to trade off money for reductions in mortality risk (EPA-SAB-EEAC-00-013). The mean value of avoiding one statistical death (the VSL) is estimated to be $6.1 million in constant 1999 dollars. This represents an intermediate value from a variety of estimates that appear in the economics literature, and it is a value EPA has frequently used in RIAs for other rules and in the Section 812 Reports to Congress.

The Alternative Estimate reflects the impact of changes to key assumptions associated with the valuation of mortality. These include: 1) an alternative interpretation of the literature on monetary valuation of VSL, 2) the use of a value of a statistical life years rather than a VSL

approach, and 3) the degree of prematurity (number of statistical life years lost) for mortalities from air pollution.

Table 8
Unit Values for Economic Valuation of Health Endpoints
(1999$)

Health Endpoint	Description	Mean Estimate
Mortality	Base Estimate – VSL based on 26 studies	$6.1 million per statistical life
	Alternative Estimates - VSLY	Age: Under 65 — at 3% $172,000, at 7% $286,000; 65 and Over — at 3% $434,000, at 7% $527,000
Chronic Bronchitis	Base Estimate – WTP	$329,409 per case
	Alternative Estimate – COI	Age: 27-44 — at 3% $144,654, at 7% $82,661 per case; 45-64 — at 3% $93,792, at 7% $69,435 per case; 65+ — at 3% $10,654, at 7% $8,677 per case
Non-Fatal Heart Attacks	COI	Age: 18-24 — at 3% $63,325, at 7% $62,739 per case; 25-44 — at 3% $71,755, at 7% $70,288 per case; 45-54 — at 3% $75,751, at 7% $73,865 per case; 55-64 — at 3% $135,148, at 7% $127,043 per case; 65+ — at 3% $63,325, at 7% $62,739 per case
Hospital Admissions	all respiratory, ages 65+	$17,635 per admission
	all respiratory, ages 0-2	$7,438 per admission
	pneumonia, ages 65+	$17,106 per admission
	COPD, ages 65+	$13,083 per admission
	COPD , ages 20-64	$11,333 per admission
	asthma, ages < 65	$7,467 per admission
	all cardiovascular ages 65+	$20,334 per admission
	all cardiovascular, ages 20-64	$21,864 per admission
	ischemic heart disease, ages 65+	$24,837 per admission
	dysrhythmias, ages 65+	$15,084 per admission
	congestive heart failure, ages 65+	$14,591 per admission
Emergency Room Visits	Asthma-related	$275 per visit
Minor Effects	Acute bronchitis	$344 per case
	Lower resp. Symptoms	$15.06 per symptom-day
	Upper resp. Symptoms	$23.84 per symptom-day
	Minor restricted activity day (MRAD)	$48.91 per day
	School loss days	$72.56 per day
	Work loss days	County-specific median daily wage
	Worker productivity	Change in daily wages adjusted by regional variations in income

In the sections that follow, we discuss in greater detail the basis for generating WTP for premature mortality risk reductions, reductions in the risk of contracting chronic bronchitis, and reductions in the risk of having a non-fatal heart attack. The mortality, chronic bronchitis, and non-fatal heart attack health endpoints account for over 98 percent of the total estimated

monetized benefits of the Clear Skies Act. In addition, we provide a brief summary of our approach to valuing visibility and agricultural yield improvements. Detailed descriptions of the basis for other economic valuation methods can be found in the Nonroad Diesel Engines Draft RIA (US EPA, 2003a).

Valuation of Premature Mortality

Base Estimate

The monetary benefit of reducing premature mortality risk was estimated using the "value of statistical lives saved" (VSL) approach, although the actual valuation is of small changes in mortality risk experienced by a large number of people. The VSL approach applies information from several published value-of-life studies, which themselves examine tradeoffs of monetary compensation for small additional mortality risks, to determine a reasonable benefit of preventing premature mortality. The mean value of avoiding one statistical death (i.e., the statistical incidence of a single death, equivalent to a product of a population risk times a population size that equals one) is estimated to be $6.1 million in 1999 dollars. This represents an intermediate value from a range of estimates that appear in the economics literature, and it is a value the EPA uses in rulemaking support analyses and in the Section 812 Reports to Congress.

This estimate is the mean of a distribution fitted to the estimates from 26 value-of-life studies identified in the Section 812 reports as "applicable to policy analysis." The approach and set of selected studies mirrors that of Viscusi (1992) (with the addition of two studies), and uses the same criteria as Viscusi in his review of value-of-life studies. The $6.1 million estimate is consistent with Viscusi's conclusion (updated to 1999$) that "most of the reasonable estimates of the value of life are clustered in the $3.7 to $8.6 million range." The $6.1 million estimate is also consistent with the results of a more recent meta-analytic effort by Viscusi and Aldy (2003) that focused on wage-risk studies. The meta-analysis includes both U.S. and international studies, and the results indicate a mean VSL of $5.0 ($4.8) to $6.2 ($6.0) million for the full sample, and $5.5 ($5.3) to $7.6 ($7.4) million for the U.S. sample (estimates in year 2000$ (1999$)). 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, controlling for other job and employee characteristics such as education and experience. As indicated in the previous section on quantification of premature mortality benefits, we assume for this analysis that some of the incidences of premature mortality related to PM exposures occur in a distributed fashion over the five years following exposure. To take this into account in the valuation of reductions in premature mortality, we apply an annual three percent discount rate to the value of premature mortality occurring in future years.[12]

The economics literature concerning the appropriate method for valuing reductions in premature mortality risk is still developing. The adoption of a value for the projected reduction in the risk of premature mortality is the subject of continuing discussion within the economic and public policy analysis community. Regardless of the theoretical economic considerations,

distinctions in the monetary value assigned to the lives saved were not drawn, even if populations differed in age, health status, socioeconomic status, gender or other characteristics.

Following the advice of the Environmental Economics Advisory Committee (EEAC) of the SAB, the VSL approach was used to calculate the Base Estimate of mortality benefits (EPA-SAB-EEAC-00-013). While there are several differences between the risk context implicit in labor market studies we use to derive a VSL estimate and the particulate matter air pollution context addressed here, those differences in the affected populations and the nature of the risks imply both upward and downward adjustments. For example, adjusting for age differences between subjects in the economic studies and those affected by air pollution may imply the need to adjust the $6.1 million VSL downward, but the involuntary nature of air pollution-related risks and the lower level of risk-aversion of the manual laborers in the labor market studies may imply the need for upward adjustments. In the absence of a comprehensive and balanced set of adjustment factors, EPA believes it is reasonable to continue to use the $6.1 million value while acknowledging the significant limitations and uncertainties in the available literature.

Some economists emphasize that the value of a statistical life is not a single number relevant for all situations. Indeed, the VSL estimate of $6.1 million (1999 dollars) is itself the central tendency of a number of estimates of the VSL for some rather narrowly defined populations. When there are significant differences between the population affected by a particular health risk and the populations used in the labor market studies, as is the case here, some economists prefer to adjust the VSL estimate to reflect those differences.

There is general agreement that the value to an individual of a reduction in mortality risk can vary based on several factors, including the age of the individual, the type of risk, the level of control the individual has over the risk, the individual's attitudes towards risk, and the health status of the individual. While the empirical basis for adjusting the $6.1 million VSL for many of these factors does not yet exist, a thorough discussion of these uncertainties is included in EPA's *Guidelines for Preparing Economic Analyses* (U.S. EPA, 2000a). The EPA recognizes the need for investigation by the scientific community to develop additional empirical support for adjustments to VSL for the factors mentioned above.

As further support for the Base Estimate, the SAB-EEAC advised in their recent report that the EPA "continue to use a wage-risk-based VSL as its Base Estimate, including appropriate sensitivity analyses to reflect the uncertainty of these estimates," and that "the only risk characteristic for which adjustments to the VSL can be made is the timing of the risk"(EPA-SAB-EEAC-00-013). In developing the Base Estimate of the benefits of premature mortality reductions, we have discounted over the lag period between exposure and premature mortality. However, in accordance with the SAB advice, we use the VSL in the Base Estimate.

It is currently unknown whether there is a delay between changes in chronic PM exposures and changes in mortality rates. The existence of such a time lag is important for the valuation of premature mortality incidences as economic theory suggests benefits occurring in the future should be discounted relative to benefits occurring today. Although there is no specific scientific evidence of a PM effects lag, current scientific literature on adverse health effects associated with smoking and the difference in the effect size between chronic exposure

studies and daily mortality studies suggest that all incidences of premature mortality reduction associated with a given incremental change in PM exposure would not occur in the same year as the exposure reduction. This literature implies that lags of a few years or longer are plausible. For our Base Estimate, we have assumed a five-year distributed lag structure, with 25 percent of premature deaths occurring in the first year, another 25 percent in the second year, and 16.7 percent in each of the remaining three years. To account for the preferences of individuals for current risk reductions relative to future risk reductions, we discount the value of avoided premature mortalities occurring beyond the analytical year (2010 or 2020) using three and seven percent discount rates. No lag adjustment is necessary for the Alternative Estimate, which focuses on premature mortality occurring within a few days of the PM exposure.

Alternative Estimate

The Alternative Estimate reflects the impact of changes to key assumptions associated with the valuation of mortality. These include: 1) the impact of using wage-risk and contingent valuation-based value of statistical life estimates in valuing risk reductions from air pollution as opposed to contingent valuation-based estimates alone, and 2) the degree of prematurity in mortalities from air pollution.

The Alternative Estimate addresses this issue by using an estimate of the value of statistical life that is based only on the set of five contingent valuation studies included in the larger set of 26 studies recommended by Viscusi (1992) as applicable to policy analysis. The mean of the five contingent valuation based VSL estimates is $3.7 million (1999$), which is approximately 60 percent of the mean value of the full set of 26 studies. Note that because these are deaths associated with short-term exposures to $PM_{2.5}$, it is assumed that there is no lag between reduced exposure and reduced risk of mortality. In order to implement the non-constant VSLY approach, we begin by using a VSL of $3.7 million based on five contingent valuation studies, which were also considered as part of the Base Estimate. This smaller VSL is also consistent with an alternative interpretation of the wage-risk literature (Mrozek and Taylor 2002). For persons under age 65, the $3.7 million VSL is assumed to reflect an average loss of 35 years. The VSLY associated with a $3.7 million VSL is $172,000, annualized using a 3 percent discount rate, or $286,000, annualized using a 7 percent discount rate. Note that the larger discount rate increases the VSLY because at a higher discount rate, a larger stream of VSLY is required to yield a VSL of $3.7 million. For those over age 65, the VSLY is derived from a $3.7 million VSL and an assumed 10-year life expectancy. This gives a VSLY of $434,000 at a 3% discount rate and a VSLY of $527,000 at a 7% discount rate.

While the Base Estimate uses a VSL approach, the Alternative Estimate is based on the number of years of life saved and economic value of saving a statistical life year (VSLY). The VSLY approach has been developed in the peer-reviewed economics literature (e.g., Viscusi and Moore, 1988) and has been applied for many years by the U.S. Food and Drug Administration. Some recent analyses, however, have raised concerns about the use of this method to value reductions in premature mortality in an environmental context (Science Advisory Board, 1999; Krupnick et al., 2002). The VSLY approach applied in this analysis recognizes that each year late in the life span may have a higher monetary value than the average life year saved in the middle of the life span. The non-constant VSLY, rising later in the lifespan, is qualitatively

compatible with theoretical economic models of an individual's demand for lifesaving as a function of age (Sheppard and Zeckhauser, 1984). The conceptional rationale for a premium on VSLY among the elderly is that they have saved through their working lifetimes and accumulated assets that can be devoted to health protection, and have rising baseline risks, which increase the marginal value of risk reductions. (Pratt and Zeckhauser 1996).

Under the alternative approach, the value of a life year for younger individuals is calculated as if they had an average life expectancy. However, instead of attempting to estimate the remaining life expectancy for different age groups, we have assumed that everyone who dies from exposure to air pollution loses five years of life. Because we assume that younger individuals do not have the accumulated assets or do not adjust the value of life years to reflect reductions in life expectancy, this approach implies that the total value of a five-year loss in life years is greater for the elderly than for younger individuals. An additional limitation of this approach is the discontinuity at age 65. A more complex approach would produce a continuous VSLY curve; however, the empirical data required to specify these models are not available.

There is no latency period (or lag) assumed in the alternative analysis since the premature deaths are assumed to occur primarily among persons with chronic disease who experience short-term elevations in daily air pollution levels. Even the latency periods associated with the distributed lag models are too short to be of significance in the valuation process.

The second issue is addressed by assuming that deaths from chronic obstructive pulmonary disease (COPD) are advanced by 6 months, and deaths from all other causes are advanced by 5 years. These reductions in life years lost are applied regardless of the age at death. Actuarial evidence suggests that individuals with serious preexisting cardiovascular conditions have a remaining life expectancy of around 5 years. While many deaths from daily exposure to PM may occur in individuals with cardiovascular disease, studies have shown relationships between all cause mortality and PM, and between PM and mortality from pneumonia (Schwartz, 2000). In addition, recent studies have shown a relationship between PM and non-fatal heart attacks, which suggests that some of the deaths due to PM may be due to fatal heart attacks (Peters et al., 2001). And, a recent meta-analysis has shown little effect of age on the relative risk from PM exposure (Stieb et al. 2002), which suggests that the number of deaths in non-elderly populations (and thus the potential for greater loss of life years) may be significant. Indeed, this analysis estimates that 21 percent of non-COPD premature deaths avoided are in populations under 65. Thus, while the assumption of 5 years of life lost may be appropriate for a subset of total avoided premature mortalities, it may over or underestimate the degree of life shortening attributable to PM for the remaining deaths.

Valuation of Avoided Cases of Chronic Bronchitis

Base Estimate

The best available estimate of WTP to avoid a case of chronic bronchitis (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 can be found in the Heavy-Duty Engine/Diesel Fuel RIA and its supporting documentation, and in the benefits technical support document (TSD) for the Nonroad Diesel Engines Draft RIA (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: (1) the WTP to avoid a case of severe CB, as described by Viscusi, et al.; (2) the severity level of an average pollution-related case of CB (relative to that of the case described by Viscusi, et al.); and (3) the elasticity of WTP with respect to severity of the illness. 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 $329,409 (1999$), is taken as the central tendency estimate of WTP to avoid a PM-related case of CB.

Alternative Estimate

For the Alternative Estimate, a cost-of illness value is used in place of willingness-to-pay to reflect uncertainty about the value of reductions in incidences of chronic bronchitis. In the Base Estimate, the willingness-to-pay estimate was derived from two contingent valuation studies (Viscusi et al., 1991; Krupnick and Cropper, 1992). These studies were experimental studies intended to examine new methodologies for eliciting values for morbidity endpoints. Although these studies were not specifically designed for policy analysis, the SAB (EPA-SAB-COUNCIL-ADV-00-002, 1999) has indicated that the severity-adjusted values from these studies provide reasonable estimates of the WTP for avoidance of chronic bronchitis. As with other contingent valuation studies, the reliability of the WTP estimates depends on the methods used to obtain the WTP values. In order to investigate the impact of using the CV based WTP estimates, the Alternative Estimate relies on estimates of lost earnings and medical costs. Using age-specific annual lost earnings and medical costs estimated by Cropper and Krupnick (1990) and a three percent discount rate, we estimated a lifetime present discounted value (in 1999$) due to chronic bronchitis of $145,000 for someone between the ages of 27 and 44; $94,000 for someone between the ages of 45 and 64; and $11,000 for someone over 65. The corresponding age-specific estimates of lifetime present discounted value (in 1999$) using a seven percent discount rate are $83,000, $69,000, and $9,000, respectively. These estimates assumed that 1) lost earnings continue only until age 65, 2) medical expenditures are incurred until death, and 3) life expectancy is unchanged by chronic bronchitis.

Valuation of Reductions in Non-Fatal Myocardial Infarctions (Heart Attacks)

EPA estimated the impact of control programs on reductions in the expected number of non-fatal heart attacks for the first time in the Nonroad Diesel Engines Rule Draft RIA. While other EPA regulatory analyses (including the analysis of the Clear Skies Act of 2002) examined the

impact of reductions in other related cardiovascular endpoints, non-fatal myocardial infarctions (heart attacks) were not valued prior to the proposed Nonroad Diesel Engines Rule.

A suitable WTP value for reductions in the risk of non-fatal heart attacks has not been identified. Instead, a cost-of-illness unit value with two components - the direct medical costs and the opportunity cost (lost earnings) associated with the illness event – is proposed. Because the costs associated with a heart attack extend beyond the initial event itself, we consider costs incurred over several years. Using age-specific annual lost earnings estimated by Cropper and Krupnick (1990) and a three percent discount rate, a present discounted value in lost earnings (in 2000$) over 5 years due to a heart attack was estimated. This value is $8,800 for someone between the ages of 25 and 44, $13,000 for someone between the ages of 45 and 54, and $75,000 for someone between the ages of 55 and 65. The corresponding age-specific estimates of lost earnings (in 2000$) using a seven percent discount rate are $7,900, $12,000, and $67,000, respectively.

We have found three possible sources in the literature of estimates of the direct medical costs of nonfatal heart attacks, each of which provided significantly different values, are presented in Table 9.

Table 9

Alternative Direct Medical Cost of Illness Estimates for Nonfatal Heart Attacks

Study	Direct Medical Costs (2000$)	Over an x-year period, for x =
Wittels et al., 1990	$109,474*	5
Russell et al., 1998	$22,331**	5
Eisenstein et al., 2001	$49,651**	10
Russell et al., 1998	$27,242**	10

*Wittels et al. did not appear to discount costs incurred in future years.
**Using a 3 percent discount rate.

As noted above, the estimates from these three studies are substantially different, and we have not adequately resolved the sources of differences in the estimates. Because the wage-related opportunity cost estimates from Cropper and Krupnick, 1990, cover a 5-year period, this analysis uses estimates for medical costs that similarly cover a 5-year period – i.e., estimates from Wittels et al., 1990, and Russell et al., 1998. The analysis uses a simple average of the two 5-year estimates, or $65,902, and add it to the 5-year opportunity cost estimate. The resulting estimates are given in Table 10a.

Table 10a

Estimated Costs Over a 5-Year Period (in 2000$) of a Non-Fatal Myocardial Infarction

Age Group	Opportunity Cost	Medical Cost**	Total Cost
18 - 24	$0	$65,902	$65,902
25-44	$8,774*	$65,902	$74,676
45 - 54	$12,253*	$65,902	$78,834
55 - 65	$70,069*	$65,902	$140,649

Table 10a Estimated Costs Over a 5-Year Period (in 2000$) of a Non-Fatal Myocardial Infarction			
Age Group	Opportunity Cost	Medical Cost**	Total Cost
> 65	$0	$65,902	$65,902

*From Cropper and Krupnick, 1990, using a 3% discount rate.
**An average of the 5-year costs estimated by Wittels et al., 1990, and Russell et al., 1998.

Table 10b presents the estimated unit cost per heart attacks in 1999 dollars, which are used in this analysis.

Table 10b Estimated Costs Over a 5-Year Period (in 1999$) of a Non-Fatal Myocardial Infarction			
Age Group	Opportunity Cost	Medical Cost	Total Cost
18 - 24	$0	$63,325	$63,325
25-44	$8,430	$63,325	$71,755
45 - 54	$11,774	$63,325	$75,751
55 - 65	$67,857	$63,325	$135,148
> 65	$0	$63,325	$63,325

2.5.2. Valuation of Changes in Visibility

Changes in the level of ambient particulate matter caused by the reduction in emissions from the Clear Skies Act would change the level of visibility in much of the U.S. Visibility directly affects people's enjoyment of a variety of daily activities. Individuals value visibility both in the places they live and work, in the places they travel to for recreational purposes, and at sites of unique public value, such as the Grand Canyon. This section discusses the measurement of the economic benefits of visibility.

It is difficult to quantitatively define a visibility endpoint that can be used for valuation. Increases in PM concentrations cause increases in light extinction. Light extinction is a measure of how much the components of the atmosphere absorb light. More light absorption means that the clarity of visual images and visual range is reduced, *ceteris paribus.* Light absorption is a variable that can be accurately measured. Sisler (1996) created a unitless measure of visibility based directly on the degree of measured light absorption called the *deciview*. Deciviews are standardized for a reference distance in such a way that one deciview corresponds to a change of about 10 percent in available light. Sisler characterized a change in light extinction of one deciview as "a small but perceptible scenic change under many circumstances." Air quality models were used to predict the change in visibility, measured in deciviews, of the areas affected by the preliminary control options.[13]

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

Residential visibility benefits are those that occur from visibility changes in urban, suburban, and rural areas, and also in recreational areas not listed as federal Class I areas.[14] For the purposes of this analysis, recreational visibility improvements are defined as those that occur specifically in federal Class I areas. A key distinction between recreational and residential benefits is that only those people living in residential areas are assumed to receive benefits from residential visibility, while all households in the U.S. are assumed to derive some benefit from improvements in Class I areas. Values are assumed to be higher if the Class I area is located close to their home.[159]

Only two existing studies provide defensible monetary estimates of the value of visibility changes. One is a study on residential visibility conducted in 1990 (McClelland, et. al., 1993) and the other is a 1988 survey on recreational visibility value (Chestnut and Rowe, 1990a; 1990b). Both utilize the contingent valuation method. There has been a great deal of controversy and significant development of both theoretical and empirical knowledge about how to conduct CV surveys in the past decade. In EPA's judgment, the Chestnut and Rowe study contains many of the elements of a valid CV study and is sufficiently reliable to serve as the basis for monetary estimates of the benefits of visibility changes in recreational areas.[16] This study serves as an essential input to our estimates of the benefits of recreational visibility improvements in the primary benefits estimates. Consistent with SAB advice, EPA has designated the McClelland, et al. study as significantly less reliable for regulatory benefit-cost analysis, although it does provide useful estimates on the order of magnitude of residential visibility benefits (EPA-SAB-COUNCIL-ADV-00-002, 1999).

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

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

[16] An SAB advisory letter indicates that"many members of the Council believe that the Chestnut and Rowe study is the best available." (EPA-SAB-COUNCIL-ADV-00-002, 1999) However, the committee did not formally approve use of these estimates because of concerns about the peer-reviewed status of the study. EPA believes the study has received adequate review and has been cited in numerous peer-reviewed publications (Chestnut and Dennis, 1997).
[17] The total value for these crops in 1998 was $47 billion.

analysis. The survey data collected were used to estimate a WTP equation for improved visibility. In addition to the visibility change variable, the estimating equation also included household income as an explanatory variable.

The Chestnut and Rowe study did not measure values for visibility improvement in Class I areas outside the three regions. Their study covered 86 of the 156 Class I areas in the U.S. We can infer the value of visibility changes in the other Class I areas by transferring values of visibility changes at Class I areas in the study regions. A complete description of the benefits transfer method used to infer values for visibility changes in Class I areas outside the study regions is provided in the benefits TSD for the Nonroad Diesel Engines Draft RIA (Abt Associates, 2003).

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

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

2.5.3. Valuation of Agricultural Benefits

EPA's Ozone Criteria Document notes that "ozone affects vegetation throughout the United States, impairing crops, native vegetation, and ecosystems more than any other air pollutant" (US EPA, 1996a). Reduced levels of ground-level ozone resulting from the Clear Skies Act would have generally beneficial results on agricultural crop yields and commercial forest growth. Well-developed techniques exist to provide monetary estimates of these benefits to agricultural producers and consumers. These techniques use models of planting decisions, yield response functions, and agricultural product supply and demand. The resulting welfare measures are based on predicted changes in market prices and production costs.

Laboratory and field experiments have shown reductions in yields for agronomic crops exposed to ozone, including vegetables (e.g., lettuce) and field crops (e.g., cotton and wheat).

The most extensive field experiments, conducted under the National Crop Loss Assessment Network (NCLAN), examined 15 species and numerous cultivars. The NCLAN results show that "several economically important crop species are sensitive to ozone levels typical of those found in the U.S." (US EPA, 1996a). In addition, economic studies have shown a relationship between observed ozone levels and crop yields (Garcia, et al., 1986).

To estimate changes in crop yields, we used biological exposure-response information derived from controlled experiments conducted by the NCLAN (NCLAN, 1996). For the purpose of our analysis, we analyze changes for the six most economically significant crops for which C-R functions are available: corn, cotton, peanuts, sorghum, soybean, and winter wheat.[17] For some crops there are multiple C-R functions, some more sensitive to ozone and some less. Our estimate assumes that crops are evenly mixed between relatively sensitive and relatively insensitive varieties.

We analyzed the economic value associated with varying levels of yield loss for ozone-sensitive commodity crops using the AGSIM© agricultural benefits model (Taylor, et al., 1993). AGSIM© is an econometric-simulation model that is based on a large set of statistically estimated demand and supply equations for agricultural commodities produced in the United States. The model is capable of analyzing the effects of changes in policies that affect commodity crop yields or production costs.[18]

The measure of benefits calculated by the model is the net change in consumer and producer surplus from baseline ozone concentrations to the ozone concentrations resulting from attainment of particular standards. Using the baseline and post-control equilibria, the model calculates the change in net consumer and producer surplus on a crop-by-crop basis.[19] Dollar values are aggregated across crops for each standard. The total dollar value represents a measure of the change in social welfare associated with implementation of the Clear Skies Act.

2.6. Adjustments for Changes in Income Over Time

Our analysis accounts for expected growth in real income over time. Economic theory argues that WTP for most goods (such as environmental protection) will increase if real incomes increase. The economics literature suggests that the severity of a health effect is a primary determinant of the strength of the relationship between changes in real income and WTP (Alberini, 1997; Miller, 2000; Evans and Viscusi, 1993). As such, we use different factors to adjust the WTP for minor health effects, severe and chronic health effects, and premature mortality. We also adjust WTP for improvements in recreational visibility.

Recent SAB deliberations on mortality and morbidity valuation approaches suggest that some adjustments to unit values are appropriate to reflect economic theory (EPA-SAB-EEAC-00-013, 2000). As noted above, we apply one adjustment by discounting lagged mortality incidence effects. A second adjustment is conducted as part of the mortality, morbidity, and

[19] Agricultural benefits differ from other health and welfare endpoints in the length of the assumed ozone season. For agriculture, the ozone season is assumed to extend from April to September. This assumption is made to ensure proper calculation of the ozone statistic used in the exposure-response functions. The only crop affected by changes in ozone during April is winter wheat.

visibility valuation procedures to incorporate the effect of changes in income over time on WTP. To estimate the effects of changes in income over time we use a procedure originally outlined in Appendix H of the Section 812 Prospective Report to Congress (EPA 1999). That procedure uses per capita income estimates generated from federal government projections of income and population growth, and applies three different income elasticities for mortality, severe morbidity, and light symptom effects.[20]

Benefits for each of the categories - minor health effects, severe and chronic health effects (which include chronic bronchitis, non-fatal heart attacks, and premature mortality), and visibility - were adjusted by multiplying the unadjusted benefits by the appropriate adjustment factor, listed in Table 11 below.

Table 11

Adjustment Factors Used to Account for Projected Real Income Growth through 2010 and 2020

Benefit Category	Adjustment Factor (2010)	Adjustment Factor (2020)
Minor Health Effect	1.034	1.084
Severe and Chronic Health Effects	1.113	1.299
Premature Mortality	1.100	1.262
Visibility	1.239	1.704

The procedure used to develop these adjustment factors is described in more detail in the Nonroad Diesel Engines Draft RIA (U.S. EPA, 2003a). Also note that no adjustments were made to benefits based on the cost-of-illness approach or to work loss days. These assumptions will also lead us to underestimate benefits since it is likely that increases in real U.S. income would also result in increased cost-of-illness (due, for example, to increases in wages paid to medical workers) and increased cost of work loss days (reflecting that if worker incomes are higher, the losses resulting from reduced worker production would also be higher). The result of applying these adjustment factors is an updated set of unit economic values used in the valuation step. We summarize these adjusted values in Table 12.

Table 12 provides a summary of the Base WTP values used to generate estimates of the economic value of avoided health effects for this analysis, adjusted to 1999 dollars, and a brief description of the basis for these values. Table 12 also provides a summary of the monetary values for the Alternative Estimate used for economic valuation of mortality and chronic bronchitis. For these two endpoints, the Alternative Estimate valuation differs from the Base Estimate values.

Table 12
Effective Unit Health Effects Valuation for The Clear Skies Act (1999 dollars), Incorporating Adjustments for Income Growth

Endpoint	Valuation per event (2010 mean est.)	Valuation per event (2020 mean est.)
Mortality		

Base Estimate: (VSL)	at 3%	at 7%	at 3%	at 7%
Chronic exposure	$6.4 million	$6.0 million	$7.3 million	$6.9 million
Alternative Estimate: (VSLY)	at 3%	at 7%	at 3%	at 7%
Short-Term Exposure, Under 65	$189,000	$315,000	$217,000	$360,000
Short-Term Exposure, 65 and Over	$477,000	$580,000	$547,000	$664,000
Chronic Illness				
Chronic Bronchitis (WTP, Base Estimate)	$366,600		$428,035	
	at 3%	at 7%	at 3%	at 7%
Chronic Bronchitis (COI, Alternative Estimate), 27-44	$160,986	$91,994	$187,964	$107,410
Chronic Bronchitis (COI, Alternative Estimate), 45-64	$104,382	$77,274	$121,874	$90,224
Chronic Bronchitis (COI, Alternative Estimate), 65 +	$11,857	$9,656	$13,844	$11,275
Non-Fatal Heart Attacks				
	at 3%	at 7%	at 3%	at 7%
18-24	$63,325	$62,739	$63,325	$62,739
25-44	$71,755	$70,288	$71,755	$70,288
45-54	$75,751	$73,865	$75,751	$73,865
55-64	$135,148	$127,043	$135,148	$127,043
65+	$63,325	$62,739	$63,325	$62,739
Hospital Admissions				
all respiratory, ages 65 +	$17,635		$17,635	
all respiratory, ages 0-2	$7,438		$7,438	
COPD, ages 65 +	$13,083		$13,083	
COPD, ages 20 - 64	$11,333		$11,333	
asthma, ages < 65	$7,467		$7,467	
all cardiovascular, ages 65 +	$20,334		$20,334	
all cardiovascular, ages 20 -64	$21,864		$21,864	
ischemic heart disease, ages 65 +	$24,837		$24,837	
dysrhythmias, ages 65 +	$15,084		$15,084	
congestive heart failure, ages 65 +	$14,591		$14,591	
Emergency Room Visits				
Asthma-related	$275		$275	
Minor Health Effects				
Acute Bronchitis	$356.06		$373.49	
Upper Respiratory Symptoms	$24.64		$25.85	
Lower Respiratory Symptoms	$15.57		$16.34	
Minor Restricted Activity Days	$50.56		$53.03	
School Loss Days	$75.01		$78.69	
Work Loss Days	County-specific median daily wage		County-specific median daily wage	
Worker productivity	Changes in daily wages adjusted by regional variations in income		Changes in daily wages adjusted by regional variations in income	

2.7. Updates to the Modeling

The framework for this analysis of the benefits of the Clear Skies Act of 2003 is similar to the framework EPA used to analyze the Clear Skies Act of 2002. This analysis of the Clear Skies Act of 2003, however, uses updated modeling assumptions and refined data to analyze the benefits of Clear Skies. This analysis uses the same health effect and valuation functions employed the Nonroad Diesel Engines Rule Draft RIA (U.S. EPA, 2003a) released in April 2003. In addition, the 2003 modeling of Clear Skies reflects new power plant controls and state and federal regulatory programs as of March 2003, changes to the air quality modeling, updated emissions inventories for all sectors, and updated assumptions and data for projecting emissions from the power sector. This analysis also reflects changes to the legislation in the 2003 version of the legislation.

Specifically, modeling updates since the 2002 Clear Skies analysis include the following:

Demographic/population data:

- Base population data has been updated from 1990 to Census 2000 block level data
- Future year population projections have been developed based on Woods and Poole Economics, Inc. 2001 Regional Projections of county population.

Health effects incidence/prevalence data:

- County-level mortality rates (all-cause, non-accidental, cardiopulmonary, lung cancer, COPD) have been updated from 1994-1996 to 1996-1998 using the CDC Wonder database.
- Hospitalization rates have been updated from 1994 to 1999 and switched from national rates to regional rates using 1999 National Hospital Discharge Survey results.
- Regional emergency room visit rates have been developed using results of the 2000 National Hospital Ambulatory Medical Care Survey.
- Prevalence of asthma and chronic bronchitis have been updated to 1999 using results of the National Health Interview Survey (HIS), as reported by the American Lung Association (ALA, 2002a and 2002 b)
- Non-fatal heart attack incidence rates have been updated based on National Hospital Discharge Survey results.
- The national acute bronchitis incidence rate has been updated using HIS data as reported in ALA, 2002a, Table 11.
- The work loss days rate has been updated using the 1996 HIS data, as reported in Adams, et al. 1999, Table 41
- School absence rates have been developed using data from the National Center for Education Statistics and the 1996 HIS, as reported in Adams, et al., 1999, Table 46.
- Baseline incidence rates for respiratory symptoms in asthmatics have been developed based on epidemiological studies (Ostro et al. 2001; Vedal et al. 1998; Yu et al; 2000; McConnell et al., 1999; Pope et al., 1991).

Concentration-Response Functions

- Several new endpoints have been added to the analysis, including:
    - > hospital admissions for all cardiovascular causes in adults 20-64, PM (Moolgavkar et al., 2000b);
    - > ER visits for asthma in children 0-18, PM (Norris et al., 1999);
    - > non-fatal heart attacks, adults over 30, PM (Peters et al, 2001);
    - > school loss days, ozone (Gilliland et al, 2001; Chen et al, 2000); and
    - > hospital admissions for all respiratory causes in children under 2, ozone (Burnett et al., 2001)
- The sources for concentration-response functions for hospital admission for pneumonia, COPD, and total cardiovascular have been changed from Samet et al, 2000 (a PM_{10} study), to Lippmann et al, 2000 and Moolgavkar, 2000b ($PM_{2.5}$ studies)
- A separate table with incidence estimates for the asthmatic subpopulation has been added, based on studies by Ostro et al, 2001; Yu et al, 2000; Vedal et al, 1998; Pope et al., 1991; Ostro et al., 1991; and McConnell et al., 1999.
- A separate table showing age-specific impacts, as well as the impact of extending the population covered by a C-R function to additional ages, has been added (i.e. extending lower respiratory symptoms to all children, rather than to children aged 7-14 only).

Valuation of Changes in Health Outcomes:

- A value for school absence days has been developed by determining the proportion of families with two working families, multiplying that proportion by the number of school loss days, and multiplying the resulting number of school loss days resulting in a parent staying home (or requiring purchase of a caregivers time) by the average daily wage.
- Age-specific values for non-fatal heart attacks have been developed using cost-of-illness methods, based on direct cost estimates reported in Wittels et al (1990) and Russell et al (1998) and lost earnings estimates reported in Cropper and Krupnick (1990). These estimates include expected medical costs in the 5 years following a myocardial infarction, as well as the lost earnings over that period.
- A previous error in the valuation of acute bronchitis episodes has been corrected. Previously, episodes were valued as if they lasted only a single day. We have corrected this value to account for multiday duration of episodes.

Air Quality:

- REMSAD PM results have been used to develop adjustment factors which were applied to ambient monitoring data to estimate future base and control ambient PM levels (consistent with past practice for ozone modeling). This change is due to the recent availability of sufficient ambient $PM_{2.5}$ monitoring data.
- REMSAD, has been updated to version 7.06, run at 36 km grid resolution. This new version fixes an error in the secondary organic aerosol mechanism and to revise certain aspects of the dry deposition code.

Emissions:

- Base and Control emissions files have been updated to reflect projected emissions reductions that would occur under new state and federal programs developed or finalized before March 2003, including:
  - State-specific caps on EGU emissions in Massachusetts, New Hampshire, North Carolina, Texas, and Wisconsin.
  - EPA's proposed Nonroad Diesel Engines Rule.
  - Newly-installed EGU controls.
- Assumptions of the impacts of Clear Skies on the power sector have also been updated. Updated modeling assumptions include:
  - costs and performance of mercury controls;
  - electricity demand growth rate;
  - natural gas prices; existing generation capacity;
  - cost and performance of new conventional units and existing nuclear units;
  - renewable energy programs and portfolio standards;
  - fuel oil assumptions;
  - coal supply curves; and
  - Acid Rain Program emissions allowance bank.
- The 1996 inventory of emissions from all sectors has been updated to a 2001 inventory. This 2001 inventory was projected using the 1996 National Emissions Inventory (NEI), the 2010 projection of that 1996 inventory, and preliminary 2001 NEI inventories for the Electric Generation Unit (EGU), Highway Vehicle, and Nonroad Vehicle sectors.

3. MAJOR UNCERTAINTIES IN THE BENEFITS ANALYSIS

The estimates of avoided health effects, improved visibility, and monetary benefits of the Clear Skies Act are based on a method that reflects peer-reviewed data, models, and approaches that are applied to support EPA rulemakings and generate reports to Congress on the benefits of air pollution regulation. Although EPA has made a concerted effort to apply well-accepted methods, in any complex analysis using estimated parameters and inputs from numerous models, there are likely to be many sources of uncertainty. This analysis is no exception. As outlined in this document, there are many inputs used to derive the final estimate of benefits, including emission inventories, air quality models (with their associated parameters and inputs), epidemiological estimates of C-R functions, estimates of values (both from WTP and cost-of-illness studies), population estimates, income estimates, and estimates of the future state of the world (i.e., regulations, technology, and human behavior). Each of these inputs may be uncertain, and depending on their location in the benefits analysis, may have a disproportionately large impact on final estimates of total benefits. For example, emissions estimates are used in the first stage of the analysis. As such, any uncertainty in emissions estimates will be propagated through the entire analysis. When compounded with uncertainty in later stages, small uncertainties in emission levels can lead to much larger impacts on total benefits. A more thorough discussion of uncertainty can be found in the benefits technical support document (TSD) for the draft Nonroad Diesel Engines RIA (Abt Associates, 2003).

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

- Gaps in scientific data and inquiry;
- Variability in estimated relationships, such as C-R functions, introduced through differences in study design and statistical modeling;
- Errors in measurement and projection for variables such as population growth rates;
- Errors due to misspecification of model structures, including the use of surrogate variables, such as using PM_{10} when $PM_{2.5}$ is not available, excluded variables, and simplification of complex functions; and
- Biases due to omissions or other research limitations.

Some of the key uncertainties in the benefits analysis are presented in Table 13. Given the wide variety of sources for uncertainty and the potentially large degree of uncertainty about any primary estimate, it is necessary for us to address this issue in several ways, based on the following types of uncertainty:

- ***Quantifiable uncertainty in benefits estimates.*** In other analyses, EPA has developed quantitative characterizations of the uncertainty and variability in the estimates developed here. Quantitative uncertainty may include measurement uncertainty or variation in estimates across or within studies. For example, the variation in VSL results across the 26 studies that underlie the Base Estimate represent a quantifiable uncertainty.

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

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

It should be noted that even for individual endpoints, there is usually more than one source of uncertainty. This makes it difficult to provide an overall quantified uncertainty estimate for individual endpoints or for total benefits. For example, the C-R function used to estimate avoided premature mortality has an associated standard error which represents the sampling error around the pollution coefficient in the estimated C-R function. It is possible to report a confidence interval around the estimated incidences of avoided premature mortality based on this standard error. However, this would omit the contribution of air quality changes, baseline population incidences, projected populations exposed, and transferability of the C-R function to diverse locations to uncertainty about premature mortality. Thus, a confidence interval based on the standard error gives a misleading picture about the overall uncertainty in the estimates. Information on the uncertainty surrounding particular C-R and valuation functions is provided in the benefits TSD for the Nonroad Diesel Engines Rule Draft RIA (Abt Associates, 2003). But this information should be interpreted within the context of the larger uncertainty surrounding the entire analysis. The total benefits estimate may understate or overstate actual benefits of the Clear Skies Act.

Our approach to characterizing model uncertainty is to present a primary estimate of the benefits, based on the best available scientific literature and methods, and to then provide sensitivity analyses to illustrate the effects of uncertainty about key analytical assumptions. Our analysis of the preliminary control options has not included formal integrated uncertainty analyses, although we have conducted several sensitivity tests and have analyzed a full Alternative Estimate based on changes to several key model parameters.

The recent National Academies of Sciences (NAS) report on estimating public health benefits of air pollution regulations recommended that EPA begin to move the assessment of uncertainties from its ancillary analyses into its primary analyses by conducting probabilistic, multiple-source uncertainty analyses (NRC, 2002). EPA are working to implement these recommendations and plans to better characterize some of this uncertainty, especially regarding mortality-related benefits in future benefits assessements and future RIAs.

In considering the monetized benefits estimates, the reader should remain aware of the many limitations of conducting these analyses mentioned throughout this document. One significant limitation of both the health and welfare benefits analyses is the inability to quantify many of the serious effects listed in Tables 3 and 4. For many health and welfare effects, such as changes in ecosystem functions and PM-related materials damage, reliable C-R functions and/or valuation functions are not currently available. In general, if it were possible to monetize these benefits categories, the benefits estimates presented in this analysis would increase. Unquantified benefits are qualitatively discussed in this document. In addition to unquantified

benefits, there may also be environmental costs that we are unable to quantify. Several of these environmental cost categories are related to nitrogen deposition, while one category is related to the issue of ultraviolet light. These endpoints are qualitatively discussed in the Nonroad Diesel Engine Rule Draft RIA (US EPA, 2003a). The net effect of excluding benefit and disbenefit categories from the estimate of total benefits depends on the relative magnitude of the effects.

In the remainder of this section, we discuss the major sources of uncertainty related to the estimate of avoided health effects, avoided ecological effects, and monetary valuation of these benefits. Our analysis of the Clear Skies Act has not included formal uncertainty analyses, although we have conducted several sensitivity tests.

Table 13

Primary Sources of Uncertainty in the Benefit Analysis

1. Uncertainties Associated With Concentration-Response Functions
– The value of the ozone- or PM-coefficient in each C-R function. – Application of a single C-R function to pollutant changes and populations in all locations. – Similarity of future year C-R relationships to current C-R relationships. – Correct functional form of each C-R relationship. – Extrapolation of C-R relationships beyond the range of ozone or PM concentrations observed in the study. – Application of C-R relationships only to those subpopulations matching the original study population.
2. Uncertainties Associated With Ozone and PM Concentrations
– Responsiveness of the models to changes in precursor emissions resulting from the control policy. – Projections of future levels of precursor emissions, especially ammonia and crustal materials. – Model chemistry for the formation of ambient nitrate concentrations. – Lack of ozone monitors in rural areas requires extrapolation of observed ozone data from urban to rural areas. – Use of separate air quality models for ozone and PM does not allow for a fully integrated analysis of pollutants and their interactions. – Full ozone season air quality distributions are extrapolated from a limited number of simulation days. – Comparison of model predictions of particulate nitrate with observed rural monitored nitrate levels indicates that REMSAD overpredicts nitrate in some parts of the Eastern US and underpredicts nitrate in parts of the Western US.
3. Uncertainties Associated with PM Mortality Risk
– No scientific literature supporting a direct biological mechanism for observed epidemiological evidence. – Direct causal agents within the complex mixture of PM have not been identified. – The extent to which adverse health effects are associated with low level exposures that occur many times in the year versus peak exposures. – The extent to which effects reported in the long-term exposure studies are associated with historically higher levels of PM rather than the levels occurring during the period of study. – Reliability of the limited ambient $PM_{2.5}$ monitoring data in reflecting actual $PM_{2.5}$ exposures.
4. Uncertainties Associated With Possible Lagged Effects
– The portion of the PM-related long-term exposure mortality effects associated with changes in annual PM levels would occur in a single year is uncertain as well as the portion that might occur in subsequent years.
5. Uncertainties Associated With Baseline Incidence Rates
– Some baseline incidence rates are not location-specific (e.g., those taken from studies) and may therefore not accurately represent the actual location-specific rates. – Current baseline incidence rates may not approximate well baseline incidence rates in 2030. – Projected population and demographics may not represent well future-year population and demographics.
6. Uncertainties Associated With Economic Valuation
– Unit dollar values associated with health and welfare endpoints are only estimates of mean WTP and therefore have uncertainty surrounding them. – Mean WTP (in constant dollars) for each type of risk reduction may differ from current estimates due to differences in income or other factors. – Future markets for agricultural products are uncertain.
7. Uncertainties Associated With Aggregation of Monetized Benefits
– Health and welfare benefits estimates are limited to the available C-R functions. Thus, unquantified or unmonetized benefits are not included.

3.1 Uncertainties Associated with Health Benefit Estimates

3.1.1. Within-Study Variation

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

3.1.2. Across-study Variation

Across-study variation refers to the fact that different published studies of the same pollutant/health effect relationship typically do not report identical findings; in some instances the differences are substantial. These differences can exist even between equally reputable studies and may result in health effect estimates that vary considerably. Across-study variation can result from two possible causes. One possibility is that studies report different estimates of the single true relationship between a given pollutant and a health effect due to differences in study design, random chance, or other factors. For example, a hypothetical study conducted in New York and one conducted in Seattle may report different C-R functions for the relationship between PM and mortality, in part because of differences between these two study populations (e.g., demographics, activity patterns). Alternatively, study results may differ because these two studies are in fact estimating different relationships; that is, the same reduction in PM in New York and Seattle may result in different reductions in premature mortality. This may result from a number of factors, such as differences in the relative sensitivity of these two populations to PM pollution and differences in the composition of PM in these two locations. In either case, where we identified multiple studies that are appropriate for estimating a given health effect, we generated a pooled estimate of results from each of those studies.

3.1.3. Application of C-R Relationship Nationwide

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

overall effect on health benefit estimates introduced by application of a single C-R function to the entire U.S. This may be a significant uncertainty in the analysis, but the current state of the scientific literature does not allow for a region-specific estimation of health benefits.[21]

3.1.4. Extrapolation of C-R Relationship Across Populations

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

3.1.5. Uncertainties in the PM Mortality Relationship

Health researchers have consistently linked air pollution, especially PM, with excess mortality. A substantial body of published scientific literature recognizes a correlation between elevated PM concentrations and increased mortality rates. However, there is much about this relationship that is still uncertain.[22] These uncertainties include:

- ***Causality.*** A substantial number of published epidemiological studies recognize a correlation between elevated PM concentrations and increased mortality rates; however these epidemiological studies, by design, can not definitively prove causation. For the analysis of the Clear Skies Act, we assumed a causal relationship between exposure to elevated PM and premature mortality, based on the consistent evidence of a correlation between PM and mortality reported in the substantial body of published scientific literature.

- ***Other Pollutants.*** PM concentrations are correlated with the concentrations of other criteria pollutants, such as ozone and CO, and it is unclear how much each of these pollutants may influence mortality rates. Recent studies (see Thurston and Ito, 2001) have explored whether ozone may have mortality effects independent of PM, but we do not view the evidence as conclusive at this time. To the extent that the C-R functions we use to evaluate the Clear Skies Act in fact capture mortality effects of other criteria pollutants

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

[22] The morbidity studies used in the Clear Skies Act benefits analysis may also be subject to many of the uncertainties listed in this section.

besides PM, we may be overestimating the benefits of reductions in PM. However, we are not providing separate estimates of the mortality benefits from the ozone and CO reductions likely to occur due to the Clear Skies Act.

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

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

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

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

3.2. Uncertainties Associated with Environmental Effects Estimation

Our analysis of the Clear Skies Act includes a quantitative estimate of only two environmental effects: recreational visibility and ozone effects on agriculture. Scientific studies, however, have reliably linked atmospheric emissions of sulfur, nitrogen, and mercury to a much wider range of other environmental and ecological effects. Some of these effects are acute in nature, and some are longer-term and could take many years to manifest. The effects include the following:

- ***Acidic Deposition***. Effects associated with the deposition of sulfuric and nitric acid, formed in the atmosphere from sulfur dioxide and nitrogen oxide emissions, include direct toxic effects to plant leaves and aquatic organisms; progressive deterioration of soil quality; and chronic acidification of surface waters.

- ***Nitrogen Deposition***. Effects associated with deposition of atmospheric nitrogen compounds include saturation of terrestrial ecosystems and progressive nitrogen enrichment of coastal estuaries. The latter can lead to excessive algal growth, which can drastically reduce dissolved oxygen levels in aquatic ecosystems, and eventually diminish stocks of commercially and recreationally important fish and shellfish species.

- ***Mercury Deposition***: Effects associated with mercury deposition include direct toxic effects to animals, conservation of mercury in biogeochemical cycles, and accumulation of mercury in the food chain. Mercury in the food chain can eventually lead to developmental effects in children and/or the substantial curtailment of commercial and recreational fishing activities.

- ***Ozone:*** Tropospheric ozone, which forms from atmospheric reactions of nitrogen oxides and volatile organic compounds, can have direct toxic effects to plant leaves (including agriculture and commercial forests) and alter ecosystem wide patterns of energy flow and nutrient cycling. Only some ozone effects on agriculture are quantified in this analysis.

These effects are left unquantified for a variety of reasons, but mostly because of the complexity of modeling these effects and the major uncertainties in reliably quantifying the incremental effects of atmospheric emissions reductions on ecological endpoints.

Individually, many of these environmental effects may be relatively small in terms of their overall ecosystem and monetary importance, particularly in the near-term. Their cumulative and longer term effects, however, some of which may be largely unknown at this time, may be substantial. As a result, the omission of this broad class of benefits from our quantitative results likely causes our estimates to substantially understate the total benefits of the Clear Skies Act.

3.3 Uncertainties Associated with Economic Valuation of Benefits

Economic valuation of benefits often involves estimation of the willingness-to-pay of individuals to avoid harmful health or environmental effects. In most cases, there are no markets in which to directly observe WTP for these types of commodities. In some cases, we can rely on indirect market transactions, such as the implicit tradeoff of wages for on-the-job mortality risk among the working population, to estimate WTP. In other cases, we must rely on survey approaches to estimate WTP, usually through a variant of the contingent valuation approach, which generally involves directly questioning respondents for their WTP in hypothetical market situations. Regardless of the method used to estimate WTP, there are measurement errors, data inadequacies, and ongoing debates about the best practices for each method that contribute to the overall uncertainty of economic estimates.

3.3.1. General Benefits Transfer Considerations

For the Clear Skies benefits analysis, we do not have the time or resources to conduct primary economic research targeted at the specific air pollution-related benefits provided. As a result, we rely on the transfer of benefits estimates from existing studies. The conduct of "benefits transfer" exercises necessarily involves some uncertainties. These uncertainties can be reduced by careful consideration of the differences in the health risk or air pollution commodity and the study populations in the underlying economic literature versus the context of benefits conferred by the Clear Skies Act. For example, we make adjustments to the mortality valuation estimates to account for the estimated lag between exposure and manifestation of the effect, reflecting the basic economic tenet that individuals prefer benefits that occur sooner to those that occur later. We also make adjustments to account for expected changes in WTP over time as per capita income increases. We cannot adjust for all benefits transfer considerations, however, thus introducing additional uncertainty into our estimates.

3.3.2. Lack of Adequate Data or Methods

The lack of adequate data or methods to characterize WTP results in our inability to present monetized benefits of some categories of effects. For example, while studies exist that estimate the benefits of visibility improvements to individuals in the places they reside, these residential visibility studies are considered by some in the resource economics community to be less reliable because of the methods applied. In the case of residential visibility, we conduct sensitivity analyses to estimate the impact of this uncertainty in the reliability of methods. To the extent effects such as these represent categories of benefits that are truly valuable to the U.S. population, we have underestimated the total benefits of the Clear Skies Act.

3.3.3. Uncertainties Specific to Premature Mortality Valuation

The economic benefits associated with premature mortality are the largest category of monetized benefits of the Clear Skies Act.[23] In addition, in prior analyses EPA has identified valuation of mortality benefits as the largest contributor to the range of uncertainty in monetized

benefits (see USEPA 1999a). Because of the uncertainty in estimates of the value of premature mortality avoidance, it is important to adequately characterize and understand the various types of economic approaches available for mortality valuation. Such an assessment also requires an understanding of how alternative valuation approaches reflect that some individuals may be more susceptible to air pollution-induced mortality, or reflect differences in the nature of the risk presented by air pollution relative to the risks studied in the relevant economic literature.

The health science literature on air pollution indicates that several human characteristics affect the degree to which mortality risk affects an individual. For example, some age groups appear to be more susceptible to air pollution than others (e.g., the elderly and children). Health status prior to exposure also affects susceptibility. At risk individuals include those who have suffered strokes or are suffering from cardiovascular disease and angina (Rowlatt, et al. 1998). An ideal benefits estimate of mortality risk reduction would reflect these human characteristics, in addition to an individual's willingness to pay (WTP) to improve one's own chances of survival plus WTP to improve other individuals' survival rates.[20] The ideal measure would also take into account the specific nature of the risk reduction commodity that is provided to individuals, as well as the context in which risk is reduced. To measure this value, it is important to assess how reductions in air pollution reduce the risk of dying from the time that reductions take effect onward, and how individuals value these changes. Each individual's survival curve, or the probability of surviving beyond a given age, should shift as a result of an environmental quality improvement. For example, changing the current probability of survival for an individual also shifts future probabilities of that individual's survival. This probability shift will differ across individuals because survival curves are dependent on such characteristics as age, health state, and the current age to which the individual is likely to survive.

Although a survival curve approach provides a theoretically preferred method for valuing the benefits of reduced risk of premature mortality associated with reducing air pollution, the approach requires a great deal of data to implement. The economic valuation literature does not yet include good estimates of the value of this risk reduction commodity. As a result, in this study we value avoided premature mortality risk using the value of statistical life approach in the Base Estimate, supplemented by valuation based on an age-adjusted value of statistical life estimate in the Alternative Estimate.

Other uncertainties specific to premature mortality valuation include the following:

- ***Across-study Variation***: The analytical procedure used in the main analysis to estimate the monetary benefits of avoided premature mortality assumes that the appropriate economic value for each incidence is a value from the currently accepted range of the value of a statistical life. This estimate is based on 26 studies of the value of mortality risks. There is considerable uncertainty as to whether the 26 studies on the value of a statistical life provide adequate estimates of the value of a statistical life saved by air pollution reduction. Although there is considerable variation in the analytical designs and data used in the 26 underlying studies, the majority of the studies involve the value of risks to a middle-aged working population. Most of the studies examine differences in wages of risky occupations, using a wage-hedonic approach. Certain characteristics of

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

both the population affected and the mortality risk facing that population are believed to affect the average willingness to pay (WTP) to reduce the risk. The appropriateness of a distribution of WTP estimates from the 26 studies for valuing the mortality-related benefits of reductions in air pollution concentrations therefore depends not only on the quality of the studies (i.e., how well they measure what they are trying to measure), but also on (1) the extent to which the risks being valued are similar, and (2) the extent to which the subjects in the studies are similar to the population affected by changes in pollution concentrations.

- ***Level of risk reduction***. The transferability of estimates of the value of a statistical life from the 26 studies to the Clear Skies Act analysis rests on the assumption that, within a reasonable range, WTP for reductions in mortality risk is linear in risk reduction. For example, suppose a study estimates that the average WTP for a reduction in mortality risk of 1/100,000 is $50, but that the actual mortality risk reduction resulting from a given pollutant reduction is 1/10,000. If WTP for reductions in mortality risk is linear in risk reduction, then a WTP of $50 for a reduction of 1/100,000 implies a WTP of $500 for a risk reduction of 1/10,000 (which is ten times the risk reduction valued in the study). Under the assumption of linearity, the estimate of the value of a statistical life does not depend on the particular amount of risk reduction being valued. This assumption has been shown to be reasonable provided the change in the risk being valued is within the range of risks evaluated in the underlying studies (Rowlatt et al. 1998).

- ***Voluntariness of risks evaluated***. Although there may be several ways in which job-related mortality risks differ from air pollution-related mortality risks, the most important difference may be that job-related risks are incurred voluntarily, or generally assumed to be, whereas air pollution-related risks are incurred involuntarily. There is some evidence that people will pay more to reduce involuntarily incurred risks than risks incurred voluntarily.[24] If this is the case, WTP estimates based on wage-risk studies may understate WTP to reduce involuntarily incurred air pollution-related mortality risks.

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

- ***Self-selection and skill in avoiding risk.*** Recent research (Shogren et al. 2002) suggests that VSL estimates based on hedonic wage studies may overstate the average value of a risk reduction. This is based on the fact that the risk-wage tradeoff revealed in hedonic

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

studies reflects the preferences of the marginal worker, i.e. that worker who demands the highest compensation for his risk reduction. This worker must have either higher risk, lower risk tolerance, or both. However, the risk estimate used in hedonic studies is generally based on average risk, so the VSL may be upwardly biased because the wage differential and risk measures do not match.

4. ESTIMATED BENEFITS OF THE CLEAR SKIES ACT IN 2010 AND 2020

4.1. Air Quality Improvements

In addition to calculating the physical effects and monetary impacts of the Clear Skies Act, we also estimated the distribution of particulate matter air quality improvements that will be experienced by the US population. Table 14 illustrates the numbers of individuals and the percent of the US population that they represent that will experience changes in ambient particulate matter concentrations in 2010 and 2020. As indicated in the table, the Clear Skies Act yields relatively modest air quality improvements for about one-fourth of the US population (i.e., changes in PM concentrations of less than 0.25 $\mu g/m^3$), in both 2010 and 2020, but more substantial improvements for a large percentage of the population, including improvements in excess of 1.5 $\mu g/m^3$ for more than 34.7 million individuals by 2020.

Table 14

Distribution of $PM_{2.5}$ Air Quality Improvements Over 2010 and 2020 Population Due to the Clear Skies Act

Change in Annual Mean $PM_{2.5}$ Concentrations (μg/m3)	2010 Population		2020 Population	
	Number (millions)	Percent	Number (millions)	Percent
0 > $\Delta PM_{2.5}$ Conc 0.25	81.3	26.7%	79.2	24.0%
0.25 > $\Delta PM_{2.5}$ Conc 0.50	57.8	19.0%	25.3	7.7%
0.50 > $\Delta PM_{2.5}$ Conc 0.75	60.1	19.7%	44.8	13.6%
0.75 > $\Delta PM_{2.5}$ Conc 1.0	57.1	18.8%	46.0	14.0%
1.0 > $\Delta PM_{2.5}$ Conc 1.25	34.9	11.5%	40.3	12.2%
1.25 > $\Delta PM_{2.5}$ Conc 1.50	9.8	3.2%	38.2	11.6%
1.50 > $\Delta PM_{2.5}$ Conc 1.75	3.1	1.0%	34.7	10.5%
1.75 > $\Delta PM_{2.5}$ Conc 2.0	0.4	0.1%	18.0	5.5%
$\Delta PM_{2.5}$ Conc > 2.0	-	-	3.2	1.0%

* Totals may not sum due to rounding.

4.2. Health and Welfare Benefits

Tables 15-17 present health benefits resulting from improvements in air quality between the Base Case and the Clear Skies Act scenarios. Table 15 presents the mean estimate of avoided health effects in 2010 and 2020 for each health endpoint included in the Base analysis, while Table 16 presents the same information by age group. We estimate that reductions in exposure to fine PM and ozone due to the Clear Skies Act will result in annual benefits of close to 8,000 fewer deaths in 2010 and over 14,000 fewer deaths in 2020.

Table 15

Reductions in Incidence of PM- and Ozone-related Adverse Health Effects Associated with the Clear Skies Analyses, 2010 and 2020: Base and Alternative Estimates

Endpoint	Pollutant	Avoided Incidence [a] (cases/year)	
		2010	2020
		Mean	Mean
Premature mortality Base Estimate: Long-term exposure (30+) Alternative Estimate, Short-term exposure:	PM	 7,900 4,700	 14,000 8,400
Chronic bronchitis (27+)	PM	5,400	8,800
Non-fatal myocardial infarctions (18+)	PM	13,000	23,000
Total hospital admissions – Respiratory (all ages)	PM and Ozone	5,200	10,000
Total hospital admissions – Cardiovascular	PM	3,200	5,800
Emergency Room Visits for Asthma (all ages)	PM and Ozone	8,500	14,000
Acute bronchitis (8-12)	PM	13,000	20,000
Lower respiratory symptoms (7-14)	PM	140,000	230,000
Upper respiratory symptoms (asthmatics 9-11)	PM	110,000	180,000
Work loss days (18-64)	PM	1,100,000	1,600,000
Minor restricted activity days (18-64)	PM and Ozone	6,600,000	10,300,000
School absence days (6-11)	Ozone	81,000	200,000

[a] Ozone-related mortality is not included in the estimate of premature mortality. Respiratory hospital admissions for PM includes admissions for COPD, pneumonia, and asthma, and ozone-related respiratory admissions includes all respiratory causes and subcategories for COPD and pneumonia. Cardiovascular hospital admissions for PM includes total cardiovascular and subcategories for ischemic heart disease, dysrhythmias, and heart failure.

Table 16

Reductions in Incidence of Health Endpoints by Age Group:
Base and Alternative Estimates

Pollutant	Endpoint/Age Group[a]	Avoided Incidence (cases/year)	
		2010	2020
Children, 0-17			
PM	Premature mortality - Alternative estimate: Short-term exposure	30	50
PM and Ozone	Hospital Admissions – Respiratory Causes	740	1,700
PM and Ozone	Emergency Room Visits for Asthma	8,300	13,000
PM	Acute bronchitis	13,000	20,000
PM	Lower respiratory symptoms	140,000	230,000
PM	Upper respiratory symptoms in asthmatics	110,000	180,000
Ozone	School absence days (two studies pooled)	81,000	200,000
Adults, 18-64			
PM	Premature mortality - Base estimate: Long-term exposure Alternative estimate: Short-term exposure	 1,900 1,100	 3,000 1,700
PM	Chronic bronchitis	4,300	6,500
PM	Non-fatal myocardial infarctions	5,400	8,500
PM	Hospital admissions – Respiratory Causes	730	1,200
PM	Hospital admissions - Cardiovascular Causes	1,300	2,100
PM	Work loss days	1,100,000	1,600,000
PM and Ozone	Minor restricted activity days	6,600,000	10,300,000
Adults, 65 and older			
PM	Premature mortality - Base estimate: Long-term exposure Alternative estimate: Short-term exposure	 6,000 3,600	 11,000 6,700
PM	Chronic Bronchitis	1,100	2,200
PM	Non-fatal Myocardial Infarctions	7,700	15,000
PM and Ozone	Hospital Admissions – Respiratory Causes	3,800	7,800
PM	Hospital Admissions - Cardiovascular Causes	1,900	3,600

[a] Ozone-related mortality is not included in the estimate of premature mortality.

Table 17 summarizes the mean Base Estimate monetized health and visibility benefits due to the Clear Skies Act. As Table 17 shows, monetized benefits of the Clear Skies Act in the continental United States will be $55 billion in 2010, including $54 billion in health benefits. In 2020, total benefits increase to $113 billion, including $110 billion in health benefits.

The total WTP for the visibility improvements in California, Southwestern, and Southeastern Class I areas brought about by the Clear Skies Act is $1.1 billion in 2010 and $2.9 billion in 2020. This value includes the value to households living in the same state as the Class

I area, as well as values for all households in the U.S. living outside the state containing the Class I area. The value also accounts for growth in real income.

Table 17 also presents the mean monetized health and visibility benefits of the Alternative Estimate, which will be $11 billion in 2010 and $23 billion in 2020. The reduced mortality incidence (40% lower for both 2010 and 2020) under the Alternative Estimate and the difference in valuation of premature mortality and chronic bronchitis explain the difference in benefits between these two approaches. Even using the Alternative Estimate benefit projections, however, the benefits of Clear Skies still outweigh the costs of $4.3 billion in 2010 and $6.9 billion in 2020. It is also important to note that both the Alternative and Base Estimate are likely to underestimate the benefits of this proposal because of the many environmental and health effects that we were unable to quantify in this analysis.

Table 17

Results of Human Health and Welfare Benefits Valuation for the Clear Skies Analyses, 2010 and 2020

Endpoint	Pollutant	Monetary Benefits [a] (millions 1999$)	
		2010 Mean	2020 Mean
Health Endpoints			
Premature mortality [b]			
Base estimate: Long-term exposure, (adults, 30 and over)			
3% discount rate	PM	$50,000	$100,000
7% discount rate	PM	$47,000	$97,000
Alternative estimate: Short-term exposure (all ages)			
3% discount rate	PM	$7,900	$17,000
7% discount rate	PM	$9,000	$19,000
Chronic bronchitis (adults, 26 and over)			
Base estimate: Willingness-to-pay	PM	$2,000	$3,800
Alternative estimate: Cost-of-illness			
3% discount rate	PM	$590	$1,100
7% discount rate	PM	$380	$680
Non-fatal myocardial infarctions			
3% discount rate	PM	$1,100	$1,900
7% discount rate	PM	$1,000	$1,800
Hospital admissions – Respiratory Causes	PM and Ozone	$73	$150
Total hospital admissions – Cardiovascular Causes	PM	$66	$120
Emergency Room Visits for Asthma	PM and Ozone	$2	$4
Acute bronchitis (children, 8-12)	PM	$5	$8
Lower respiratory symptoms (children, 7-14)	PM	$2	$4
Upper respiratory symptoms (asthmatic children, 9-11)	PM	$3	$5
Work loss days (adults, 18-65)	PM	$130	$200
Minor restricted activity days (adults, age 18-65)	PM and Ozone	$350	$540
School absence days (children, age 6-11)	Ozone	$6	$15
Worker productivity (outdoor workers, age 18-65)	Ozone	$10	$22
Welfare Endpoints			
Recreational visibility (86 Class I Areas)	PM	$1,100	$2,900
Agricultural crop damage (6 crops)	Ozone	$32	$31
Monetized Total			
Base estimate			
3% discount rate		$55,000	$113,000
7% discount rate		$52,000	$107,000
Alternative estimate			
3% discount rate		$11,300	$23,000
7% discount rate		$12,100	$25,000

[a] Ozone-related mortality is not included in the estimate of premature mortality. Respiratory hospital admissions for PM includes admissions for COPD, pneumonia, and asthma, and ozone-related respiratory admissions includes all respiratory causes and subcategories for COPD and pneumonia. Cardiovascular hospital admissions for PM includes total cardiovascular and subcategories for ischemic heart disease, dysrhythmias, and heart failure.

[b] Long-term exposure mortality was modeled as lagged, both in the base analysis and in the sensitivity analyses. The values shown here were adjusted to take this into account. For example, the base analysis assumes that 25 percent of premature deaths occur in the first year, 25 percent occur in the second year, and 16.7 percent occur in each of the three subsequent years after exposure. Using this lag structure, to account for the preferences of individuals for current risk reductions relative to future risk reductions, we discount the value of avoided premature mortalities occurring beyond the analytical year (2010 or 2020) using three and seven percent discount rates. No lag adjustment is necessary for the alternative estimate, which focuses on premature mortality occurring within a few days of the PM exposure.

5. SENSITIVITY ANALYSES OF KEY PARAMETERS IN THE BENEFITS ANALYSIS

The Base Estimate is based on EPA's current interpretation of the scientific and economic literature; its judgments regarding the best available data, models, and modeling methodologies; and the assumptions it considers most appropriate to adopt in the face of important uncertainties. The majority of the analytical assumptions used to develop the Base Estimate have been reviewed and approved by EPA's Science Advisory Board (SAB). However, data and modeling limitations as well as simplifying assumptions can introduce significant uncertainty into the benefit results and that reasonable alternative assumptions exist for some inputs to the analysis, such as the mortality C-R functions.

To address these concerns, the Base Estimate of benefits is supplemented with a series of sensitivity calculations that make use of other sources of concentration-response and valuation data for key benefits categories. These estimates are not meant to be comprehensive. Rather, they reflect some of the key issues identified by EPA or commentors as likely to have a significant impact on total benefits. Individual adjustments in the tables should not be added together without addressing potential issues of overlap and low joint probability among the endpoints.

5.1. Premature Mortality: Long term exposure

Given current evidence regarding their value, reductions in the risk of premature mortality is the most important PM-related health outcome in terms of contribution to dollar benefits. There are four important analytical assumptions that may significantly impact the estimates of the number and valuation of avoided premature mortalities. These include selection of the C-R function, structure of the lag between reduced exposure and reduced mortality risk, the relationship between age and VSL, and effect thresholds. Results of this set of sensitivity analyses are presented in Table 19.

Although the Krewski, et al. (2000) mean-based ("$PM_{2.5}$(DC), All Causes") model has been used exclusively to derive our Base Estimate of avoided premature mortality, this analysis also examined the sensitivity of the benefit results to the selection of alternative C-R functions for premature mortality. There are several sources of alternative C-R functions for this sensitivity analysis, including: (1) an extended analysis of the American Cancer Society data, reported in Table 2 of Pope et al. (2002); and (2) the Krewski et al. "Harvard Six Cities" estimate. The Pope et al (2002) analysis provides estimates of the relative risk for all-cause, cardiopulmonary, and lung cancer mortality, using a longer followup period relative to the original data examined in Krewski et al (2000). The SAB has noted that "the [Harvard Six Cities] study had better monitoring with less measurement error than did most other studies" (EPA-SAB-COUNCIL-ADV-99-012, 1999). For comparison with earlier benefits analyses, such as the first Section 812 Prospective Report to Congress, we also include estimates of avoided incidences of premature mortality based on the original ACS/Pope et al. (1995) analysis in the fifth row of Table 19. In addition to these alternative C-R functions, a broader set of

alternative mortality C-R functions is shown in rows six through ten of Table 19.

The results of these sensitivity analysis demonstrate that choice of C-R function can have a large impact on benefits, potentially doubling the effect estimate if the C-R function is derived from the HEI reanalysis of the Harvard Six-cities data (Krewski et al., 2000).

Table 19

Sensitivity Calculation: Alternative and Supplementary C-R Functions for Mortality

Reference	Age	PM Metric	Incidence (Cases/Year)		Benefits Impact on Base (% Change) (millions 1999$) [a]	
			2010	*2020*	*2010*	*2020*
Pope et al. (2002)	30+	Annual Mean $PM_{2.5}$	6,800	12,300	$44,000 -$6,600 (-12%)	$90,000 -$14,000 (-12%)
Pope et al. (2002), Cardiopulmonary-Related	30+	Annual Mean $PM_{2.5}$	5,000	9,000	$32,000 NA (NA)	$66,000 NA (NA)
Pope et al. (2002), Lung-Cancer-Related	30+	Annual Mean $PM_{2.5}$	900	1,800	$6,000 NA (NA)	$13,000 NA (NA)
Krewski et al. (2000) - Dockery et al. (1993) Reanalysis	25+	Annual Mean $PM_{2.5}$	22,700	40,500	$140,000 +$95,000 (+172%)	$300,000 +$194,000 (+171%)
Pope et al. (1995)	30+	Annual Median $PM_{2.5}$	8,700	15,700	$55,000 +$5,100 (+9%)	$120,000 +$12,000 (+11%)
Krewski et al. (2000) - Pope et al. (1995) Reanalysis	30+	Annual Median $PM_{2.5}$	7,200	13,100	$46,000 -$4,000 (-7%)	$96,000 -$7,000 (-6%)
Krewski et al. (2000) - Pope et al. (1995) Reanalysis - Random Effects, Independent Cities	30+	Annual Median $PM_{2.5}$	14,000	25,500	$90,000 +$39,000 (+72%)	$190,000 +$83,000 (+74%)
Krewski et al. (2000) - Pope et al. (1995) Reanalysis - Random Effects, Regional Adjustment	30+	Annual Median $PM_{2.5}$	8,200	14,900	$52,000 +$2,100 (+4%)	$110,000 +$5,700 (+5%)
Dockery et al. (1993)	25+	Annual Mean $PM_{2.5}$	21,200	38,000	$140,000 +$85,000 (+155%)	$280,000 +$180,000 (+155%)

[a] When calculating benefits for the alternative mortality C-R functions, we assume a 5-year distributed lag adjustment calculated using a three percent discount rate.

5.2. Alternative Lag Structures

The primary analysis, based on SAB advice, assumes that mortality occurs over a five year period, with 25 percent of the deaths occurring in the first year, 25 percent in the second year, and 16.7 percent in each of the third, fourth, and fifth years. Readers should note that the selection of a five-year lag is not supported by any scientific literature on PM-related mortality (NRC 2002). Rather it is intended to be a best guess at the appropriate distribution of avoided incidences of PM-related mortality. Although the SAB recommended the five-year distributed lag be used for the primary analysis, the SAB has also recommended that alternative lag structures be explored as a sensitivity analysis (EPA-SAB-COUNCIL-ADV-00-001, 1999).

Specifically, they recommended an analysis of 0, 8, and 15-year lags. It is important to keep in mind that changes in the lag assumptions do not change the total number of estimated deaths, but rather the timing of those deaths. The estimated impacts of alternative lag structures on the monetary benefits associated with reductions in PM-related premature mortality (estimated with the Krewski et al ACS C-R function) are presented in Table 20. These estimates are based on the value of statistical lives saved approach, i.e. $6.1 million per incidence, and are presented for both a 3 and 7 percent discount rate over the lag period. Even with an extreme lag assumption of 15 years, benefits are reduced by less than half relative to the no lag and primary (5-year distributed lag) benefit estimates.

Due to discounting of delayed benefits, the lag structure may also have a large impact on monetized benefits, reducing benefits by 30 percent if an extreme assumption that no effects occur until after 15 years is applied. If no lag is assumed, benefits are increased by around five percent.

5.3. Thresholds

Although the consistent advice from EPA's Science Advisory Board has been to model premature mortality associated with PM exposure as a non-threshold effect, that is, with harmful effects to exposed populations regardless of the absolute level of ambient PM concentrations, some analysts have hypothesized the presence of a threshold relationship. The nature of the hypothesized relationship is that there might exist a PM concentration level below which further reductions no longer yield premature mortality reduction benefits. EPA does not necessarily endorse any particular threshold and virtually every study to consider the issue indicates absence of a threshold.

A sensitivity analysis has been constructed by assigning different cutpoints below which changes in $PM_{2.5}$ are assumed to have no impact on premature mortality. The sensitivity analysis illustrates how our estimates of the number of premature mortalities in the Base Estimate might change under a range of alternative assumptions for a PM mortality threshold. However, this type of cutoff is unlikely, as supported by the recent NAS report, which stated that "for pollutants such as PM_{10} and $PM_{2.5}$, there is no evidence for any departure of linearity in the observed range of exposure, nor any indication of a threshold." (NRC, 2002) Another possible sensitivity analysis which has not been conducted at this time might examine the potential for a nonlinear relationship at lower exposure levels.

One important assumption that was adopted for the threshold sensitivity analysis is that no adjustments are made to the shape of the C-R function above the assumed threshold. Instead, thresholds were applied by simply assuming that any changes in ambient concentrations below the assumed threshold have no impacts on the incidence of premature mortality. If there were actually a threshold, then the shape of the C-R function would likely change and there would be no health benefits to reductions in PM below the threshold. However, as noted by the NAS, "the assumption of a zero slope over a portion of the curve will force the slope in the remaining segment of the positively sloped concentration-response function to be greater than was indicated in the original study" and that "the generation of the steeper slope in the remaining portion of the concentration-response function may fully offset the effect of assuming a threshold." The NAS

suggested that the treatment of thresholds should be evaluated in a formal uncertainty analysis.

The threshold analysis indicates that approximately 80 percent of the premature mortality related benefits are due to changes in $PM_{2.5}$ concentrations occurring above 10 $\mu g/m^3$, and around 10 percent are due to changes above 15 $\mu g/m^3$, the current $PM_{2.5}$ standard.

Table 20

Sensitivity Calculation: Alternative Threshold and Alternative Lag Structures for Long-term Mortality

Analysis	Description	Incidence (Cases/Year)		Benefits (millions 1999$) [a]	
		2010	*2020*	*2010*	*2020*
Alternative Threshold					
Mortality, Long-Term, 30+' Krewski et al. (2000) Annual Mean $PM_{2.5}$	No Threshold (Base case)	7,900	14,000	$50,000	$103,500
	5 ug/m3	7,900	14,000	$50,200	$103,300
	10 ug/m3	6,500	11,000	$41,600	$80,100
	15 ug/m3	890	760	$5,700	$5,600
	20 ug/m3	19	28	$120	$210
	25 ug/m3	6	9	$36	$63
Alternative Lag					
Mortality, Long-Term, 30+ Krewski et al. (2000) Annual Mean $PM_{2.5}$	No lag	7,900	14,000	$52,900	$110,000
	Base (5 Year) Distributed Lag: 25%, 25%, 17%, 17%, 16%	7,900	14,000	$50,300	$104,000
	8 Year Lag: Incidence Occurs 8th Year	7,900	14,000	$43,000	$89,000
	15 Year Lag: Incidence Occurs 15th Year	7,900	14,000	$35,000	$72,000

[a] When calculating benefits for the alternative threshold, a 5-year distributed lag adjustment is assumed, calculated using a three percent discount rate. For the alternative lag benefits, a three percent discount rate.

5.4. Overlapping Endpoints

We estimated the benefits of the modeled preliminary control options using the most comprehensive set of endpoints available. For some health endpoints, this meant using a C-R function that linked a larger set of effects to a change in pollution, rather than using C-R functions for individual effects. For example, for premature mortality, we selected a C-R function that captured reductions in incidences due to long-term exposures to ambient concentrations of particulate matter, assuming that most incidences of mortality associated with short-term exposures would be captured. In addition, the long-term exposure premature mortality C-R function for $PM_{2.5}$ is expected to capture at least some of the mortality effects associated with exposure to ozone.

In order to provide the reader with a fuller understanding of the health effects associated with reductions in air pollution associated with the preliminary control options, this set of sensitivity estimates examines those health effects which, if included in the primary estimate, could result in double-counting of benefits. For some endpoints, such as ozone mortality, additional research is needed to provide separate estimates of the effects for different pollutants, i.e. PM and ozone. These supplemental estimates should not be considered as additive to the total estimate of benefits, but illustrative of these issues and uncertainties. Sensitivity estimates included here include premature mortality associated with short-term exposures to ozone and acute respiratory symptoms in adults. Results of this set of sensitivity analyses are presented in Tables 21 and 22.

Table 21

Sensitivity Estimates for Potentially Overlapping Endpoints

Sensitivity	Reference	Pollutant	*2010*	*2020*	*2010*	*2020*
Ozone Mortality [a]	4 U.S. Studies [b]	Daily Mean and 1-hour Max ozone	200	540	$1,300	$4,000
Any-of-19 Respiratory Symptoms	Krupnick et al. (1990)	Daily 1-hour Max ozone	1,800,000	4,000,000	$42	$100
		Daily Mean PM_{10}	18,000,000	28,000,000	$440	$670

[a] All estimates rounded to two significant digits.
[b] The ozone mortality sensitivity estimate is calculated using results from four U.S. studies (Ito and Thurston, 1996; Kinney et al., 1995; Moolgavkar et al., 1995; and Samet et al., 1997)

Table 22 presents a suite of sensitivity estimates related to respiratory symptoms in the asthmatic population. These health endpoints are not considered independent from each other or from the base estimates.[25]

Table 22

Reductions in Incidence of Respiratory Symptoms in the Asthmatic Population

PM-Related Endpoint	Study	Pollutant	Description of Study Population	Avoided Incidence (cases/year)	
				Study Population Only	
				2010	*2020*
Asthma Attack Indicators [a]					
Shortness of Breath	Ostro et al. (2001)	$PM_{2.5}$	African American asthmatics, 8-13	14,000	24,000
Cough	Ostro et al. (2001)	$PM_{2.5}$	African American asthmatics, 8-13	25,000	43,000
Wheeze	Ostro et al. (2001)	$PM_{2.5}$	African American asthmatics, 8-13	23,000	40,000
Asthma Exacerbation – one or more symptoms	Yu et al. (2000)	PM_{10}	Asthmatics, 5-13	490,000	790,000
Cough	Vedal et al. (1998)	PM_{10}	Asthmatics, 6-13	230,000	360,000
Other symptoms/illness endpoints					
Upper Respiratory Symptoms	Pope et al. (1991)	PM_{10}	Asthmatics 9-11	110,000	180,000
Moderate or Worse Asthma	Ostro et al. (1991)	$PM_{2.5}$	Asthmatics, all ages	110,000	190,000
Acute Bronchitis	McConnell et al. (1999)	$PM_{2.5}$	Asthmatics, 9-15	4,500	7,000
Chronic Phlegm	McConnell et al. (1999)	$PM_{2.5}$	Asthmatics, 9-15	11,000	17,000
Asthma Attacks	Whittemore and Korn (1980)	Ozone	Asthmatics, all ages	74,000	180,000

[a] Note that these symptoms of asthma are not necessarily independent. Combinations of these symptoms may occur in the same individuals, so that the sum of the avoided incidences is not necessarily equal to the sum of the affected populations. Also, some asthma studies cover the same or similar endpoints in overlapping populations. For example, the Vedal et al (1998) and Ostro et al (2000) studies both examined cough. However, the Ostro et al (2000) estimate examined a more restricted population than Vedal et al (1998), so estimates should be combined with caution.

5.5. Alternative and Supplementary Estimates

We also examine how the value for individual endpoints or total benefits would change if we were to make a different assumption about specific elements of the benefits analysis. Specifically, in Table 23, we show the impact of alternative assumptions about other parameters, including infant mortality associated with exposure to PM, treatment of reversals in chronic bronchitis as lowest severity cases, effects of ozone on new incidences of chronic asthma, alternative C-R function for chronic bronchitis, alternative C-R functions for PM hospital and ER admissions, valuation of residential visibility, valuation of recreational visibility at Class I areas outside of the study regions examined in the Chestnut and Rowe (1990a, 1990b) study, and valuation of household soiling damages. Note that the low number of avoided cases of infant mortality per year reflects the low baseline infant mortality rate in the U.S.

Table 23

Sensitivity Analyses for the Clear Skies Act

Alternative Calculation	Description of Estimate	Impact on Base Benefit Estimate (3% discount rate) (million 1999$)	
		2010	***2020***
Infant Mortality	Avoided incidences of mortality in infants are estimated using the Woodruff et al (1997) C-R function. The number of avoided incidences of infant mortality is 23 in 2010 and 37 in 2020.	+$140 (+0.2%)	+$210 (+0.2%)
Chronic Asthma[a]	Avoided incidences of chronic asthma are estimated using the McDonnell, et al. (1999) C-R function relating annual average ozone levels to new incidences of asthma in adult males over the age of 27. The number of avoided incidences of chronic asthma is 1,973 in 2010 and 4,872 in 2020.	+$74 (+0.1%)	+$180 (+0.2%)
Reversals in chronic bronchitis treated as lowest severity cases	Instead of omitting cases of chronic bronchitis that reverse after a period of time, they are treated as being cases with the lowest severity rating. The number of avoided chronic bronchitis incidences is 4,678 in 2010 and 7,630 in 2020.	+$680 (+1.2%)	+$1,100 (+1.0%)
Hospital Admissions	Avoided incidences of hospital admissions are estimated using the Samet et.al. (2000) C-R function. The number of avoided incidences of COPD admission is 988 in 2010 and 1,958 in 2020; the number of avoided incidences of pneumonia admissions is 1,190 in 2010 and 2,360 in 2020; the number of avoided incidences of all cardiovascular admissions is 3,390 in 2010 and 6,700 in 2020.	-$13 (-0.02%)	-$10 (-0.02%)
Value of visibility changes in all Class I areas	Values of visibility changes at Class I areas in California, the Southwest, and the Southeast are transferred to visibility changes in Class I areas in other regions of the country.	+$430 (+0.8%)	+$870 (+0.8%)
Value of visibility changes in Eastern U.S. residential areas	Value of visibility changes outside of Class I areas are estimated for the Eastern U.S. based on the reported values for Chicago and Atlanta from McClelland et al. (1990).	+$1,100 (+2.0)	+$2,900 (+2.5%)
Value of visibility changes in Western U.S. residential areas	Value of visibility changes outside of Class I areas are estimated for the Western U.S. based on the reported values for Chicago and Atlanta from McClelland et al. (1990).	+$140 +0.3%)	+$320 (-+0.3%
Household soiling damage	Value of decreases in expenditures on cleaning are estimated using values derived from ESEERCO (1994).	+$200 (+0.4%)	+$320 (+0.3%)

[a] While no causal mechanism has been identified linking new incidences of chronic asthma to ozone exposure, two epidemiological studies shows a statistical association between long-term exposure to ozone and incidences of chronic asthma in exercising children and some non-smoking men (McConnell, 2002; McDonnell, et al., 1999).

The Alternative Estimate is based on several key parameters, including the starting point value of a statistical life used to calculate the value of a statistical life year and the number of life years gained for each premature death from air pollution avoided. This set of sensitivity analyses examines how changes to each of these assumptions will impact the Alternative Estimate. Two alternative values are examined for each parameter. For the starting VSL, values of $1 million and $10 million are used. For the number of life years gained, values of 1 year and 14 years are used. Results are presented in Table 24.

Table 24

Impacts of VSL and Life Years Gained Assumptions on Alternative Benefits Estimates

Alternative Calculation		Description of Estimate	Impact on Alternative Benefit Estimate (3% discount rate) (billion 1999$)	
			2010	2020
1	$1 million VSL	Derivation of VSLY based on starting VSL of $1 million	-$5.8 (-50%)	-$12.1 (-51%)
2	$10 million VSL	Derivation of VSLY based on starting VSL of $10 million	+$13.4 (118%)	+$28.1 (119%)
3	1 life year gained	Assumes each premature mortality avoided due to reductions in short-term exposures to $PM_{2.5}$ results in 1 life year gained.	-$6.0 (-53%)	-$12.6 (-53%)
4	14 life years gained	Assumes each premature mortality avoided due to reductions in short-term exposures to $PM_{2.5}$ results in 14 life years gained.	+$13.8 (121%)	+$29.6 (125%)

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